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R&S 114771

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DOW CHEMICAL OF CANADA, LIMITED

MODELAND ROAD, P.O. B X 1012, SARNIA, ONTARI N7T 7K7

VC0000514

July 30, 1981

The Honourable Bill W. Diachuk
Minister Responsible for Workers' Health,
Safety and Compensation
Government of Alberta
Legislative Building
Edmonton, Alberta

R&S 114772

Dear Mr. Diachuk:

I welcome this opportunity of meeting with you to express our views on the proposed Alberta regulations on vinyl chloride monomer. I would like to reiterate Dow's continuing support in the development of reasonable and practicable standards and regulations for the protection of human health. These must be based on the best available scientific information which has received the benefit of peer review.

Over the past year we have had a number of meetings and discussions with officials of your Occupational Health and Safety Division and we value this frank and open exchange of viewpoints and the opportunity to share our company's knowledge in vinyl chloride toxicology and epidemiology. Unfortunately we have failed to reach agreement on the proposed standard and we therefore would like to lay before you, briefly, the scientific basis for Dow's contention that the existing 5 ppm Alberta standard is more than adequate to protect the health of workers in this industry.

We submit the following points to support this conclusion:

- At a 1979 toxicology conference in Paris, vinyl chloride experts agreed with Professor Maltoni's statement:

  > "None (or no increase) of these specifically vinyl chloride-related tumours has been observed [in rats] at doses below 10 ppm."

- Dr. David Rall, Director of the National Institute of Environmental Health Sciences, has noted the wide difference in susceptibility to vinyl chloride between

...../2

man and rats. He stated that the available data on liver angiosarcoma in rats

> "project a cancer incidence rate 500 times higher than has so far been reported in man by epidemiological studies for vinyl chloride."

- Human epidemiological evidence, based on forty years' experience in the manufacture and use of vinyl chloride, should be given greater importance than animal studies. These human studies confirm that man is less sensitive to vinyl chloride than is the rat. The two major epidemiological studies have linked liver angiosarcoma with chronic exposure to high (> 200 ppm) levels of vinyl chloride, but no angiosarcomas were observed below 200 ppm. Furthermore, workers with long-term occupational exposure to vinyl chloride levels which were low, but well in excess of 5 ppm, had no angiosarcoma or any other vinyl chloride related disease.

- The cancer risk of exposure to 5 ppm of vinyl chloride (the present Alberta standard) is low and well within the risks of ordinary living. Thus, a work force of 200 people exposed to 5 ppm would be expected to suffer one liver angiosarcoma every 35,000 years.

- Further lowering the current 5 ppm standard will result in increased costs with no real health benefits.

- The contention of the Occupational Health and Safety Division that tumours observed below 10 ppm in rats have "biological significance" is clearly based on incorrect scientific methodology. Maltoni and the world-wide vinyl chloride experts, in a thorough analysis of the results, drew no such conclusions in the Paris Symposium of November, 1979.

In conclusion, while our position is still 10 ppm, we believe that your officials exhibited sound judgement in 1975 when they set the standard at 5 ppm, judgement which has been vindicated by subsequent scientific data. We submit, with respect, that this standard is reasonable, practicable and scientifically supportable and that it should be retained.

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Our reason for raising this issue with you goes beyond our immediate concern for vinyl chloride per se. It stems from our belief that Alberta's approach to regulation of all chemicals should have a sound scientific basis.

Yours very truly,

J.M. Hay
President

Attachments: 1. Dow Position Statement on Alberta's Proposed Occupational Standard for Vinyl Chloride

2. Critical Appraisal of Data Presented by the Alberta Occupational Health and Safety Division

Appendix: "Analyzing the Daily Risks of Life" by Richard Wilson

JMH/ib

July 30, 1981

DOW CHEMICAL OF CANADA, LIMITED: POSITION STATEMENT ON ALBERTA'S PROPOSED OCCUPATIONAL STANDARD FOR VINYL CHLORIDE

After carefully examining the position taken by the Occupational Health and Safety Division at our last meeting and again reviewing the scientific information on vinyl chloride, we remain of the opinion that the current vinyl chloride occupational exposure limit of 5 parts per million (ppm) as an 8 hour average is an appropriate standard supported by the relevant toxicological and epidemiological data. This conclusion is supported by the following evidence.

1. Liver angiosarcoma is the tumour type caused by low vinyl chloride exposure in rats, other tumours being formed only at higher concentrations (Maltoni, 1979). In humans, liver angiosarcoma is the only tumour type firmly associated with vinyl chloride exposure (Fox, 1977 and Equitable Environmental Health, Inc., 1978). Thus, protecting against angiosarcoma of the liver would protect against all other tumour types.

2. The most recent data of Maltoni indicate a possible increase in angiosarcoma in rats exposed to 10 ppm of vinyl chloride but no statistically significant increase in tumours was found in experimental rats exposed below 10 ppm (Maltoni, 1979). The tumours observed below 10 ppm are not due to vinyl chloride exposure and they have no toxicological significance because:

   - The number of tumours from the vinyl chloride exposed rats is not statistically different from the unexposed (control) animals;
   - The tumours do not exhibit a dose-response relationship to vinyl chloride exposure;
   - At a 1979 toxicology conference in Paris, vinyl chloride experts agreed with Maltoni's statement:

     "None (or no increase) of these specifically vinyl chloride-related tumours has been observed at doses below 10 ppm." (Maltoni, 1979).

3. It is clearly evident from the metabolic studies by Buchter et al. (Buchter, 1980) and Gehring et al. (Gehring, 1979) that rodents are more susceptible to tumour induction by vinyl chloride than humans and that human experience should be given higher importance than animal studies. Dr. David Rall, the director of the U.S. National Institute of Environmental Health Sciences, has stated that the available data on liver angiosarcoma in rats (Rall, 1977 and 1979):

   "project a cancer incidence rate 500 times higher than has so far been reported in man by epidemiological studies for vinyl chloride."

4. Extensive human experience and epidemiological studies based on forty years' manufacture and use of vinyl chloride should be given precedence over animal studies:

   - Angiosarcoma of the liver has been linked with chronic exposure to high (>200 ppm) levels of vinyl chloride in the two major human epidemiological studies (Fox, 1977 and Equitable Environmental Health, Inc., 1978); no angiosarcomas were observed below 200 ppm.

   - Groups of workers chronically exposed to levels which were low, but well in excess of 5 ppm, were found to have no angiosarcoma (Ott, 1975 and Chiazze, 1977).

   - No liver damage or any other vinyl chloride related diseases are associated with a chronic occupational exposure to vinyl chloride of 50 ppm or less (Kramer, 1972).

   Thus the current Alberta occupational standard of 5 ppm is more than adequate to protect the health of workers.

5. The cancer risk of workers exposed for 35 years to 5 ppm of vinyl chloride has been estimated to be 3.7×10^{-6} (Gehring, 1979 and Reitz, 1979), which is well within the risks of everyday life (see Appendix). This means that, for example, a work force of 200 people exposed to 5 ppm of vinyl chloride would be expected to suffer one liver angiosarcoma every 35,000 years.

6. The world-wide mortality statistics from liver angiosarcoma among polyvinyl production workers are shown in the accompanying graph. Despite increase in production of vinyl chloride and polyvinyl chloride and the associated increase in the number of exposed workers, the total world-wide angiosarcoma mortality has declined since 1976. The 10 Canadian cases all occurred in a vinyl plant at Shawinigan, Quebec, which was built in 1941.

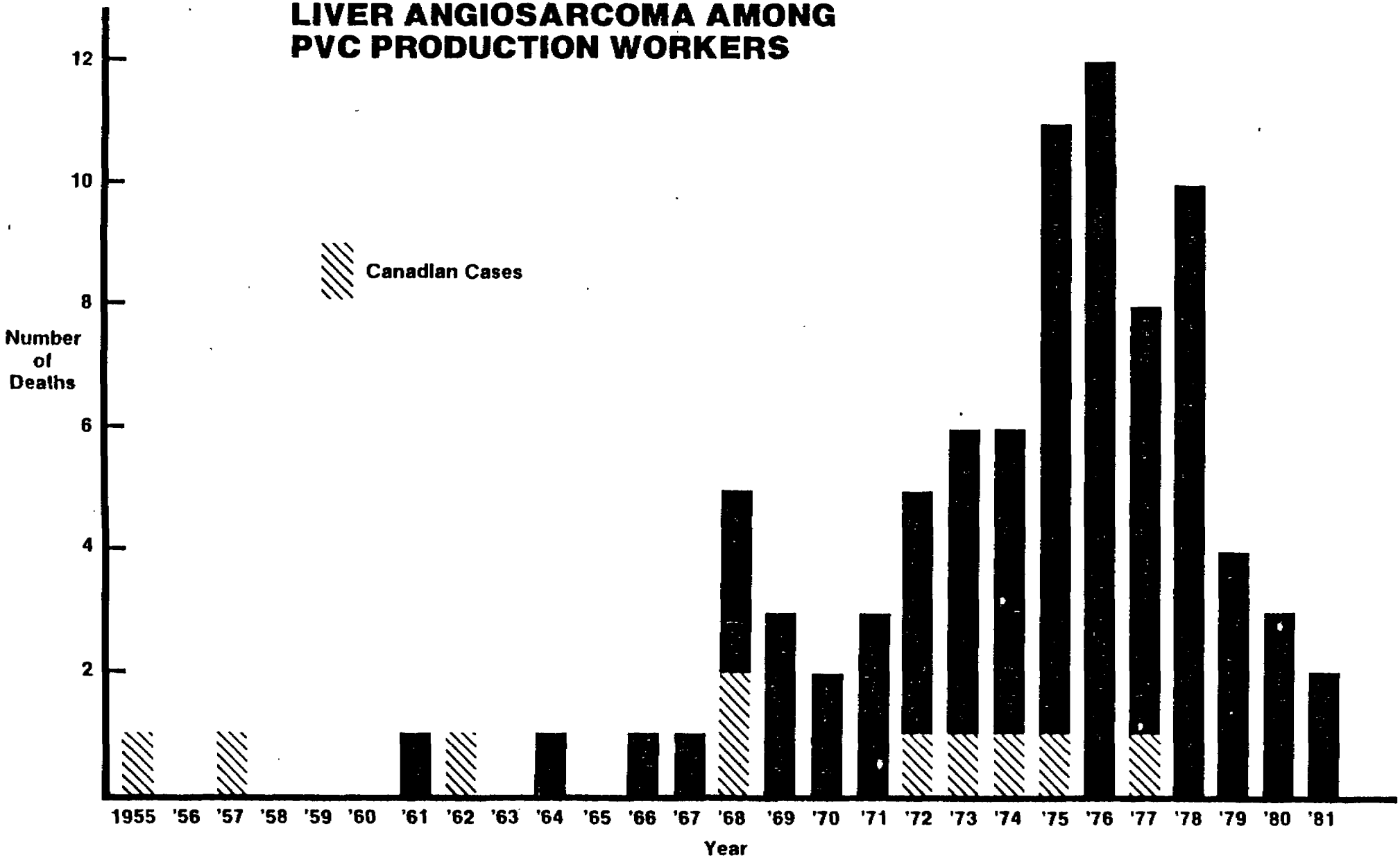
Having reviewed the relevant toxicological, epidemiological, and medical data, we conclude that the current vinyl chloride occupational exposure limit of 5 parts per million (ppm) as an 8 hour time-weighted average is reasonable, practicable and scientifically supportable and should be retained.

A critical appraisal of the data presented by the Alberta Occupational Health and Safety Division follows.

William Chen

William L. Chen, B.Sc., Ph.D.
Diplomate, American Board of Toxicology
Corporate Toxicologist

WORLD-WIDE MORTALITY FROM LIVER ANGIOSARCOMA AMONG PVC PRODUCTION WORKERS



Based on Data Compiled by Dr. John Stafford, 1981.

R&S 114778

[DR. IRVING R. TABERSHAW IS THE ORIGINAL INVESTIGATOR OF THE HUMAN EPIDEMIOLOGICAL STUDY OF POLYVINYL CHLORIDE/VINYL CHLORIDE WORKERS IN THE UNITED STATES, AND THE AUTHOR OF THE PRESTIGIOUS TABERSHAW-GAFFEY REPORT. DOW CANADA'S POSITION STATEMENT ON VINYL CHLORIDE AND THE ACCOMPANYING DOCUMENTS WERE SENT TO DR. TABERSHAW FOR HIS CRITICAL COMMENT. THE FOLLOWING TELEX IS HIS RESPONSE].

JULY 27, 1981
DOCTOR WILLIAM L. CHEN
CORPORATE RESEARCH AND DEVELOPMENT
DOW CHEMICAL OF CANADA
MODELAND CENTER, MODELAND ROAD
SARNIA, ONTARIO
CANADA

YOUR SUMMARY OF THE CURRENT STATUS OF THE SCIENTIFIC INFORMATION RELEVANT TO A HEALTH STANDARD FOR VINYL CHLORIDE IS BOTH CONCISE AND ACCURATE. I AGREE WITH YOUR CONCLUSION THAT 5 PARTS PER MILLION (PPM) ON AN 8 HOUR TIME WEIGHTED AVERAGE (TWA) IS ADEQUATE TO PROTECT THOSE WORKERS EXPOSED TO THIS CHEMICAL.

VINYL CHLORIDE AT HIGH DOSES CAN DEFINITELY BE CAUSALLY RELATED TO ANGIO-SARCOMA BUT A FIRM CONCLUSION THAT IT IS A MULTIPLE CARCINOGEN IS NOT JUSTIFIED FROM OUR STUDY. OUR DATA SUGGESTED THE POSSIBILITY AND WAS STATED AS AN "HYPOTHESIS" TO GENERATE INTEREST IN FOLLOW-UP. SUBSEQUENT EPIDEMIOLOGICAL STUDIES HAVE NOT CONFIRMED THIS CONCLUSION AT LOW DOSES. OUR DATA HAD NO ENVIRONMENTAL MEASUREMENTS, BUT BEAR IN MIND THAT THE STANDARD FOR VINYL CHLORIDE IN THE UNITED STATES AT THAT TIME WAS 500 PPM. IT APPEARS TO ME FROM THE DATA ON WORKERS RECENTLY EXPOSED TO VINYL CHLORIDE THAT 5 PPM (TWA) IS A SAFE LEVEL.

