MAY 1976

Appendix 2.

CANCER AND THE ENVIRONMENT

Let's Protect Our Earth

New Jersey Department of Environmental Protection

BRENDAN BYRNE
GOVERNOR

DAVID J. BARDIN
COMMISSIONER

RSV 0013461

STATE OF NEW JERSEY
DEPARTMENT OF ENVIRONMENTAL PROTECTION
DAVID J. BARDIN, COMMISSIONER
P. O. BOX 1390
TRENTON, N. J. 08625
609 292 2885

May 25, 1976

Honorable Brendan T. Byrne
Governor, State of New Jersey
State House
Trenton, New Jersey

Dear Governor Byrne:

The statistics on cancer mortality in New Jersey recently released by the National Cancer Institute call for a multi-Department effort to learn more about the possible causes, and to develop the necessary preventive and control programs which the citizens of New Jersey deserve.

The American Cancer Society estimates that approximately one out of every four Americans now living will eventually develop some form of cancer. Cancer for some time has been the second leading cause of death in the United States. Although the exact cause of observed cases of cancer still cannot be completely defined, there is a growing belief on the part of scientific and health experts that a majority of cancers of certain sites may be related to exposure to cancer-causing agents in the environment (as contrasted with such possible non-environmental causes as genetic tendencies or viruses). The environmental factors, based on growing evidence, may be particularly related to repeated and unprotected exposures to carcinogenic agents within the work environment, or sometimes amongst the general public near industrial facilities. Environmental factors also include exposure to trace contaminants in air, water and food, dietary habits, and solar radiation.

The New Jersey Department of Health has received a grant from the National Cancer Institute to conduct epidemiologic studies to further define the relationships between New Jersey's cancer mortality rates and occupational exposures. The goal is not only to further explain the high rates in New Jersey from certain kinds of cancer, but also to study living cancer patients and their work exposure histories. The development of programs of prevention and control will involve cooperation amongst several agencies of State government to include at least the Departments of Health, Labor and Industry, and Environmental Protection, as well as a number of federal agencies.

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Attached is a report prepared by my Department summarizing the extent of cancer deaths in New Jersey as compared with the nation, and outlining a long-term program to reduce the presence of cancer-causing agents in New Jersey's environment. The Department of Health will be furnishing you a report shortly which takes a somewhat different approach, with emphasis on the importance of a cancer registry, and which outlines the roles of the Departments of Health, Environmental Protection, and Labor and Industry in developing cancer protection and control programs based on the epidemiologic knowledge of what persons are most at risk, where they are located, and to what substances they have been exposed.

Your Administration has been most instrumental in urging Congress and the federal Executive branches to exercise their current authorities more actively, and where necessary, as in the case of the National Cancer Institute and the National Institute of Occupational Safety and Health to change their funding priorities, so as to put more emphasis on human health protection. Under your leadership, the agencies of state government will continue to work together on this vital issue affecting the public's health.

Faithfully,

David J. Bardin
Commissioner

Attachment

State of New Jersey
DEPARTMENT OF ENVIRONMENTAL PROTECTION
JOHN FITCH PLAZA, P. O. BOX 1390, TRENTON, N. J. 08625

May 24, 1976

Honorable David J. Bardin
Commissioner
Department of Environmental
Protection
Office of the Commissioner
P.O. Box 1390
Trenton, New Jersey 08625

Dear Commissioner Bardin:

Attached is a report on Cancer and the Environment which we prepared pursuant to your request. Part I of this report is a distillation and summary of the available literature on the nature and extent of cancer both in the United States and New Jersey, and a brief description of the potential causes and consequences of this broad family of diseases. Part II defines a program by which the Department of Environmental Protection, by building on its existing legal authorities and regulatory programs, could take sound steps toward minimizing the threat that cancer-causing agents and other toxic and hazardous pollutants pose to human health in New Jersey.