IRVING R. TABERSHAW, M.D.
PRESIDENT
TABERSHAW OCCUPATIONAL MEDICINE ASSOCIATES
ROCKVILLE, MD.
U.S.A.

July 30, 1981

CRITICAL APPRAISAL OF THE DATA PRESENTED BY THE ALBERTA OCCUPATIONAL HEALTH AND SAFETY DIVISION.

I. General Comments

1. Commenting on stastical analysis as applied to liver angiosarcoma, Maltoni has observed:

> "There may be smaller differences between exposed and control groups which do not reach statistical significance, while these differences could still have meaning from an oncological point of view, (particularly in the case of tumours which are unfrequent (sic) in the animal colony). Therefore, the most important data should be commented on both in light of the performed statistical analysis and with a biological approach."

This point appears to have been misunderstood by Division scientists, who have incorrectly applied Maltoni's argument to tumours which have positive spontaneous background rates such as leukemia and mammary tumours. Tumours which have such background rates can be judged to be treatment-related only if a dose repsonse is demonstrated and a statistically significant increase over the control animals, as well as over the historical background tumour incidence for the strain of animal used, is established.

2. It makes no biological or toxicological sense to ignore dose-response in the assessment of whether a tumour type is treatment related. Dose-response does not mean simply an increase over control, because the control may be low. It means an increasing tumour response over some dose range. Maltoni has carried out experiments with vinyl chloride exposures ranging from 30,000 ppm to 1 ppm and all the results should be taken into account in assessing dose-response.

3. Simply counting excess tumours over the control and interpreting them as treatment related is meaningless since the comparison of two untreated control groups will also show some tumours to be numerically increased and some numerically decreased. This simply reflects minor biological variations in the spontaneous tumour rate.

4. Other regulatory agencies such as the U.S. Occupational Health and Safety Administration, U.S. Environmental Protection Agency, and the International Agency for Research on Cancer all require statistical signficance and dose-response to be demonstrated before the tumour is to be considered as chemical exposure related.

5. Liver angiosarcoma is the tumour type caused by low vinyl chloride exposure in rats, other tumours being found only at higher concentrations (Maltoni, 1979). In humans, liver angiosarcoma is the only tumour type firmly associated with vinyl chloride exposure (Fox, 1977 and Equitable Environmental Health, Inc., 1978). Thus, protecting against angiosarcoma of the liver would protect against all other tumour types.

6. The most recent data of Maltoni indicate a possible increase in angiosarcoma in the rats exposed to 10 ppm of vinyl chloride but no statistically significant increase in tumours was found in experimental rats exposed below 10 ppm. At a 1979 toxicology conference in Paris, vinyl chloride experts agreed with Maltoni's statement:

   > "None (or no increase) of these specifically vinyl chloride-related tumours has been observed at doses below 10 ppm." (Maltoni, 1979).

7. It is clearly evident from the metabolic studies by Buchter et al. (Buchter, 1980) and Gehring et al. (Gehring, 1979) that rodents are more susceptible to tumour induction by vinyl chloride than humans and that human experience should be given higher importance than animal studies. Dr. David Rall, the director of the U.S. National Institute of Environmental Health Sciences, has stated that the available data on liver angiosarcoma in rats (Rall, 1977 and 1979):

   > "project a cancer incidence rate 500 times higher than has so far been reported in man by epidemiological studies for vinyl chloride."

8. Extensive human experience and epidemiological studies based on forty years' manufacture and use of vinyl chloride should be given precedence over animal studies:

   - Angiosarcoma of the liver has been linked with chronic exposure to high (>200 ppm) levels of vinyl chloride in the two major human epidemiological studies (Fox, 1977 and Equitable Environmental Health, Inc., 1978); no angiosarcomas were observed below 200 ppm.

- Groups of workers chronically exposed to levels which were low, but well in excess of 5 ppm, were found to have no angiosarcoma (Ott, 1975 and Chiazze, 1977).

- No liver damage or any other vinyl chloride related diseases are associated with a chronic occupational exposure to vinyl chloride of 50 ppm or less (Kramer, 1972).

Thus the current Alberta occupational standard of 5 ppm is more than adequate to protect the health of workers.

II. Specific Comments

Specific comments regarding the pertinent data presented by the Alberta Occupational Health and Safety Division on May 14, 1981 are presented in the following pages.

SLIDE 1 (by the Occupational Health and Safety Division)

C. EXPERIMENT BT 15

ONE MOST RELEVANT TO US SINCE IT EXAMINES INHALATION EXPOSURES FROM 25 - 1 PPM.

PROTOCOL: EXPOSURE BY INHALATION TO VC IN AIR AT 25, 10, 5 AND 1 PPM; 4 HR/DAY, 5 DAY/WK., 52 WKS. RESULTS AFTER 147 WKS. ALL ANIMALS OBSERVED UNTIL SPONTANEOUS DEATH.

11 MAJOR TUMOUR TYPES SCREENED FOR:

MAMMARY

ZYMBAL GLAND

LEUKEMIAS

NEPHROBLASTOMAS

ANGIOSARCOMAS — LIVER / OTHER SITES

ANGIOMAS & FIBROANGIOMAS — LIVER / OTHER SITES

HEPATOMAS

FORESTOMACH EPITHELIAL

SKIN CARCINOMAS

SUBCUTANEOUS SARCOMAS

ENCEPHALIC TUMOURS — NEURO BLASTOMAS / OTHER TYPES

OTHERS

COMMENT ON SLIDE 1

MALTONI HAS CARRIED OUT EXPERIMENTS WITH VINYL CHLORIDE EXPOSURES RANGING FROM 30,000 PPM TO 1 PPM. EXPERIMENT BT 15 MUST NOT BE CONSIDERED ON ITS OWN, BUT IN CONJUNCTION WITH ALL THE OTHER STUDIES.

July 30, 1981

SLIDE 2 (by the Occupational Health and Safety Division)

TOTAL NUMBER TUMOURS (25-1 PPM VC)

	TUMOUR TYPE	MALE	FEMALE	TOTAL # TUMOURS	GREATER PROPORTION THAN CONTROLS
1.	MAMMARY	35	230	265	+
2.	ZYMBAL GLAND	5	3	8	-
3.	LEUKEMIAS	13	6	19	-
4.	NEPHROBLASTOMA	1	0	1	+
5.	ANGIOSARCOMA (LIVER)	1	5	6	+
6.	ANGIOSARCOMA (OTHER SITES)	2	0	2	+
7.	ANGIOMA/FIBROANGIOMA (LIVER)	0	1	1	+
8.	ANGIOMA/FIBROANGIOMA (OTHER SITES)	5	1	6	+
9.	HEPATOMAS	0	0	0	-
10.	FORESTOMACH EPITHELIAL TUMOURS	0	0	0	-
11.	SKIN CARCINOMAS	2	0	2	+
12.	SUBCUTANEOUS SARCOMAS	7	1	8	+
13.	ENCEPHALIC	2	1	3	-
14.	OTHERS	39	31	70	-
	TOTAL	112	279	391	+
	TOTAL NON-MAMMARY TUMOURS	77	49	126	+

TOTAL NO. TREATED MALE RATS EXAMINED = 236; TOTAL NO. CONTROLS EXAMINED = 60

TOTAL NO. TREATED FEMALE RATS EXAMINED = 240; TOTAL NO. CONTROLS EXAMINED = 60

R&S 114785

COMMENT ON SLIDE 2

IT IS SCIENTIFICALLY INVALID TO SIMPLY ADD UP ALL THE TUMOURS FROM FOUR DIFFERENT TREATMENT GROUPS AS WELL AS MALE AND FEMALE ANIMALS AND THEN COMPARE WITH THE SINGLE CONTROL GROUP. THIS IS CONTRARY TO ACCEPTED PRACTICE IN BIOLOGICAL RESEARCH. FURTHERMORE, SIMPLY COUNTING EXCESS TUMOURS OVER THE CONTROL WITHOUT STATISTICAL SIGNIFICANCE AND DOSE-RESPONSE IS MEANINGLESS (SEE GENERAL COMMENTS 1 TO 4).

SLIDE 3 (by the Occupational Health and Safety Division)

EXPERIMENT B1 15: EXPOSURE OF SPRAGUE DAWLEY RATS TO VCM IN AIR AT 25, 10, 5, 1 PPM; 4 HR/DAY, 5 DAY/WK., 52 WK. RESULTS AFTER 147 WK. (END OF EXPERIMENT).

R&S 114787

MAMMARY TUMOUR DATA

VCM Conc. (ppm)	Sex	Total No. Tumours	No. Tumours/ Tumour Bearing Animal	% Malignant (A)	% Benign (B)	No. Fibroma/ Fibroangioma	No. Carcinoma	No. Sarcoma	No. Carcino-Sarcoma
25	M	11	1.1	9	91	10	1	0	0
	F	60	1.9	28	72	43	15	0	2
10	M	6	1.0	0	100	6	0	0	0
	F	50	1.8	35	65	39	21	0	0
5	M	10	1.2	0	100	10	0	0	0
	F	66	2.1	35	65	43	22	1	0
1	M	8	1.1	13	87	7	0	1	0
	F	54	1.7	26	74	40	12	0	2
0 (CONTROL)	M	8	1.2	13	87	7	1	0	0
	F	33	1.6	18	82	27	6	0	0

(A) % MALIGNANT = $\frac{\text{No. CARCINOMAS + CARCINOSARCOMAS + SARCOMAS}}{\text{TOTAL NO. TUMORS}}$

(B) % BENIGN = $\frac{\text{No. FIBROMAS + FIBROANGIOMAS}}{\text{TOTAL NO. TUMOURS}}$

COMMENT ON SLIDE 3

THE INCREASE OF MAMMARY TUMOURS IN FEMALES IN THE VINYL CHLORIDE EXPOSED GROUP OVER THE CONTROL GROUP, WHILE IT MAY BE STATISTICALLY SIGNIFICANT, IS MOST LIKELY DUE TO AN ARTIFACT IN MALTONI'S EXPERIMENTAL DESIGN. THE CONTROL ANIMALS, UNLIKE THE EXPOSED ANIMALS, WERE NEVER INTRODUCED INTO THE INHALATION CHAMBERS. IT IS WELL KNOWN THAT INCREASE IN STRESS CAN INDUCE AN INCREASE IN MAMMARY TUMOURS IN FEMALE RODENTS (RILEY, 1975). THEREFORE THE OCCURRENCE OF MAMMARY TUMOURS IN THE EXPOSED GROUP IN MANY OF MALTONI'S STUDIES CAN BE EXPLAINED ON THE GROUND OF AN ENORMOUS DIFFERENCE IN STRESS CONDITIONS BETWEEN EXPOSED ANIMALS (IN INHALATION CHAMBERS) AND NON-EXPOSED CONTROL ANIMALS (NOT IN INHALATION CHAMBERS).

SLIDE 4 (by the Occupational Health and Safety Division

INTERPRETATIONS

1. NUMBER OF MAMMARY TUMOURS HIGHER IN FEMALES THAN MALES AND > FEMALE CONTROLS.

2. MALE TUMOURS ALMOST ALL BENIGN. GREATER NUMBER THAN CONTROLS TO 5 PPM LEVEL.

3. FEMALE TUMOURS PREDOMINANTLY BENIGN.

4. NUMBER OF MALIGNANT TUMOURS IN FEMALES > MALES AND CONTROLS TO 1 PPM LEVEL.

5. FEMALES EXHIBITED A GREATER NO. TUMOURS/TUMOUR BEARING ANIMAL THAN CORRESPONDING CONTROLS TO 1 PPM LEVEL.

COMMENT ON SLIDE 4

THE INCREASE OF MAMMARY TUMOURS IN FEMALES IN THE VINYL CHLORIDE EXPOSED GROUP OVER THE CONTROL GROUP, WHILE IT MAY BE STATISTICALLY SIGNIFICANT, IS MOST LIKELY DUE TO AN ARTIFACT IN MALTONI'S EXPERIMENTAL DESIGN. THE CONTROL ANIMALS, UNLIKE THE EXPOSED ANIMALS, WERE NEVER INTRODUCED INTO THE INHALATION CHAMBERS. IT IS WELL KNOWN THAT INCREASE IN STRESS CAN INDUCE AN INCREASE IN MAMMARY TUMOURS IN FEMALE RODENTS (RILEY, 1975). THEREFORE THE OCCURRENCE OF MAMMARY TUMOURS IN THE EXPOSED GROUP IN MANY OF MALTONI'S STUDIES CAN BE EXPLAINED ON THE GROUND OF AN ENORMOUS DIFFERENCE IN STRESS CONDITIONS BETWEEN EXPOSED ANIMALS (IN INHALATION CHAMBERS) AND NON-EXPOSED CONTROL ANIMALS (NOT IN INHALATION CHAMBERS).

IT IS WELL KNOWN THAT SPRAGUE-DAWLEY RATS, THE STRAIN USED BY MALTONI, EXHIBIT AN EXCEPTIONALLY HIGH SPONTANEOUS BACKGROUND INCIDENCE OF MAMMARY TUMOURS.

SLIDE 5 (by the Occupational Health and Safety Division

EXPERIMENT BT 15: SUMMARY OF MOST SIGNIFICANT TUMOURS

SEX	MALIGNANT	TUMOUR TYPE	LOWEST CONCENTRATION OBSERVED AT*
M	+	LEUKEMIA	25
M	+	NEPHROBLASTOMA	25
M	+	ANGIOSARCOMA (LIVER)	25
M	+	ANTIOSARCOMA (OTHER SITE)	10
M	-	ANGIOMA/FIBROANGIOMA (OTHER SITE)	10
M	+	SUBCUTANEOUS SARCOMA	5
M	±	OTHERS	5
M	±	MAMMARY	5
M	+	SKIN CARCINOMA	1
F	-	ANGIOMA/FIBROANGIOMA (LIVER)	25
F	-	ANGIOMA/FIBROANGIOMA (OTHER SITE)	10
F	+	ANGIOSARCOMA (LIVER)	10
F	+	ZYMBAL GLAND CARCINOMA	5
F	±	MAMMARY	1
F	+	SUBCUTANEOUS SARCOMA	1

*I.E. LOWEST CONCENTRATION AT WHICH INCIDENCE WAS GREATER THAN CONTROLS

COMMENT ON SLIDE 5

THE TUMOURS PRESENTED IN THIS SLIDE, WITH THE EXCEPTION OF ANGIOSARCOMA, ARE NOT DUE TO VINYL CHLORIDE EXPOSURE AND THEY HAVE NO TOXICOLOGICAL SIGNIFICANCE BUT RATHER REFLECT MINOR VARIATIONS IN SPONTANEOUS TUMOUR RATE. THIS IS BECAUSE:

- THE NUMBER OF TUMOURS FROM THE VINYL CHLORIDE EXPOSED RATS IS NOT STATISTICALLY DIFFERENT FROM THE UNEXPOSED CONTROL ANIMALS;

- THE TUMOURS DO NOT EXHIBIT A DOSE-RESPONSE RELATIONSHIP TO VINYL CHLORIDE EXPOSURE;

- THE CONTROL ANIMALS, UNLIKE THE EXPOSED ANIMALS, WERE NOT SUBJECTED TO THE STRESS OF BEING PLACED IN THE INHALATION CHAMBERS.

CLEARLY, THE DIVISION HAS DERIVED FROM MALTONI'S DATA MORE CONCLUSIONS THAN MALTONI HIMSELF. AT A 1979 TOXICOLOGY CONFERENCE IN PARIS, VINYL CHLORIDE EXPERTS AGREED WITH MALTONI'S STATEMENT:

> "NONE (OR NO INCREASE) OF THESE SPECIFICALLY VINYL CHLORIDE-RELATED TUMOURS HAS BEEN OBSERVED AT DOSES BELOW 10 PPM". (MALTONI, 1979).

SLIDE 6 (by the Occupational Health and Safety Division

INTERPRETATIONS:

1. MALES HAD GREATEST PROPORTION OF TUMOURS AT VC CONCENTRATIONS $\leq$ 25 PPM.

   MOST SIGNIFICANT TUMOURS WERE : ANGIOSARCOMA (OTHER SITES)
   ANGIOMA/FIBROANGIOMA (OTHER SITES)
   SUBCUTANEOUS SARCOMA
   OTHERS
   MAMMARY
   SKIN CARCINOMA

2. MOST SIGNIFICANT TUMOURS FOR FEMALES: ANGIOMA/FIBROANGIOMA (OTHER SITES)
   ANGIOSARCOMA (LIVER)
   ZYMBAL GLAND CARCINOMA
   MAMMARY
   SUBCUTANEOUS SARCOMA

3. NO SIGNIFICANT INCIDENCE FOR MALES OR FEMALES AT VC CONCENTRATIONS $\leq$ 25 PPM FOR HEPATOMAS, FORESTOMACH EPITHELIAL TUMOURS, AND ENCEPHALIC (NEUROBLASTOMAS - OTHER TYPES).

COMMENT ON SLIDE 6

THE MOST RECENT DATA OF MALTONI INDICATE A POSSIBLE INCREASE IN ANGIOSARCOMA IN RATS EXPOSED TO 10 PPM OF VINYL CHLORIDE BUT NO STATISTICALLY SIGNIFICANT INCREASE IN TUMOURS WAS FOUND IN EXPERIMENTAL RATS EXPOSED BELOW 10 PPM (MALTONI, 1979). THE TUMOURS OBSERVED BELOW 10 PPM ARE NOT DUE TO VINYL CHLORIDE EXPOSURE AND THEY HAVE NO TOXICOLOGICAL SIGNIFICANCE BECAUSE:

- THE NUMBER OF TUMOURS FROM THE VINYL CHLORIDE EXPOSED RATS IS NOT STATISTICALLY DIFFERENT FROM THE UNEXPOSED (CONTROL) ANIMALS;

- THE TUMOURS DO NOT EXHIBIT A DOSE-RESPONSE RELATIONSHIP TO VINYL CHLORIDE EXPOSURE;

- THE CONTROL ANIMALS, UNLIKE THE EXPOSED ANIMALS, WERE NOT SUBJECTED TO THE STRESS OF BEING PLACED IN THE INHALATION CHAMBERS.

CLEARLY, THE DIVISION HAS DERIVED FROM MALTONI'S DATA MORE CONCLUSIONS THAN MALTONI HIMSELF. AT A 1979 TOXICOLOGY CONFERENCE IN PARIS, VINYL CHLORIDE EXPERTS AGREED WITH MALTONI'S STATEMENT:

"NONE (OR NO INCREASE) OF THESE SPECIFICALLY VINYL CHLORIDE-RELATED TUMOURS HAS BEEN OBSERVED AT DOSES BELOW 10 PPM". (MALTONI, 1979).

July 30, 1981

SLIDE 7A (by the Occupational Health and Safety Division

HEALTH EFFECTS OF VINYL CHLORIDE (CH_2CHCL)

ACUTE EXPOSURE (HIGH CONCENTRATION)

ANAESTHETIC EFFECTS.

EUPHORIA, FEELING OF DROWSINESS, SLEEPINESS AND NARCOSIS

CHRONIC EXPOSURE

1. ACROOSTEOLYSIS
   (RAYNAUD'S PHENOMENON)

2. IMMUNOLOGIC CHANGES

3. PULMONARY CHANGES
   OBSTRUCTIVE AIRWAY DISEASE (?PVC)

4. HEPATIC DAMAGE
   THICKENED LIVER CAPSULE
   SUB-CAPSULAR FIBROSIS
   PROGRESSIVE PORTAL FIBROSIS
   SINUSOIDAL CELL STIMULATION

SLIDE 7B (by the Occupational Health and Safety Division)

MUTAGENETIC AND TERATOGENIC EFFECTS

NOT CONCLUSIVE

DOSE RESPONSE CURVE WITH PERIPHERAL LYMPHOCYTE CHANGES

CARCINOGENICITY

HAEMANGIOSARCOMA OF THE LIVER

COMMENT ON SLIDE 7A AND 7B

ALL THE HUMAN HEALTH EFFECTS PRESENTED IN THIS SLIDE ARE RELATED TO HIGH VINYL CHLORIDE EXPOSURES -- AT LEVELS MANY ORDERS OF MAGNITUDE HIGHER THAN THE CURRENT ALBERTA OCCUPATIONAL STANDARD OF 5 PPM. STUDIES HAVE DEMONSTRATED THAT LESSER EXPOSED POPULATIONS HAVE SHOWN NONE OF THESE HEALTH EFFECTS.

- ANGIOSARCOMA OF THE LIVER HAS BEEN LINKED WITH CHRONIC EXPOSURE TO HIGH (>200 PPM) LEVELS OF VINYL CHLORIDE IN THE TWO MAJOR HUMAN EPIDEMIOLOGICAL STUDIES (FOX, 1977 AND EQUITABLE ENVIRONMENTAL HEALTH, INC., 1978); NO ANGIOSARCOMAS WERE OBSERVED BELOW 200 PPM.

- GROUPS OF WORKERS CHRONICALLY EXPOSED TO LEVELS WHICH WERE LOW, BUT WELL IN EXCESS OF 5 PPM, WERE FOUND TO HAVE NO ANGIOSARCOMAS (OTT, 1975 AND CHIAZZE, 1977).

- NO LIVER DAMAGE OR ANY OTHER VINYL CHLORIDE-RELATED DISEASES ARE ASSOCIATED WITH A CHRONIC OCCUPATIONAL EXPOSURE TO VINYL CHLORIDE OF 50 PPM OR LESS (KRAMER, 1972).

July 30, 1981

SLIDE 8A (by the Occupational Health and Safety Division)

EPIDEMIOLOGY

1) DEFINITE CAUSE/EFFECT RELATIONSHIP -

VCM EXPOSURE - LIVER ANGIOSARCOMA

2) SUGGESTED CAUSE/EFFECT RELATIONSHIP -

VCM EXPOSURE - RESPIRATORY TRACT CANCER
- OTHER LIVER CANCER
- BRAIN CANCER

SLIDE 8B (by the Occupational Health and Safety Division

Tumours Presently Correlated to VC Exposure (by inhalation) on Experimental Rodents and Man

Species	Angio-Sarcomas of Liver	Tumours of Brain	Tumours of Lung	Lymphomas and Leukemias	Hepa-tomas	Angio-sarcomas	Nephro-blastomas	Sebaceous Cutaneous Carcinomas	Other Cutaneous Epithelial Tumours	Mammary Carcinomas	Fore-stomach Papillomas and Acanthomas
Rat	+	+		+	+	+	+	+	+	(+)	+
Mouse	+		+			+		+	+	+	(+)
Hamster	+		(+)	+		(+)			+		+
Man	+	(+)	(+)	(+)	(+)	+					

Maltoni, C. 1977. Cold Spring Harbour Conference on Cell Proliferation

COMMENT ON SLIDE 8A AND 8B

IN THE TWO LARGE SCALE EPIDEMIOLOGICAL STUDIES (FOX, 1977 AND EQUITABLE ENVIRONMENTAL HEALTH INC., 1978) ANGIOSARCOMA OF THE LIVER WAS THE ONLY TUMOUR TYPE SHOWN TO BE ASSOCIATED WITH VINYL CHLORIDE PRODUCTION. NO OTHER TUMOURS WERE DEMONSTRATED TO BE STATISTICALLY INCREASED AT A CONFIDENCE LEVEL OF 0.05.

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Analyzing the Daily Risks of Life

by Richard Wilson

In our most trivial activities we incur risks. These hazards can be quantified and compared, but can they be eliminated from our lives?

Anthony Schultz

The world seems a very hazardous place. Every day the newspapers announce that some chemical has been found to be carcinogenic, or some catastrophic accident has occurred in some far-off place. This leads some of us to hanker after a simpler world where there are fewer risks to life. But does such a world really exist?

If we look back at the world of a century ago, we find that expectation of life was 50 years; now it is 70 years. Therefore the sum of all the risks to which we are now exposed must be less than it was. We find that many of the large risks of the last century have been eliminated, leaving us conscious of a myriad of small risks, most of which have always existed.

The moment I climb out of bed I start taking risks. As I drowsily turn on the light I feel a slight tingle; my house is old with old wiring and there is a small risk of electrocution. Every year 500 people are electrocuted in the United States. I take a shower, and as I reach for the soap, I wonder about the many chemicals it contains. Are they all good for the skin, as the advertisements claim? My clothes have been cleaned with the best bleaching detergent. Most bleaching agents contain a chemical that fluoresces slightly in the sunlight to enhance the whiteness. Does this make bleaches carcinogenic?

I ponder this risk as I walk down to breakfast, taking care not to fall upon the stairs. Falls kill 16,000 people per year — mostly in domestic accidents. Shall I drink coffee or tea with my breakfast? Both contain caffeine, a well-known stimulant which may be carcinogenic. I have a sweet tooth; do I use sugar which makes me fat and gives me heart disease, or saccharin which we now know causes cancer? It is better to abstain.

After breakfast I make a sandwich for lunch. My son likes peanut butter. But improperly stored peanuts can develop a mold which produces a potent carcinogen — aflatoxin. In Africa and Southeast Asia, where aflatoxin appears more frequently, it has been blamed for numerous cases of liver cancer. In our (less natural) society storage facilities are better, so the risk is less — but it is not zero.

I prefer meat. But Americans, like other prosperous people, eat too much meat. It is not certain, but a meat-heavy diet probably contributes to cancer of the colon.

I live seven miles from work and can commute by car, by bicycle, or by bus. Which has the lowest risk? To travel by bicycle would keep my weight down, and bicycle riding does not cause pollution — but

statistics show that it is more likely to involve me in an accident. And since a bicyclist is unprotected, fatal accidents are also frequent on a bicycle. A car would be safer, but a bus is safest. I am happy that I no longer have to choose between a horse and a canoe; both are more dangerous (per mile) than a bicycle.

As I approach Boston, I see the urban haze caused by air pollution. There are toxic parts of air pollution which are not visible, as well. The risk to life caused by air pollution is high. Asthma victims have known this for a long time and fled the industrialized eastern United States for the purer air of the West. A press release from a government laboratory states that air pollution kills 20,000 people a year in the eastern United States. Air pollution, though still bad, has been reduced in most cities.

I remember the pea-soup London fogs of my youth caused by burning soft coal, where I could not see ahead ten feet; and the infamous week in December of 1952 where 3,000 people died from air pollution in four days.

I go to a committee meeting in a small, unventilated room. Although I don't smoke tobacco, half of the committee does, and I am exposed to the poison which causes 40 per cent of all cancers and kills 15 per cent of all Americans. Even though I breathe less tobacco smoke than my smoking colleagues, I often get a headache. One of my friends, who is more allergic than I, wears goggles at work.

At mid-morning I take a drink of water. The water tastes of chlorine, showing that the city's sanitation engineers use chlorine to kill microbes in the water. By such methods the country has nearly wiped out cholera and typhus. But the chlorine reacts with organic matter in the water to produce many known carcinogens. One of them, chloroform, is produced in a concentration of 100 parts per billion; enough to present a health hazard.

My office walls are brick and cinder block. Both contain radioactive materials, and radiation can increase my risk of cancer. One of these radioactive materials, radon, is a gas which is not chemically active. It is released by the brick and I can breathe it, which accentuates the hazard. I could prevent the release of this radioactive gas by painting the walls with thick epoxy paint to seal them, but that would introduce another risk. As the epoxy paint cures, it emits gaseous chemicals which are themslves carcinogenic. Which is worse?