Sincerely yours,

Glenn Paulson, Ph.D.

Glenn Paulson, Ph.D.
Assistant Commissioner for Science

Peter W. Preuss

Peter W. Preuss, Ph.D.
Special Assistant to the Commissioner

Encl.

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Part I. Introduction: The Extent and Causes of Cancer

In 1974, cancer killed more than 350,000 Americans. About 1,000,000 people in the United States are currently under treatment for various forms of cancer. Each year, 900,000 new cases are diagnosed; of these, one-third are skin cancers which are troublesome, sometimes painful, but usually treatable; they have no significant impact on life expectancy. The remaining 600,000 cases, however, are serious and potentially fatal. The American Cancer Society estimates that 25 percent of the 213 million people now living in the United States will ultimately develop some form of cancer.

The economic and social impacts of cancer are immense. An estimated $1.8 billion is spent annually just for direct hospital care of cancer patients. Additional costs, such as doctor bills, medication, specialized treatment, etc. raise the direct expenditures for cancer to tens of billions of dollars each year.

Cancer is a group of diseases found in all races and ages of man, as well as in animals and even plants. It may be considered a single disease only in the sense that all cancers are characterized by the unrestrained or uncontrolled growth of cells, the basic unit of all life. This unrestrained cell growth results in tumors which invade and suppress the functions of normal tissues. If not treated, these tumors usually lead to death by eventually destroying the normal tissues.

There are different types of tumors, each of which behaves differently; thus the course of the disease can vary from person to person. Sometimes the specific type of tumor found may indicate a probable cause. For example, mesothelioma, a cancer of the chest or stomach lining, is associated almost exclusively with exposure to air-borne asbestos fibers.

Cancer in humans often develops only after a delay of years or even decades between the original (or the start of exposure) cause and the actual disease. This delay between cause and effect is termed the latency period. For mesothelioma, this period is 20-30 years.

Cancer affects people of all ages, but is predominantly a disease of people at or above middle-age. However, the exact picture is more complicated. For children between the ages of 5 and 14, cancer ranks second only to accidents as a cause of death. The distribution of cancer types among children is, however, quite different from that in adults. Leukemia is the most common form of cancer in children; it accounts for about 32 percent of the cancers in children under five. In adults, leukemia accounts for 1 percent of total cancer.

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Overall, total recorded deaths in the United States due to cancer have nearly doubled since 1935. Cancer incidence has remained relatively uniform for some organs of the body since 1930 but has changed markedly for others; both increases and decreases have occurred. The most dramatic changes are a nearly 20-fold increase in lung cancer for males, and a two-thirds decrease in stomach cancer in both males and females.

There are several partial explanations for the increase in cancer incidence over recent decades. Table I shows the change in the leading causes of death from 1900 to 1970. In 1900, cancer was eighth, with a rate of 64/100,000 population. In 1970, cancer ranked second as a cause of death, with a rate of 162/100,000. These increases are partially explained by the data presented in Chart I from the U.S. Department of Health, Education and Welfare. In this chart, line A represents the base number of cancer deaths in 1900. The increase between A and B is due to the growth of the population since then. The increase between B and C is due to the increase in the age of the population since 1900. However, the increase between C and D - the "residual increase" - cannot be explained by any of these factors. In our judgement, the factors we have described earlier play a major role in this "residual increase".

Most of the details of how and why cancers develop are poorly understood, so that the exact cause of all observed cancers cannot presently be defined. Three factors appear closely linked, both individually and in combination, with observed rates. These are hereditary factors, viruses and environmental factors. Current data strongly indicate that hereditary factors are involved in only a small portion of observed cancers; in previous years they were thought to be more significant. The role of viruses in human cancer is not well understood, although certain viruses may be involved in a few specific types of cancer.

The World Health Organization estimates that 60 to 90 percent of all cancers are related to environmental factors. They include, among others: the personal highly portable form of air pollution caused by cigarette smoking; exposure to cancer-causing agents in industry; exposure to exotic pollutants in air, water and food; and other causes such as dietary habits and solar and cosmic radiation.