Radiation enters all of my life. State law requires that I have a regular chest x-ray to see whether I have tuberculosis and may convey that dread disease to my students. But this adds to my risk of cancer from radiation. Is it correct for society to demand that I accept this risk, even to protect the rest of society from a greater one?

I frequently travel to meetings. Should I go by car, bus, train, or airplane? Thirty years ago the statistics were clear; the airplane was far more dangerous than all the others, since many airplanes crashed. Now, for journeys of 1,000 miles or more, air travel is the safest. But airplane travel causes an often-ignored radiation hazard, exposure to cosmic radiation from outer space. Airplanes fly at 30,000 feet, and at that altitude cosmic radiation exposure is 40 times what it is at sea level. Even a vacation trip to the high altitudes of Colorado and Wyoming can increase cosmic ray exposure. Sunlight at these altitudes, and excessive exposure even at sea level, showers us with ultraviolet light, which causes skin cancer.

These are personal concerns, and it might be argued that they are of no concern to anyone else, since I can avoid some of them. But in doing so I may well cause problems for others in society.

In the bad old days of my childhood we burnt coal in the house. If I heat my house by electricity I will not personally pollute the air with the products of fossil fuel burning; but these may still be produced at the power plant. One hopes the electric company is more careful about these pollutants than my parents used to be.

Whether I burn the coal myself or let the electric utility company do so, coal miners must still go underground. Anyone who has read *How Green Is My Valley* knows that 100 years ago coal mining was one of the most dangerous occupations. Even though mine safety has improved, it still has hazards: 156 out of every 100,000 miners were killed in accidents in 1972 in the United States. Yet accidents are not the worst hazard of coal mining: 800 miners yearly contract the dread black lung disease — coal workers' pneumoconiosis — from inhaling coal dust. One quarter of all American miners working in 1977 will probably contract this disease during their lifetimes. As an environmentalist I hate to see the beautiful western states laid bare by strip mining; but do I have a right to allow miners to die by refusing to let them work above ground?

Our society has a quirk which is fostered by our news media. We are far more concerned with infrequent large accidents than with numerous small accidents which, in total, cause many more deaths.

Congress was prompted into insisting on better mine ventilation to prevent black lung disease only after a much smaller number were killed in a single accident. A single accident of a school bus receives more newspaper coverage than the thousands of children killed yearly in automobile accidents.

This obsession with large accidents is getting worse. We are apprehensive at the *thought* of a large accident in a nuclear power plant, although none has happened so far, and experts are optimistic that none will ever happen. Nor is the fear unique to nuclear power. We bring to the United States considerable quantities of liquefied natural gas (LNG) and worry about the possibility of the ship leaking and blowing up. LNG *has* caused problems in the past; 30 years ago, an LNG tank, one-tenth the size of modern ones, collapsed and killed 133 people. We now know why this tank collapsed, and new tanks will not collapse in the same way since the metal from which the tanks are made has been changed.

Comparing the Risks We Face

There are those who would try to eliminate all known risks and would try to force this by law. This sounds plausible, but it creates an incentive for ignorance, not an incentive for safety. Under this procedure if we do not know whether something is risky and close our eyes to the possibility of risk, no one will bother us. On the other hand, if we look carefully and find there is a risk — even though it is small — some regulatory agency may stop us.

It would be a better policy to try to measure our risks quantitatively, and to give an *upper limit* on a risk when there is uncertainty. Then we could compare risks and decide which to accept or reject. I suspect most of us would decide to reduce the largest risks first.

To compare risks we must calculate them. As I prepared the table on page 45, I realized that an increased risk of death of one in a million is often seen as acceptable, but people instinctively think about large risks. I list here several actions which increase the chance of death in any year by one in a million.

Of course, if the risk of death in one year is increased, the risk of dying from another cause in a later year is decreased. The average expectation of life is shortened. Accidents often occur early in life, and life may be shortened 30 years by a typical accident. Cancer, black lung, and bronchitis kill later in life, and life is shortened only about 15 years. Therefore, a risk of 0.000001 (or 10^{-6}) shortens life on the

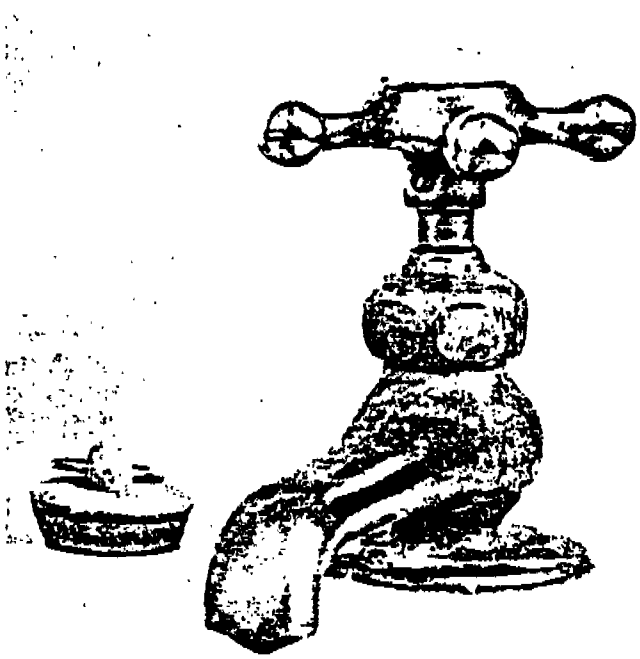


Photo Fredrik D Bodin, Stock Boston

"The moment I climb out of bed I start taking risks . . . I take a drink of water . . . the chlorine reacts with organic matter to produce many known carcinogens."

Photo: Michael Dobo, Stock, Boston

"As I approach Boston, I see the urban haze . . . Air pollution kills 20,000 people a year in the eastern U.S."

average by 30×10^{-6} years, or 15 minutes if it is an accident risk, 8 minutes if it is a risk of fatal illness.

I illustrate what this table means by calculating examples. In the United States 627 billion cigarettes were made in 1975. This is enough for 3,000 per person (including children), or a little less than half a pack a day. It is estimated that 15 per cent of all Americans (30 per cent of all smokers) die from lung or other cancers or heart disease due to smoking. We describe this as an average lifetime risk of 0.15. Dividing by the 70-year lifetime gives a yearly risk of 0.002 or 2×10^{-3}; dividing again by 3,000 gives a risk per cigarette of 0.7×10^{-6}. It is amusing to note that smoking a cigarette takes ten minutes and reduces the expectation of life by five minutes.

Human affairs are much more random than we like to think. One boy playing on a street can be killed by a passing car while his playmates are unharmed. All were equally at risk before the accident, but only one died. Similarly, one out of three lifetime smokers dies of cancer or heart disease because of the habit; the rest are unaffected and die of other causes. Moreover, those that die of cancer and heart disease do so at different ages. We have no way of telling which particular smokers will die of cancer, so we say that all are equally at risk.

It has been shown that those who smoke 40 cigarettes a day are ten times more likely to develop cancer as those who smoke four cigarettes a day. Perhaps there is a level of consumption where the risk becomes zero, but we cannot measure that low. It is easier to assume that every cigarette contributes the same amount to the total risk.

Brookhaven National Laboratory recently estimated that 20,000 Americans die every year from air pollution east of the Mississippi. This is partly due to sulphur emitted from burning coal and oil, and measurements suggest that the sulphate particulates spread themselves roughly uniformly over town and country. About 100 million Americans are exposed to this dirty air, so the average risk is 20,000/100,000,000 every year or 2×10^{-4} or 0.0002. Two days in New York City give a risk smaller by 2/365 or about 10^{-6} (one in a million).

Recent aircraft accident statistics tell us that aircraft in the United States carry passengers 100 billion passenger-miles every year and only about 100 people a year are killed in airplane crashes. This gives a risk of one in a million for one thousand miles of flight.

Professor Norman G. Rasmussen of M.I.T. made a study of nuclear reactor accident probabilities for

the Nuclear Regulatory Commission. He concluded that a reactor accident involving loss of life is very unlikely. The chance of an accident with more than 1,000 deaths is less than one in a 100 million per year of operation for each reactor. Most of these would be among the 20,000 or so people living within five miles of the reactor. So the probability of an individual living near a reactor being killed in a large accident is 1/2000 million. But those close by might also suffer in smaller accidents which, even though still unlikely, are more probable, leading to a risk of 1/50 million for persons living close to reactors.

Other more dangerous radiation hazards, such as natural radioactivity in brick, cosmic radiation, and diagnostic x-rays, are calculated by measuring the radiation dose and dividing it by the measured effect of large doses. The risks of these commonly accepted radiation hazards are far greater than those estimated for nuclear power.

I find these comparisons help me evaluate risks, and I imagine that they may help others do so, as well. But the most important use of these comparisons must be to help the decisions we make, as a nation, to improve our health and reduce our accident rate.

Taxing a Risk

Economists are fond of using taxation to control human affairs. Indeed, the invention of money by Croesus made a great simplification in the relationships in society. One suggestion, then, is to tax anyone who introduces a risk into society. This tax could pay for medical care, for compensating society for the loss of services, etc. The question arises: How much should the tax be? I suggest, as a basis for discussion, that this tax be at the rate of $1 million for every life that is lost by this extra risk, or one dollar for a risk of one in a million. Conversely, anyone that can save a life by an expenditure of $1 million must be encouraged to do so.

For example, the manufacturer who panders to the bad habit of cigarette smoking would pay an increased tax of 70 cents *per cigarette*. This is more than enough to pay the societal cost of cigarette smoking (hospital costs, fire hazards, reduced working time), which is variously estimated at from $1 to $2 per pack. Other taxes — five cents per diet soda — are less dramatic and might have to be accompanied by a tax of five cents on other sodas as well to prevent a switch to sugar.

Risks which increase chance of death by 0.000001*

Smoking 1.4 cigarettes	Cancer, heart disease
Drinking 1/2 liter of wine	Cirrhosis of the liver
Spending 1 hour in a coal mine	Black lung disease
Spending 3 hours in a coal mine	Accident
Living 2 days in New York or Boston	Air pollution
Travelling 6 minutes by canoe	Accident
Travelling 10 miles by bicycle	Accident
Travelling 300 miles by car	Accident
Flying 1000 miles by jet	Accident
Flying 6000 miles by jet	Cancer caused by cosmic radiation
Living 2 months in Denver on vacation from N.Y.	Cancer caused by cosmic radiation
Living 2 months in average stone or brick building	Cancer caused by natural radioactivity
One chest x-ray taken in a good hospital	Cancer caused by radiation
Living 2 months with a cigarette smoker	Cancer, heart disease
Eating 40 tablespoons of peanut butter	Liver cancer caused by aflatoxin B
Drinking Miami drinking water for 1 year	Cancer caused by chloroform
Drinking 30 12 oz. cans of diet soda	Cancer caused by saccharin
Living 5 years at site boundary of a typical nuclear power plant in the open	Cancer caused by radiation
Drinking 1000 24 oz. soft drinks from recently banned plastic bottles	Cancer from acrylonitrile monomer
Living 20 years near PVC plant	Cancer caused by vinyl chloride (1976 standard)
Living 150 years within 20 miles of a nuclear power plant	Cancer caused by radiation
Eating 100 charcoal broiled steaks	Cancer from benzopyrene
Risk of accident by living within 5 miles of a nuclear reactor for 50 years	Cancer caused by radiation

*(1 part in 1 million)

Photo Peter Menzel, Stock, Boston

"I go to a committee meeting in a small, unventilated room. Although I don't smoke tobacco . . . I am exposed to the poison which causes 40 per cent of all cancers and kills 15 per cent of all Americans."

These taxes might be earmarked to pay for risk reductions such as converting an existing sanitation system to using ozone instead of chlorine for sanitation, to avoid the production of chloroform.

Whether we quantify these risks or not, we must and do constantly make decisions about them. We do this as individuals, and our politicians make these decisions for us on a larger scale. What we are not doing, and need to do, is comparing the risks of various activities and then reducing the largest risks — which may not be the obvious ones.

After calculating these risks all day, I go home. I am still faced with decisions about risks. If I cook a meal in the microwave oven and the door doesn't fit tightly, I will be exposed to microwaves. It has recently been claimed that microwaves, even at low concentrations, give people nervous problems. Or I can use the gas stove, but the burning gas can fill my kitchen with both noxious carbon monoxide and nitrogen oxides.

Just as I go to bed I take a glass of beer. Alcohol causes cirrhosis of the liver and has been associated with oral and other cancers. However, the relaxing effect of the beer will reduce my stresses and permit a good night's sleep. This will prolong my life and is worth the risk.

The beer is in a green glass bottle which contains chromium, a small amount of which enters the beer. Chromium is a known carcinogen when ingested in moderate quantities, but it must not be avoided altogether because it is essential to life in small concentrations. How much chromium should I take to minimize the risk? Is the amount in the beer too much? Should I drink the beer from a plastic bottle? A plastic bottle suitable for beer has just been banned because a trace of the chemical from which the plastic was made could dissolve in the contents, and there is a suspicion that the chemical is carcinogenic.

I ponder this decision as I put on my pajamas. Are the pajamas inflammable? There is always a small risk of a fire starting while I am in bed. Is the risk of being burnt in a fire greater or smaller than the risk of cancer caused by a flame retardant such as TRIS?

I remember the truism "more people die in bed than anywhere else," so at least I'm in the right place.

Richard Wilson is professor of physics at Harvard University. Educated at Christ Church, Oxford, he received his Ph.D. in 1950. For many years he has been concerned with energy and the environment. He served on the National Science Foundation Physics Advisory Panel, as a consultant on nuclear power to the Attorney General's Office of the state of Maine, and as a consultant to the Nuclear Regulatory Commission. He is Assistant Editor of *Annals of Physics*.

DOW CHEMICAL OF CANADA, LIMITED

MODELAND ROAD, P.O. BOX 1012, SARNIA, ONTARIO N7T 7K7

July 30, 1981

The Honourable Bill W. Diachuk
Minister Responsible for Workers' Health,
Safety and Compensation
Government of Alberta
Legislative Building
Edmonton, Alberta

Dear Mr. Diachuk:

I welcome this opportunity of meeting with you to express our views on the proposed Alberta regulations on vinyl chloride monomer. I would like to reiterate Dow's continuing support in the development of reasonable and practicable standards and regulations for the protection of human health. These must be based on the best available scientific information which has received the benefit of peer review.

Over the past year we have had a number of meetings and discussions with officials of your Occupational Health and Safety Division and we value this frank and open exchange of viewpoints and the opportunity to share our company's knowledge in vinyl chloride toxicology and epidemiology. Unfortunately we have failed to reach agreement on the proposed standard and we therefore would like to lay before you, briefly, the scientific basis for Dow's contention that the existing 5 ppm Alberta standard is more than adequate to protect the health of workers in this industry.

We submit the following points to support this conclusion:

- At a 1979 toxicology conference in Paris, vinyl chloride experts agreed with Professor Maltoni's statement:

  > "None (or no increase) of these specifically vinyl chloride-related tumours has been observed [in rats] at doses below 10 ppm."

- Dr. David Rall, Director of the National Institute of Environmental Health Sciences, has noted the wide difference in susceptibility to vinyl chloride between

...../2

man and rats. He stated that the available data on liver angiosarcoma in rats

> "project a cancer incidence rate 500 times higher than has so far been reported in man by epidemiological studies for vinyl chloride."

- Human epidemiological evidence, based on forty years' experience in the manufacture and use of vinyl chloride, should be given greater importance than animal studies. These human studies confirm that man is less sensitive to vinyl chloride than is the rat. The two major epidemiological studies have linked liver angiosarcoma with chronic exposure to high (> 200 ppm) levels of vinyl chloride, but no angiosarcomas were observed below 200 ppm. Furthermore, workers with long-term occupational exposure to vinyl chloride levels which were low, but well in excess of 5 ppm, had no angiosarcoma or any other vinyl chloride related disease.

- The cancer risk of exposure to 5 ppm of vinyl chloride (the present Alberta standard) is low and well within the risks of ordinary living. Thus, a work force of 200 people exposed to 5 ppm would be expected to suffer one liver angiosarcoma every 35,000 years.

- Further lowering the current 5 ppm standard will result in increased costs with no real health benefits.

- The contention of the Occupational Health and Safety Division that tumours observed below 10 ppm in rats have "biological significance" is clearly based on incorrect scientific methodology. Maltoni and the world-wide vinyl chloride experts, in a thorough analysis of the results, drew no such conclusions in the Paris Symposium of November, 1979.

In conclusion, while our position is still 10 ppm, we believe that your officials exhibited sound judgement in 1975 when they set the standard at 5 ppm, judgement which has been vindicated by subsequent scientific data. We submit, with respect, that this standard is reasonable, practicable and scientifically supportable and that it should be retained.

.../3

Our reason for raising this issue with you goes beyond our immediate concern for vinyl chloride per se. It stems from our belief that Alberta's approach to regulation of all chemicals should have a sound scientific basis.

Yours very truly,

J.M. Hay
President

Attachments: 1. Dow Position Statement on Alberta's Proposed Occupational Standard for Vinyl Chloride

2. Critical Appraisal of Data Presented by the Alberta Occupational Health and Safety Division

Appendix: "Analyzing the Daily Risks of Life" by Richard Wilson

JMH/ib

July 30, 1981

DOW CHEMICAL OF CANADA, LIMITED: POSITION STATEMENT ON ALBERTA'S PROPOSED OCCUPATIONAL STANDARD FOR VINYL CHLORIDE

After carefully examining the position taken by the Occupational Health and Safety Division at our last meeting and again reviewing the scientific information on vinyl chloride, we remain of the opinion that the current vinyl chloride occupational exposure limit of 5 parts per million (ppm) as an 8 hour average is an appropriate standard supported by the relevant toxicological and epidemiological data. This conclusion is supported by the following evidence.

1. Liver angiosarcoma is the tumour type caused by low vinyl chloride exposure in rats, other tumours being formed only at higher concentrations (Maltoni, 1979). In humans, liver angiosarcoma is the only tumour type firmly associated with vinyl chloride exposure (Fox, 1977 and Equitable Environmental Health, Inc., 1978). Thus, protecting against angiosarcoma of the liver would protect against all other tumour types.

2. The most recent data of Maltoni indicate a possible increase in angiosarcoma in rats exposed to 10 ppm of vinyl chloride but no statistically significant increase in tumours was found in experimental rats exposed below 10 ppm (Maltoni, 1979). The tumours observed below 10 ppm are not due to vinyl chloride exposure and they have no toxicological significance because:

   - The number of tumours from the vinyl chloride exposed rats is not statistically different from the unexposed (control) animals;
   - The tumours do not exhibit a dose-response relationship to vinyl chloride exposure;
   - At a 1979 toxicology conference in Paris, vinyl chloride experts agreed with Maltoni's statement:

     > "None (or no increase) of these specifically vinyl chloride-related tumours has been observed at doses below 10 ppm." (Maltoni, 1979).

3. It is clearly evident from the metabolic studies by Buchter et al. (Buchter, 1980) and Gehring et al. (Gehring, 1979) that rodents are more susceptible to tumour induction by vinyl chloride than humans and that human experience should be given higher importance than animal studies. Dr. David Rall, the director of the U.S. National Institute of Environmental Health Sciences, has stated that the available data on liver angiosarcoma in rats (Rall, 1977 and 1979):

   > "project a cancer incidence rate 500 times higher than has so far been reported in man by epidemiological studies for vinyl chloride."

4. Extensive human experience and epidemiological studies based on forty years' manufacture and use of vinyl chloride should be given precedence over animal studies:

   - Angiosarcoma of the liver has been linked with chronic exposure to high (>200 ppm) levels of vinyl chloride in the two major human epidemiological studies (Fox, 1977 and Equitable Environmental Health, Inc., 1978); no angiosarcomas were observed below 200 ppm.

   - Groups of workers chronically exposed to levels which were low, but well in excess of 5 ppm, were found to have no angiosarcoma (Ott, 1975 and Chiazze, 1977).

   - No liver damage or any other vinyl chloride related diseases are associated with a chronic occupational exposure to vinyl chloride of 50 ppm or less (Kramer, 1972).

   Thus the current Alberta occupational standard of 5 ppm is more than adequate to protect the health of workers.

5. The cancer risk of workers exposed for 35 years to 5 ppm of vinyl chloride has been estimated to be 3.7×10^{-6} (Gehring, 1979 and Reitz, 1979), which is well within the risks of everyday life (see Appendix). This means that, for example, a work force of 200 people exposed to 5 ppm of vinyl chloride would be expected to suffer one liver angiosarcoma every 35,000 years.

6. The world-wide mortality statistics from liver angiosarcoma among polyvinyl production workers are shown in the accompanying graph. Despite increase in production of vinyl chloride and polyvinyl chloride and the associated increase in the number of exposed workers, the total world-wide angiosarcoma mortality has declined since 1976. The 10 Canadian cases all occurred in a vinyl plant at Shawinigan, Quebec, which was built in 1941.

Having reviewed the relevant toxicological, epidemiological, and medical data, we conclude that the current vinyl chloride occupational exposure limit of 5 parts per million (ppm) as an 8 hour time-weighted average is reasonable, practicable and scientifically supportable and should be retained.

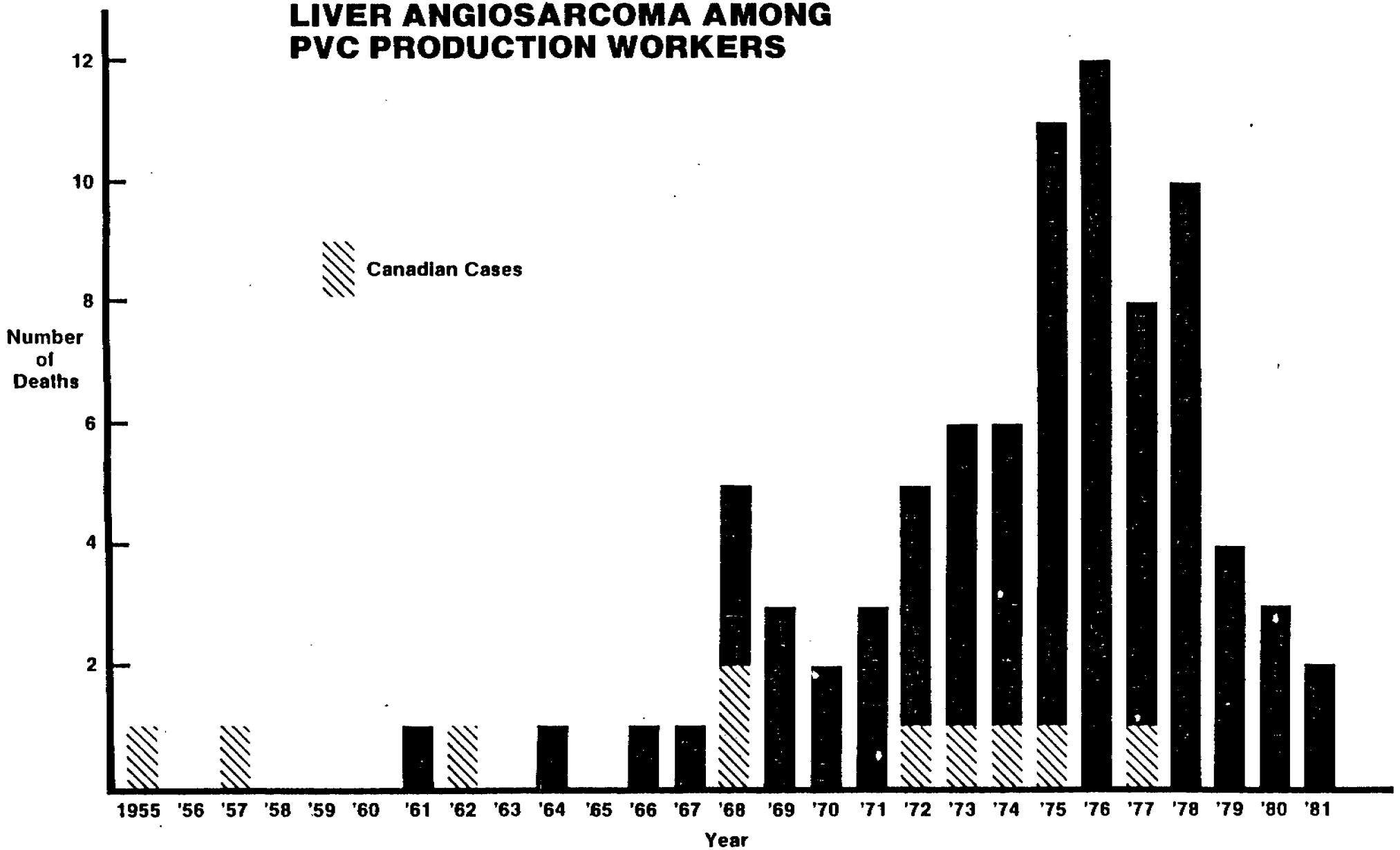
A critical appraisal of the data presented by the Alberta Occupational Health and Safety Division follows.

William Chen

William L. Chen, B.Sc., Ph.D.
Diplomate, American Board of Toxicology
Corporate Toxicologist

R&S 114814

WORLD-WIDE MORTALITY FROM LIVER ANGIOSARCOMA AMONG PVC PRODUCTION WORKERS



Based on Data Compiled by Dr. John Stafford, 1981.

[DR. IRVING R. TABERSHAW IS THE ORIGINAL INVESTIGATOR OF THE HUMAN EPIDEMIOLOGICAL STUDY OF POLYVINYL CHLORIDE/VINYL CHLORIDE WORKERS IN THE UNITED STATES, AND THE AUTHOR OF THE PRESTIGIOUS TABERSHAW-GAFFEY REPORT. DOW CANADA'S POSITION STATEMENT ON VINYL CHLORIDE AND THE ACCOMPANYING DOCUMENTS WERE SENT TO DR. TABERSHAW FOR HIS CRITICAL COMMENT. THE FOLLOWING TELEX IS HIS RESPONSE].

JULY 27, 1981
DOCTOR WILLIAM L. CHEN
CORPORATE RESEARCH AND DEVELOPMENT
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SARNIA, ONTARIO
CANADA

YOUR SUMMARY OF THE CURRENT STATUS OF THE SCIENTIFIC INFORMATION RELEVANT TO A HEALTH STANDARD FOR VINYL CHLORIDE IS BOTH CONCISE AND ACCURATE. I AGREE WITH YOUR CONCLUSION THAT 5 PARTS PER MILLION (PPM) ON AN 8 HOUR TIME WEIGHTED AVERAGE (TWA) IS ADEQUATE TO PROTECT THOSE WORKERS EXPOSED TO THIS CHEMICAL.

VINYL CHLORIDE AT HIGH DOSES CAN DEFINITELY BE CAUSALLY RELATED TO ANGIO-SARCOMA BUT A FIRM CONCLUSION THAT IT IS A MULTIPLE CARCINOGEN IS NOT JUSTIFIED FROM OUR STUDY. OUR DATA SUGGESTED THE POSSIBILITY AND WAS STATED AS AN "HYPOTHESIS" TO GENERATE INTEREST IN FOLLOW-UP. SUBSEQUENT EPIDEMIOLOGICAL STUDIES HAVE NOT CONFIRMED THIS CONCLUSION AT LOW DOSES. OUR DATA HAD NO ENVIRONMENTAL MEASUREMENTS, BUT BEAR IN MIND THAT THE STANDARD FOR VINYL CHLORIDE IN THE UNITED STATES AT THAT TIME WAS 500 PPM. IT APPEARS TO ME FROM THE DATA ON WORKERS RECENTLY EXPOSED TO VINYL CHLORIDE THAT 5 PPM (TWA) IS A SAFE LEVEL.