The predominant role assigned to environmental factors is based in large part on the extreme variations in both incidence and distribution of different kinds of cancers throughout the world and within the United States. Such estimates are necessarily crude, giving rise to the relatively wide range (60 to 90 percent) for the role of environmental factors.

One set of observations which supports this estimate is the variation in cancer rates between natives of a nation and immigrants from that nation living in another country. Immigrants to another country generally have the same cancer rates as found in their nation of origin for the first generation only; succeeding generations more nearly approximate the rates of the adopted country.

Cancer rates vary significantly between regions in the United States. In general, states with high rates are the industrial states. Also, certain cancers vary on a geographical basis; for example, certain skin cancers are more common in the southern part of the country, where the amount of ultraviolet radiation reaching the earth from the sun is greater.

We cannot precisely estimate, for New Jersey or for the nation, the extent to which observed cancer rates and changes in those rates are caused by chemicals. Some cancer undoubtedly arises from natural sources, such as cosmic or solar radiation; our best judgement is that this is a small fraction. However, there are many examples of cancer directly traceable to industrial or other human activities that involve chemical or other cancer-producing agents. Liver cancer caused by vinyl chloride, mesothelioma caused by asbestos fibers, lung cancer caused by arsenic compounds, uranium dust, and other agents, a particularly debilitating lung cancer caused by beryllium, and bladder cancer caused by dye and chemical intermediates such as beta-naphthylamine are a few of the well-understood examples. Unfortunately even the primitive knowledge available at this time leads us to conclude that more such examples will be found in future years.

Exposure to carcinogenic materials is not confined to the industrial work place. In a preliminary assessment of drinking water carried out by the U.S. Environmental Protection Agency (EPA), known and suspected carcinogens (cancer-causing agents) were found in the two New Jersey sources of drinking water that were tested, the Passaic Valley Water Commission and Toms River Water Company. The significance of the levels that were found is not clear, however. This was a preliminary assessment; EPA made no attempt to measure other familes of known or suspected carcinogenic agents.

Thousands of known and suspected carcinogens have already been identified; more are being discovered. Vinyl chloride is one recent example. Biological testing has not kept pace with the need for information. Industry's record in developing and marketing new chemical substances exceeds the ability and resources of medical and scientific institutions to determine the carcinogenic or other toxic potentials of new chemicals.

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The Cancer Atlas published in 1975 by the National Cancer Institute (NCI) shows the death rates for cancer on a county by county basis for the 20 years from 1950 and 1969. For a large number of different cancers, the State of New Jersey as a whole and many counties within the state rank significantly above the national average.

At the end of this section, we have included selected data from the Cancer Atlas. Appendix I-A contains the death rates for each cancer evaluated by the NCI for the entire State of New Jersey and the United States as a whole. Both tabular and graphical rankings are included. Appendix I-B contains the county-by-county death rates for New Jersey, and, for comparison, the state-wide and national rates. Again, both tables and graphs are included.