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U.S.A.

July 30, 1981

CRITICAL APPRAISAL OF THE DATA PRESENTED BY THE ALBERTA OCCUPATIONAL HEALTH AND SAFETY DIVISION.

I. General Comments

1. Commenting on stastical analysis as applied to liver angiosarcoma, Maltoni has observed:

> "There may be smaller differences between exposed and control groups which do not reach statistical significance, while these differences could still have meaning from an oncological point of view, (particularly in the case of tumours which are unfrequent (sic) in the animal colony). Therefore, the most important data should be commented on both in light of the performed statistical analysis and with a biological approach."

This point appears to have been misunderstood by Division scientists, who have incorrectly applied Maltoni's argument to tumours which have positive spontaneous background rates such as leukemia and mammary tumours. Tumours which have such background rates can be judged to be treatment-related only if a dose repsonse is demonstrated and a statistically significant increase over the control animals, as well as over the historical background tumour incidence for the strain of animal used, is established.

2. It makes no biological or toxicological sense to ignore dose-response in the assessment of whether a tumour type is treatment related. Dose-response does not mean simply an increase over control, because the control may be low. It means an increasing tumour response over some dose range. Maltoni has carried out experiments with vinyl chloride exposures ranging from 30,000 ppm to 1 ppm and all the results should be taken into account in assessing dose-response.

3. Simply counting excess tumours over the control and interpreting them as treatment related is meaningless since the comparison of two untreated control groups will also show some tumours to be numerically increased and some numerically decreased. This simply reflects minor biological variations in the spontaneous tumour rate.

4. Other regulatory agencies such as the U.S. Occupational Health and Safety Administration, U.S. Environmental Protection Agency, and the International Agency for Research on Cancer all require statistical signficance and dose-response to be demonstrated before the tumour is to be considered as chemical exposure related.

5. Liver angiosarcoma is the tumour type caused by low vinyl chloride exposure in rats, other tumours being found only at higher concentrations (Maltoni, 1979). In humans, liver angiosarcoma is the only tumour type firmly associated with vinyl chloride exposure (Fox, 1977 and Equitable Environmental Health, Inc., 1978). Thus, protecting against angiosarcoma of the liver would protect against all other tumour types.

6. The most recent data of Maltoni indicate a possible increase in angiosarcoma in the rats exposed to 10 ppm of vinyl chloride but no statistically significant increase in tumours was found in experimental rats exposed below 10 ppm. At a 1979 toxicology conference in Paris, vinyl chloride experts agreed with Maltoni's statement:

> "None (or no increase) of these specifically vinyl chloride-related tumours has been observed at doses below 10 ppm." (Maltoni, 1979).

7. It is clearly evident from the metabolic studies by Buchter et al. (Buchter, 1980) and Gehring et al. (Gehring, 1979) that rodents are more susceptible to tumour induction by vinyl chloride than humans and that human experience should be given higher importance than animal studies. Dr. David Rall, the director of the U.S. National Institute of Environmental Health Sciences, has stated that the available data on liver angiosarcoma in rats (Rall, 1977 and 1979):

> "project a cancer incidence rate 500 times higher than has so far been reported in man by epidemiological studies for vinyl chloride."

8. Extensive human experience and epidemiological studies based on forty years' manufacture and use of vinyl chloride should be given precedence over animal studies:

   - Angiosarcoma of the liver has been linked with chronic exposure to high (>200 ppm) levels of vinyl chloride in the two major human epidemiological studies (Fox, 1977 and Equitable Environmental Health, Inc., 1978); no angiosarcomas were observed below 200 ppm.

- Groups of workers chronically exposed to levels which were low, but well in excess of 5 ppm, were found to have no angiosarcoma (Ott, 1975 and Chiazze, 1977).

- No liver damage or any other vinyl chloride related diseases are associated with a chronic occupational exposure to vinyl chloride of 50 ppm or less (Kramer, 1972).

Thus the current Alberta occupational standard of 5 ppm is more than adequate to protect the health of workers.

II. Specific Comments

Specific comments regarding the pertinent data presented by the Alberta Occupational Health and Safety Division on May 14, 1981 are presented in the following pages.

July 30, 1981

SLIDE 1 (by the Occupational Health and Safety Division)

C. EXPERIMENT BT 15

ONE MOST RELEVANT TO US SINCE IT EXAMINES INHALATION EXPOSURES FROM 25 - 1 PPM.

PROTOCOL: EXPOSURE BY INHALATION TO VC IN AIR AT 25, 10, 5 AND 1 PPM; 4 HR/DAY, 5 DAY/WK., 52 WKS. RESULTS AFTER 147 WKS. ALL ANIMALS OBSERVED UNTIL SPONTANEOUS DEATH.

11 MAJOR TUMOUR TYPES SCREENED FOR:

MAMMARY

ZYMBAL GLAND

LEUKEMIAS

NEPHROBLASTOMAS

ANGIOSARCOMAS — LIVER / OTHER SITES

ANGIOMAS & FIBROANGIOMAS — LIVER / OTHER SITES

HEPATOMAS

FORESTOMACH EPITHELIAL

SKIN CARCINOMAS

SUBCUTANEOUS SARCOMAS

ENCEPHALIC TUMOURS — NEURO BLASTOMAS / OTHER TYPES

OTHERS

COMMENT ON SLIDE 1

MALTONI HAS CARRIED OUT EXPERIMENTS WITH VINYL CHLORIDE EXPOSURES RANGING FROM 30,000 PPM TO 1 PPM. EXPERIMENT BT 15 MUST NOT BE CONSIDERED ON ITS OWN, BUT IN CONJUNCTION WITH ALL THE OTHER STUDIES.

July 30, 1981

SLIDE 2 (by the Occupational Health and Safety Division)

TOTAL NUMBER TUMOURS (25-1 PPM VC)

	TUMOUR TYPE	MALE	FEMALE	TOTAL # TUMOURS	GREATER PROPORTION THAN CONTROLS
1.	MAMMARY	35	230	265	+
2.	ZYMBAL GLAND	5	3	8	-
3.	LEUKEMIAS	13	6	19	-
4.	NEPHROBLASTOMA	1	0	1	+
5.	ANGIOSARCOMA (LIVER)	1	5	6	+
6.	ANGIOSARCOMA (OTHER SITES)	2	0	2	+
7.	ANGIOMA/FIBROANGIOMA (LIVER)	0	1	1	+
8.	ANGIOMA/FIBROANGIOMA (OTHER SITES)	5	1	6	+
9.	HEPATOMAS	0	0	0	-
10.	FORESTOMACH EPITHELIAL TUMOURS	0	0	0	-
11.	SKIN CARCINOMAS	2	0	2	+
12.	SUBCUTANEOUS SARCOMAS	7	1	8	+
13.	ENCEPHALIC	2	1	3	-
14.	OTHERS	39	31	70	-
	TOTAL	112	279	391	+
	TOTAL NON-MAMMARY TUMOURS	77	49	126	+

TOTAL NO. TREATED MALE RATS EXAMINED = 236; TOTAL NO. CONTROLS EXAMINED = 60

TOTAL NO. TREATED FEMALE RATS EXAMINED = 240; TOTAL NO. CONTROLS EXAMINED = 60

R&S 114821

COMMENT ON SLIDE 2

IT IS SCIENTIFICALLY INVALID TO SIMPLY ADD UP ALL THE TUMOURS FROM FOUR DIFFERENT TREATMENT GROUPS AS WELL AS MALE AND FEMALE ANIMALS AND THEN COMPARE WITH THE SINGLE CONTROL GROUP. THIS IS CONTRARY TO ACCEPTED PRACTICE IN BIOLOGICAL RESEARCH. FURTHERMORE, SIMPLY COUNTING EXCESS TUMOURS OVER THE CONTROL WITHOUT STATISTICAL SIGNIFICANCE AND DOSE-RESPONSE IS MEANINGLESS (SEE GENERAL COMMENTS 1 TO 4).

SLIDE 3 (by the Occupational Health and Safety Division)

EXPERIMENT B1 15: EXPOSURE OF SPRAGUE DAWLEY RATS TO VCM IN AIR AT 25, 10, 5, 1 PPM; 4 HR/DAY, 5 DAY/WK., 52 WK. RESULTS AFTER 147 WK. (END OF EXPERIMENT).

MAMMARY TUMOUR DATA

VCM Conc. (ppm)	Sex	Total No. Tumours	No. Tumours/ Tumour Bearing Animal	% Malignant (A)	% Benign (B)	No. Fibroma/ Fibroangioma	No. Carcinoma	No. Sarcoma	No. Carcino-Sarcoma
25	M	11	1.1	9	91	10	1	0	0
	F	60	1.9	28	72	43	15	0	2
10	M	6	1.0	0	100	6	0	0	0
	F	50	1.8	35	65	39	21	0	0
5	M	10	1.2	0	100	10	0	0	0
	F	66	2.1	35	65	43	22	1	0
1	M	8	1.1	13	87	7	0	1	0
	F	54	1.7	26	74	40	12	0	2
0 (control)	M	8	1.2	13	87	7	1	0	0
	F	33	1.6	18	82	27	6	0	0

(A) % MALIGNANT = $\frac{\text{No. CARCINOMAS + CARCINOSARCOMAS + SARCOMAS}}{\text{TOTAL NO. TUMORS}}$

(B) % BENIGN = $\frac{\text{No. FIBROMAS + FIBROANGIOMAS}}{\text{TOTAL NO. TUMOURS}}$

R&S 114823

COMMENT ON SLIDE 3

THE INCREASE OF MAMMARY TUMOURS IN FEMALES IN THE VINYL CHLORIDE EXPOSED GROUP OVER THE CONTROL GROUP, WHILE IT MAY BE STATISTICALLY SIGNIFICANT, IS MOST LIKELY DUE TO AN ARTIFACT IN MALTONI'S EXPERIMENTAL DESIGN. THE CONTROL ANIMALS, UNLIKE THE EXPOSED ANIMALS, WERE NEVER INTRODUCED INTO THE INHALATION CHAMBERS. IT IS WELL KNOWN THAT INCREASE IN STRESS CAN INDUCE AN INCREASE IN MAMMARY TUMOURS IN FEMALE RODENTS (RILEY, 1975). THEREFORE THE OCCURRENCE OF MAMMARY TUMOURS IN THE EXPOSED GROUP IN MANY OF MALTONI'S STUDIES CAN BE EXPLAINED ON THE GROUND OF AN ENORMOUS DIFFERENCE IN STRESS CONDITIONS BETWEEN EXPOSED ANIMALS (IN INHALATION CHAMBERS) AND NON-EXPOSED CONTROL ANIMALS (NOT IN INHALATION CHAMBERS).

SLIDE 4 (by the Occupational Health and Safety Division

INTERPRETATIONS

1. NUMBER OF MAMMARY TUMOURS HIGHER IN FEMALES THAN MALES AND > FEMALE CONTROLS.

2. MALE TUMOURS ALMOST ALL BENIGN. GREATER NUMBER THAN CONTROLS TO 5 PPM LEVEL.

3. FEMALE TUMOURS PREDOMINANTLY BENIGN.

4. NUMBER OF MALIGNANT TUMOURS IN FEMALES > MALES AND CONTROLS TO 1 PPM LEVEL.

5. FEMALES EXHIBITED A GREATER NO. TUMOURS/TUMOUR BEARING ANIMAL THAN CORRESPONDING CONTROLS TO 1 PPM LEVEL.

COMMENT ON SLIDE 4

THE INCREASE OF MAMMARY TUMOURS IN FEMALES IN THE VINYL CHLORIDE EXPOSED GROUP OVER THE CONTROL GROUP, WHILE IT MAY BE STATISTICALLY SIGNIFICANT, IS MOST LIKELY DUE TO AN ARTIFACT IN MALTONI'S EXPERIMENTAL DESIGN. THE CONTROL ANIMALS, UNLIKE THE EXPOSED ANIMALS, WERE NEVER INTRODUCED INTO THE INHALATION CHAMBERS. IT IS WELL KNOWN THAT INCREASE IN STRESS CAN INDUCE AN INCREASE IN MAMMARY TUMOURS IN FEMALE RODENTS (RILEY, 1975). THEREFORE THE OCCURRENCE OF MAMMARY TUMOURS IN THE EXPOSED GROUP IN MANY OF MALTONI'S STUDIES CAN BE EXPLAINED ON THE GROUND OF AN ENORMOUS DIFFERENCE IN STRESS CONDITIONS BETWEEN EXPOSED ANIMALS (IN INHALATION CHAMBERS) AND NON-EXPOSED CONTROL ANIMALS (NOT IN INHALATION CHAMBERS).

IT IS WELL KNOWN THAT SPRAGUE-DAWLEY RATS, THE STRAIN USED BY MALTONI, EXHIBIT AN EXCEPTIONALLY HIGH SPONTANEOUS BACKGROUND INCIDENCE OF MAMMARY TUMOURS.

SLIDE 5 (by the Occupational Health and Safety Division

EXPERIMENT BT 15: SUMMARY OF MOST SIGNIFICANT TUMOURS

SEX	MALIGNANT	TUMOUR TYPE	LOWEST CONCENTRATION OBSERVED AT*
M	+	LEUKEMIA	25
M	+	NEPHROBLASTOMA	25
M	+	ANGIOSARCOMA (LIVER)	25
M	+	ANTIOSARCOMA (OTHER SITE)	10
M	-	ANGIOMA/FIBROANGIOMA (OTHER SITE)	10
M	+	SUBCUTANEOUS SARCOMA	5
M	±	OTHERS	5
M	±	MAMMARY	5
M	+	SKIN CARCINOMA	1
F	-	ANGIOMA/FIBROANGIOMA (LIVER)	25
F	-	ANGIOMA/FIBROANGIOMA (OTHER SITE)	10
F	+	ANGIOSARCOMA (LIVER)	10
F	+	ZYMBAL GLAND CARCINOMA	5
F	±	MAMMARY	1
F	+	SUBCUTANEOUS SARCOMA	1

*I.E. LOWEST CONCENTRATION AT WHICH INCIDENCE WAS GREATER THAN CONTROLS

COMMENT ON SLIDE 5

THE TUMOURS PRESENTED IN THIS SLIDE, WITH THE EXCEPTION OF ANGIOSARCOMA, ARE NOT DUE TO VINYL CHLORIDE EXPOSURE AND THEY HAVE NO TOXICOLOGICAL SIGNIFICANCE BUT RATHER REFLECT MINOR VARIATIONS IN SPONTANEOUS TUMOUR RATE. THIS IS BECAUSE:

- THE NUMBER OF TUMOURS FROM THE VINYL CHLORIDE EXPOSED RATS IS NOT STATISTICALLY DIFFERENT FROM THE UNEXPOSED CONTROL ANIMALS;

- THE TUMOURS DO NOT EXHIBIT A DOSE-RESPONSE RELATIONSHIP TO VINYL CHLORIDE EXPOSURE;

- THE CONTROL ANIMALS, UNLIKE THE EXPOSED ANIMALS, WERE NOT SUBJECTED TO THE STRESS OF BEING PLACED IN THE INHALATION CHAMBERS.

CLEARLY, THE DIVISION HAS DERIVED FROM MALTONI'S DATA MORE CONCLUSIONS THAN MALTONI HIMSELF. AT A 1979 TOXICOLOGY CONFERENCE IN PARIS, VINYL CHLORIDE EXPERTS AGREED WITH MALTONI'S STATEMENT:

> "NONE (OR NO INCREASE) OF THESE SPECIFICALLY VINYL CHLORIDE-RELATED TUMOURS HAS BEEN OBSERVED AT DOSES BELOW 10 PPM". (MALTONI, 1979).

SLIDE 6 (by the Occupational Health and Safety Division

INTERPRETATIONS:

1. MALES HAD GREATEST PROPORTION OF TUMOURS AT VC CONCENTRATIONS $\leq$ 25 PPM.

   MOST SIGNIFICANT TUMOURS WERE : ANGIOSARCOMA (OTHER SITES)
   ANGIOMA/FIBROANGIOMA (OTHER SITES)
   SUBCUTANEOUS SARCOMA
   OTHERS
   MAMMARY
   SKIN CARCINOMA

2. MOST SIGNIFICANT TUMOURS FOR FEMALES: ANGIOMA/FIBROANGIOMA (OTHER SITES)
   ANGIOSARCOMA (LIVER)
   ZYMBAL GLAND CARCINOMA
   MAMMARY
   SUBCUTANEOUS SARCOMA

3. NO SIGNIFICANT INCIDENCE FOR MALES OR FEMALES AT VC CONCENTRATIONS $\leq$ 25 PPM FOR HEPATOMAS, FORESTOMACH EPITHELIAL TUMOURS, AND ENCEPHALIC (NEUROBLASTOMAS - OTHER TYPES).

COMMENT ON SLIDE 6

THE MOST RECENT DATA OF MALTONI INDICATE A POSSIBLE INCREASE IN ANGIOSARCOMA IN RATS EXPOSED TO 10 PPM OF VINYL CHLORIDE BUT NO STATISTICALLY SIGNIFICANT INCREASE IN TUMOURS WAS FOUND IN EXPERIMENTAL RATS EXPOSED BELOW 10 PPM (MALTONI, 1979). THE TUMOURS OBSERVED BELOW 10 PPM ARE NOT DUE TO VINYL CHLORIDE EXPOSURE AND THEY HAVE NO TOXICOLOGICAL SIGNIFICANCE BECAUSE:

- THE NUMBER OF TUMOURS FROM THE VINYL CHLORIDE EXPOSED RATS IS NOT STATISTICALLY DIFFERENT FROM THE UNEXPOSED (CONTROL) ANIMALS;

- THE TUMOURS DO NOT EXHIBIT A DOSE-RESPONSE RELATIONSHIP TO VINYL CHLORIDE EXPOSURE;

- THE CONTROL ANIMALS, UNLIKE THE EXPOSED ANIMALS, WERE NOT SUBJECTED TO THE STRESS OF BEING PLACED IN THE INHALATION CHAMBERS.

CLEARLY, THE DIVISION HAS DERIVED FROM MALTONI'S DATA MORE CONCLUSIONS THAN MALTONI HIMSELF. AT A 1979 TOXICOLOGY CONFERENCE IN PARIS, VINYL CHLORIDE EXPERTS AGREED WITH MALTONI'S STATEMENT:

> "NONE (OR NO INCREASE) OF THESE SPECIFICALLY VINYL CHLORIDE-RELATED TUMOURS HAS BEEN OBSERVED AT DOSES BELOW 10 PPM". (MALTONI, 1979).

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SLIDE 7A (by the Occupational Health and Safety Division

HEALTH EFFECTS OF VINYL CHLORIDE (CH_2CHCL)

ACUTE EXPOSURE (HIGH CONCENTRATION)

ANAESTHETIC EFFECTS.

EUPHORIA, FEELING OF DROWSINESS, SLEEPINESS AND NARCOSIS

CHRONIC EXPOSURE

1. ACROOSTEOLYSIS
   (RAYNAUD'S PHENOMENON)

2. IMMUNOLOGIC CHANGES

3. PULMONARY CHANGES
   OBSTRUCTIVE AIRWAY DISEASE (?PVC)

4. HEPATIC DAMAGE
   THICKENED LIVER CAPSULE
   SUB-CAPSULAR FIBROSIS
   PROGRESSIVE PORTAL FIBROSIS
   SINUSOIDAL CELL STIMULATION

July 30, 1981

SLIDE 7B (by the Occupational Health and Safety Division)

MUTAGENETIC AND TERATOGENIC EFFECTS

NOT CONCLUSIVE

DOSE RESPONSE CURVE WITH PERIPHERAL LYMPHOCYTE CHANGES

CARCINOGENICITY

HAEMANGIOSARCOMA OF THE LIVER

COMMENT ON SLIDE 7A AND 7B

ALL THE HUMAN HEALTH EFFECTS PRESENTED IN THIS SLIDE ARE RELATED TO HIGH VINYL CHLORIDE EXPOSURES -- AT LEVELS MANY ORDERS OF MAGNITUDE HIGHER THAN THE CURRENT ALBERTA OCCUPATIONAL STANDARD OF 5 PPM. STUDIES HAVE DEMONSTRATED THAT LESSER EXPOSED POPULATIONS HAVE SHOWN NONE OF THESE HEALTH EFFECTS.

- ANGIOSARCOMA OF THE LIVER HAS BEEN LINKED WITH CHRONIC EXPOSURE TO HIGH (>200 PPM) LEVELS OF VINYL CHLORIDE IN THE TWO MAJOR HUMAN EPIDEMIOLOGICAL STUDIES (FOX, 1977 AND EQUITABLE ENVIRONMENTAL HEALTH, INC., 1978); NO ANGIOSARCOMAS WERE OBSERVED BELOW 200 PPM.

- GROUPS OF WORKERS CHRONICALLY EXPOSED TO LEVELS WHICH WERE LOW, BUT WELL IN EXCESS OF 5 PPM, WERE FOUND TO HAVE NO ANGIOSARCOMAS (OTT, 1975 AND CHIAZZE, 1977).

- NO LIVER DAMAGE OR ANY OTHER VINYL CHLORIDE-RELATED DISEASES ARE ASSOCIATED WITH A CHRONIC OCCUPATIONAL EXPOSURE TO VINYL CHLORIDE OF 50 PPM OR LESS (KRAMER, 1972).

SLIDE 8A (by the Occupational Health and Safety Division)

EPIDEMIOLOGY

1) DEFINITE CAUSE/EFFECT RELATIONSHIP -

VCM EXPOSURE - LIVER ANGIOSARCOMA

2) SUGGESTED CAUSE/EFFECT RELATIONSHIP -

VCM EXPOSURE - RESPIRATORY TRACT CANCER
- OTHER LIVER CANCER
- BRAIN CANCER

SLIDE 8B (by the Occupational Health and Safety Division

TUMOURS PRESENTLY CORRELATED TO VC EXPOSURE (BY INHALATION) ON EXPERIMENTAL RODENTS AND MAN

SPECIES	ANGIO-SARCOMAS OF LIVER	TUMOURS OF BRAIN	TUMOURS OF LUNG	LYMPHOMAS AND LEUKEMIAS	HEPA-TOMAS	ANGIO-SARCOMAS	NEPHRO-BLASTOMAS	SEBA-CEOUS CUTA-NEOUS CAR-CINOMAS	OTHER CUTA-NEOUS EPI THELIAL TUMOURS	MAM-MARY CAR-CINOMAS	FORE-STOMACH PAPILLOMAS AND ACAN-THOMAS
RAT	+	+		+	+	+	+	+	+	(+)	+
MOUSE	+		+			+		+	+	+	(+)
HAMSTER	+		(+)	+		(+)			+		+
MAN	+	(+)	(+)	(+)	(+)	+					

MALTONI, C. 1977. COLD SPRING HARBOUR CONFERENCE ON CELL PROLIFERATION

R&S 114835

COMMENT ON SLIDE 8A AND 8B

IN THE TWO LARGE SCALE EPIDEMIOLOGICAL STUDIES (FOX, 1977 AND EQUITABLE ENVIRONMENTAL HEALTH INC., 1978) ANGIOSARCOMA OF THE LIVER WAS THE ONLY TUMOUR TYPE SHOWN TO BE ASSOCIATED WITH VINYL CHLORIDE PRODUCTION. NO OTHER TUMOURS WERE DEMONSTRATED TO BE STATISTICALLY INCREASED AT A CONFIDENCE LEVEL OF 0.05.

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APPENDIX

Analyzing the Daily Risks of Life

by Richard Wilson

In our most trivial activities we incur risks. These hazards can be quantified and compared, but can they be eliminated from our lives?

Anthony Schultz

The world seems a very hazardous place. Every d. the newspapers announce that some chemical has been found to be carcinogenic, or some catastrophic accident has occurred in some far-off place. This leads some of us to hanker after a simpler world where there are fewer risks to life. But does such a world really exist?

If we look back at the world of a century ago, we find that expectation of life was 50 years; now it is 70 years. Therefore the sum of all the risks to which we are now exposed must be less than it was. We find that many of the large risks of the last century have been eliminated, leaving us conscious of a myriad of small risks, most of which have always existed.

The moment I climb out of bed I start taking risks. As I drowsily turn on the light I feel a slight tingle; my house is old with old wiring and there is a small risk of electrocution. Every year 500 people are electrocuted in the United States. I take a shower, and as I reach for the soap, I wonder about the many chemicals it contains. Are they all good for the skin, as the advertisements claim? My clothes have been cleaned with the best bleaching detergent. Most bleaching agents contain a chemical that fluoresces slightly in the sunlight to enhance the whiteness. Does this make bleaches carcinogenic?

I ponder this risk as I walk down to breakfast, taking care not to fall upon the stairs. Falls kill 16,000 people per year — mostly in domestic accidents. Shall I drink coffee or tea with my breakfast? Both contain caffeine, a well-known stimulant which may be carcinogenic. I have a sweet tooth; do I use sugar which makes me fat and gives me heart disease, or saccharin which we now know causes cancer? It is better to abstain.

After breakfast I make a sandwich for lunch. My son likes peanut butter. But improperly stored peanuts can develop a mold which produces a potent carcinogen — aflatoxin. In Africa and Southeast Asia, where aflatoxin appears more frequently, it has been blamed for numerous cases of liver cancer. In our (less natural) society storage facilities are better, so the risk is less — but it is not zero.

I prefer meat. But Americans, like other prosperous people, eat too much meat. It is not certain, but a meat-heavy diet probably contributes to cancer of the colon.

I live seven miles from work and can commute by car, by bicycle, or by bus. Which has the lowest risk? To travel by bicycle would keep my weight down, and bicycle riding does not cause pollution — but

statistics show that it is more likely to involve me in an accident. And since a bicyclist is unprotected, fatal accidents are also frequent on a bicycle. A car would be safer, but a bus is safest. I am happy that I no longer have to choose between a horse and a canoe; both are more dangerous (per mile) than a bicycle.

As I approach Boston, I see the urban haze caused by air pollution. There are toxic parts of air pollution which are not visible, as well. The risk to life caused by air pollution is high. Asthma victims have known this for a long time and fled the industrialized eastern United States for the purer air of the West. A press release from a government laboratory states that air pollution kills 20,000 people a year in the eastern United States. Air pollution, though still bad, has been reduced in most cities.

I remember the pea-soup London fogs of my youth caused by burning soft coal, where I could not see ahead ten feet; and the infamous week in December of 1952 where 3,000 people died from air pollution in four days.

I go to a committee meeting in a small, unventilated room. Although I don't smoke tobacco, half of the committee does, and I am exposed to the poison which causes 40 per cent of all cancers and kills 15 per cent of all Americans. Even though I breathe less tobacco smoke than my smoking colleagues, I often get a headache. One of my friends, who is more allergic than I, wears goggles at work.

At mid-morning I take a drink of water. The water tastes of chlorine, showing that the city's sanitation engineers use chlorine to kill microbes in the water. By such methods the country has nearly wiped out cholera and typhus. But the chlorine reacts with organic matter in the water to produce many known carcinogens. One of them, chloroform, is produced in a concentration of 100 parts per billion; enough to present a health hazard.

My office walls are brick and cinder block. Both contain radioactive materials, and radiation can increase my risk of cancer. One of these radioactive materials, radon, is a gas which is not chemically active. It is released by the brick and I can breathe it, which accentuates the hazard. I could prevent the release of this radioactive gas by painting the walls with thick epoxy paint to seal them, but that would introduce another risk. As the epoxy paint cures, it emits gaseous chemicals which are themselves carcinogenic. Which is worse?

Radiation enters all of my life. State law requires that I have a regular chest x-ray to see whether I have tuberculosis and may convey that dread disease to my students. But this adds to my risk of cancer from radiation. Is it correct for society to demand that I accept this risk, even to protect the rest of society from a greater one?

I frequently travel to meetings. Should I go by car, bus, train, or airplane? Thirty years ago the statistics were clear; the airplane was far more dangerous than all the others, since many airplanes crashed. Now, for journeys of 1,000 miles or more, air travel is the safest. But airplane travel causes an often-ignored radiation hazard, exposure to cosmic radiation from outer space. Airplanes fly at 30,000 feet, and at that altitude cosmic radiation exposure is 40 times what it is at sea level. Even a vacation trip to the high altitudes of Colorado and Wyoming can increase cosmic ray exposure. Sunlight at these altitudes, and excessive exposure even at sea level, showers us with ultraviolet light, which causes skin cancer.

These are personal concerns, and it might be argued that they are of no concern to anyone else, since I can avoid some of them. But in doing so I may well cause problems for others in society.

In the bad old days of my childhood we burnt coal in the house. If I heat my house by electricity I will not personally pollute the air with the products of fossil fuel burning; but these may still be produced at the power plant. One hopes the electric company is more careful about these pollutants than my parents used to be.

Whether I burn the coal myself or let the electric utility company do so, coal miners must still go underground. Anyone who has read *How Green Is My Valley* knows that 100 years ago coal mining was one of the most dangerous occupations. Even though mine safety has improved, it still has hazards: 156 out of every 100,000 miners were killed in accidents in 1972 in the United States. Yet accidents are not the worst hazard of coal mining: 800 miners yearly contract the dread black lung disease — coal workers' pneumoconiosis — from inhaling coal dust. One quarter of all American miners working in 1977 will probably contract this disease during their lifetimes. As an environmentalist I hate to see the beautiful western states laid bare by strip mining; but do I have a right to allow miners to die by refusing to let them work above ground?

Our society has a quirk which is fostered by our news media. We are far more concerned with infrequent large accidents than with numerous small accidents which, in total, cause many more deaths.

Congress was prompted into insisting on better mine ventilation to prevent black lung disease only after a much smaller number were killed in a single accident. A single accident of a school bus receives more newspaper coverage than the thousands of children killed yearly in automobile accidents.

This obsession with large accidents is getting worse. We are apprehensive at the *thought* of a large accident in a nuclear power plant, although none has happened so far, and experts are optimistic that none will ever happen. Nor is the fear unique to nuclear power. We bring to the United States considerable quantities of liquefied natural gas (LNG) and worry about the possibility of the ship leaking and blowing up. LNG *has* caused problems in the past; 30 years ago, an LNG tank, one-tenth the size of modern ones, collapsed and killed 133 people. We now know why this tank collapsed, and new tanks will not collapse in the same way since the metal from which the tanks are made has been changed.

Comparing the Risks We Face

There are those who would try to eliminate all known risks and would try to force this by law. This sounds plausible, but it creates an incentive for ignorance, not an incentive for safety. Under this procedure if we do not know whether something is risky and close our eyes to the possibility of risk, no one will bother us. On the other hand, if we look carefully and find there is a risk — even though it is small — some regulatory agency may stop us.

It would be a better policy to try to measure our risks quantitatively, and to give an *upper limit* on a risk when there is uncertainty. Then we could compare risks and decide which to accept or reject. I suspect most of us would decide to reduce the largest risks first.

To compare risks we must calculate them. As I prepared the table on page 45, I realized that an increased risk of death of one in a million is often seen as acceptable, but people instinctively think about large risks. I list here several actions which increase the chance of death in any year by one in a million.

Of course, if the risk of death in one year is increased, the risk of dying from another cause in a later year is decreased. The average expectation of life is shortened. Accidents often occur early in life, and life may be shortened 30 years by a typical accident. Cancer, black lung, and bronchitis kill later in life, and life is shortened only about 15 years. Therefore, a risk of 0.000001 (or 10^{-6}) shortens life on the

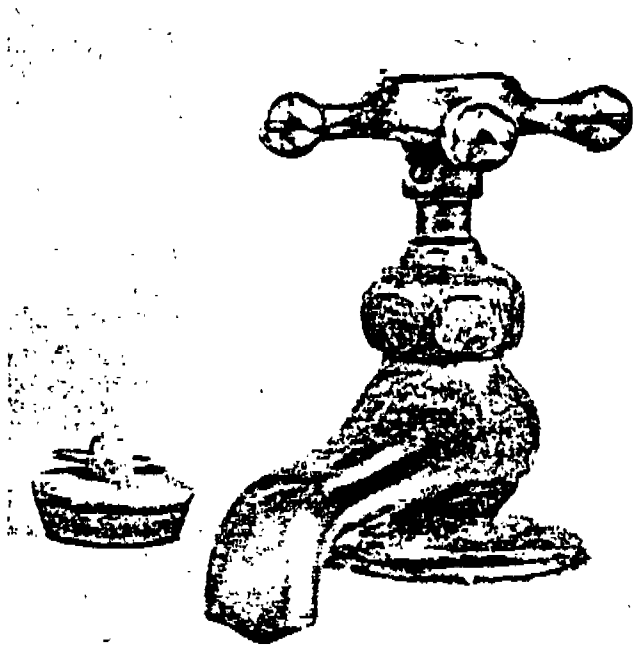

Photo Freda D Bodin, Stock, Boston

"The moment I climb out of bed I start taking risks . . . I take a drink of water . . . the chlorine reacts with organic matter to produce many known carcinogens."

Photo Michael Dubo, Stock, Boston

"As I approach Boston, I see the urban haze . . . Air pollution kills 20,000 people a year in the eastern U.S."

average by 30×10^{-6} years, or 15 minutes if it is an accident risk, 8 minutes if it is a risk of fatal illness.

I illustrate what this table means by calculating examples. In the United States 627 billion cigarettes were made in 1975. This is enough for 3,000 per person (including children), or a little less than half a pack a day. It is estimated that 15 per cent of all Americans (30 per cent of all smokers) die from lung or other cancers or heart disease due to smoking. We describe this as an average lifetime risk of 0.15. Dividing by the 70-year lifetime gives a yearly risk of 0.002 or 2×10^{-3}; dividing again by 3,000 gives a risk per cigarette of 0.7×10^{-6}. It is amusing to note that smoking a cigarette takes ten minutes and reduces the expectation of life by five minutes.

Human affairs are much more random than we like to think. One boy playing on a street can be killed by a passing car while his playmates are unharmed. All were equally at risk before the accident, but only one died. Similarly, one out of three lifetime smokers dies of cancer or heart disease because of the habit; the rest are unaffected and die of other causes. Moreover, those that die of cancer and heart disease do so at different ages. We have no way of telling which particular smokers will die of cancer, so we say that all are equally at risk.

It has been shown that those who smoke 40 cigarettes a day are ten times more likely to develop cancer as those who smoke four cigarettes a day. Perhaps there is a level of consumption where the risk becomes zero, but we cannot measure that low. It is easier to assume that every cigarette contributes the same amount to the total risk.

Brookhaven National Laboratory recently estimated that 20,000 Americans die every year from air pollution east of the Mississippi. This is partly due to sulphur emitted from burning coal and oil, and measurements suggest that the sulphate particulates spread themselves roughly uniformly over town and country. About 100 million Americans are exposed to this dirty air, so the average risk is 20,000/100,000,000 every year or 2×10^{-4} or 0.0002. Two days in New York City give a risk smaller by 2/365 or about 10^{-6} (one in a million).

Recent aircraft accident statistics tell us that aircraft in the United States carry passengers 100 billion passenger-miles every year and only about 100 people a year are killed in airplane crashes. This gives a risk of one in a million for one thousand miles of flight.

Professor Norman G. Rasmussen of M.I.T. made a study of nuclear reactor accident probabilities for

the Nuclear Regulatory Commission. He concluded that a reactor accident involving loss of life is very unlikely. The chance of an accident with more than 1,000 deaths is less than one in a 100 million per year of operation for each reactor. Most of these would be among the 20,000 or so people living within five miles of the reactor. So the probability of an individual living near a reactor being killed in a large accident is 1/2000 million. But those close by might also suffer in smaller accidents which, even though still unlikely, are more probable, leading to a risk of 1/50 million for persons living close to reactors.

Other more dangerous radiation hazards, such as natural radioactivity in brick, cosmic radiation, and diagnostic x-rays, are calculated by measuring the radiation dose and dividing it by the measured effect of large doses. The risks of these commonly accepted radiation hazards are far greater than those estimated for nuclear power.

I find these comparisons help me evaluate risks, and I imagine that they may help others do so, as well. But the most important use of these comparisons must be to help the decisions we make, as a nation, to improve our health and reduce our accident rate.

Taxing a Risk

Economists are fond of using taxation to control human affairs. Indeed, the invention of money by Croesus made a great simplification in the relationships in society. One suggestion, then, is to tax anyone who introduces a risk into society. This tax could pay for medical care, for compensating society for the loss of services, etc. The question arises: How much should the tax be? I suggest, as a basis for discussion, that this tax be at the rate of $1 million for every life that is lost by this extra risk, or one dollar for a risk of one in a million. Conversely, anyone that can save a life by an expenditure of $1 million must be encouraged to do so.

For example, the manufacturer who panders to the bad habit of cigarette smoking would pay an increased tax of 70 cents *per cigarette*. This is more than enough to pay the societal cost of cigarette smoking (hospital costs, fire hazards, reduced working time), which is variously estimated at from $1 to $2 per pack. Other taxes — five cents per diet soda — are less dramatic and might have to be accompanied by a tax of five cents on other sodas as well to prevent a switch to sugar.

Risks which increase chance of death by 0.000001*

Smoking 1.4 cigarettes	Cancer, heart disease
Drinking 1/2 liter of wine	Cirrhosis of the liver
Spending 1 hour in a coal mine	Black lung disease
Spending 3 hours in a coal mine	Accident
Living 2 days in New York or Boston	Air pollution
Travelling 6 minutes by canoe	Accident
Travelling 10 miles by bicycle	Accident
Travelling 300 miles by car	Accident
Flying 1000 miles by jet	Accident
Flying 6000 miles by jet	Cancer caused by cosmic radiation
Living 2 months in Denver on vacation from N.Y.	Cancer caused by cosmic radiation
Living 2 months in average stone or brick building	Cancer caused by natural radioactivity
One chest x-ray taken in a good hospital	Cancer caused by radiation
Living 2 months with a cigarette smoker	Cancer, heart disease
Eating 40 tablespoons of peanut butter	Liver cancer caused by aflatoxin B
Drinking Miami drinking water for 1 year	Cancer caused by chloroform
Drinking 30 12 oz. cans of diet soda	Cancer caused by saccharin
Living 5 years at site boundary of a typical nuclear power plant in the open	Cancer caused by radiation
Drinking 1000 24 oz. soft drinks from recently banned plastic bottles	Cancer from acrylonitrile monomer
Living 20 years near PVC plant	Cancer caused by vinyl chloride (1976 standard)
Living 150 years within 20 miles of a nuclear power plant	Cancer caused by radiation
Eating 100 charcoal broiled steaks	Cancer from benzopyrene
Risk of accident by living within 5 miles of a nuclear reactor for 50 years	Cancer caused by radiation

*(1 part in 1 million)

Photo Peter Menzel, Stock, Boston

"I go to a committee meeting in a small, unventilated room. Although I don't smoke tobacco . . . I am exposed to the poison which causes 40 per cent of all cancers and kills 15 per cent of all Americans."

These taxes might be earmarked to pay for risk reductions such as converting an existing sanitation system to using ozone instead of chlorine for sanitation, to avoid the production of chloroform.

Whether we quantify these risks or not, we must and do constantly make decisions about them. We do this as individuals, and our politicians make these decisions for us on a larger scale. What we are not doing, and need to do, is comparing the risks of various activities and then reducing the largest risks — which may not be the obvious ones.

After calculating these risks all day, I go home. I am still faced with decisions about risks. If I cook a meal in the microwave oven and the door doesn't fit tightly, I will be exposed to microwaves. It has recently been claimed that microwaves, even at low concentrations, give people nervous problems. Or I can use the gas stove, but the burning gas can fill my kitchen with both noxious carbon monoxide and nitrogen oxides.

Just as I go to bed I take a glass of beer. Alcohol causes cirrhosis of the liver and has been associated with oral and other cancers. However, the relaxing effect of the beer will reduce my stresses and permit a good night's sleep. This will prolong my life and is worth the risk.

The beer is in a green glass bottle which contains chromium, a small amount of which enters the beer. Chromium is a known carcinogen when ingested in moderate quantities, but it must not be avoided altogether because it is essential to life in small concentrations. How much chromium should I take to minimize the risk? Is the amount in the beer too much? Should I drink the beer from a plastic bottle? A plastic bottle suitable for beer has just been banned because a trace of the chemical from which the plastic was made could dissolve in the contents, and there is a suspicion that the chemical is carcinogenic.

I ponder this decision as I put on my pajamas. Are the pajamas inflammable? There is always a small risk of a fire starting while I am in bed. Is the risk of being burnt in a fire greater or smaller than the risk of cancer caused by a flame retardant such as TRIS?

I remember the truism "more people die in bed than anywhere else," so at least I'm in the right place.

Richard Wilson is professor of physics at Harvard University. Educated at Christ Church, Oxford, he received his Ph.D. in 1950. For many years he has been concerned with energy and the environment. He served on the National Science Foundation Physics Advisory Panel, as a consultant on nuclear power to the Attorney General's Office of the state of Maine, and as a consultant to the Nuclear Regulatory Commission. He is Assistant Editor of *Annals of Physics*.