TABLE I

Leading Causes of Death, 1900, 1950, and 1970

Rank	Cause of death	Deaths per 100,000 population	Percent of all deaths
	1900		
	(All causes)	(1,719)	(100)
1	Pneumonia and influenza	202.2	11.8
2	Tuberculosis (all forms)	194.4	11.3
3	Gastritis, etc	142.7	8.3
4	Diseases of the heart	137.4	8.0
5	Vascular lesions affecting the central nervous system	106.9	6.2
6	Chronic nephritis	81.0	4.7
7	All accidents[1]	72.3	4.2
8	Malignant neoplasms (cancer)	64.0	3.7
9	Certain diseases of early infancy	62.5	3.6
10	Diphtheria	40.3	2.3
	Total		64
	1950		
	(All causes)	(955)	(100)
1	Diseases of the heart	364.4	38.7
2	Malignant neoplasms (cancer)	147.4	15.6
3	Vascular lesions affecting the central nervous system	107.3	11.3
4	All accidents[2]	51.9	5.5
5	Certain diseases of early infancy	37.0	3.9
6	Pneumonia and influenza	33.0	3.5
7	General arteriosclerosis	20.2	2.1
8	Diabetes mellitus	17.1	1.8
9	Congenital malformations	12.0	1.3
10	Cirrhosis of the liver	11.2	1.2
	Total		85
	1970		
	(All causes)	(945.3)	(100)
1	Diseases of heart	362.0	38.3
2	Malignant neoplasms (cancer)	162.8	17.2
3	Cerebrovascular diseases	101.9	10.8
4	Accidents	56.4	6.0
5	Influenza and pneumonia	30.9	3.3
6	Certain causes of mortality in early infancy[3]	21.3	2.2
7	Diabetes mellitus	18.9	2.0
8	Arteriosclerosis	15.6	1.6
9	Cirrhosis of the liver	15.5	1.6
10	Bronchitis, emphysema, and asthma	15.2	1.6
	Total		85

[1] Violence would add 1.4 percent; horse, vehicle, and railroad accidents provide 0.8 percent.

[2] Violence would add 1.5 percent; motor vehicle accidents provide 2.3 percent; railroad accidents provide less than 0.1 percent.

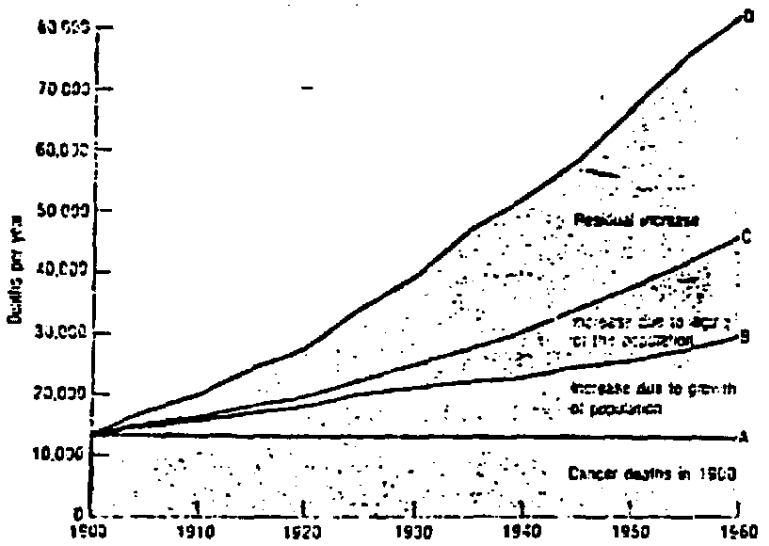
[3] Birth injuries, asphyxia, infections of newborn, ill-defined diseases, immaturity, etc.

Source: President's Science Advisory Committee Panel on Chemicals, *Chemicals and Health* (Washington, D.C.: Government Printing Office, 1973), p. 157; U.S. Department of Health, Education, and Welfare, Public Health Service, *Facts of Life and Death*, DHEW Pub. No. (HRA) 74-1222 (Washington, D.C.: Government Printing Office, 1974), p. 31.

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CHART I

Deaths from Cancer, 1900-1960
(for U.S. Death Registration Area of 1900)



Source: J.C. Bailar, H. King and M.J. Cohen, Cancer Rates and Risks, DHEW Pub. No. 1148 (Washington, D.C.: Government Printing Office, 1969), p. 6.

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Appendix 1 - A

Comparison of New Jersey and United States Cancer Mortality Rates by Cancer Site, 1950-1969

The accompanying table and graphs illustrate the mortality rates of the major cancer sites and show New Jersey's rank, among all states, for each site.

APPENDIX 1-A

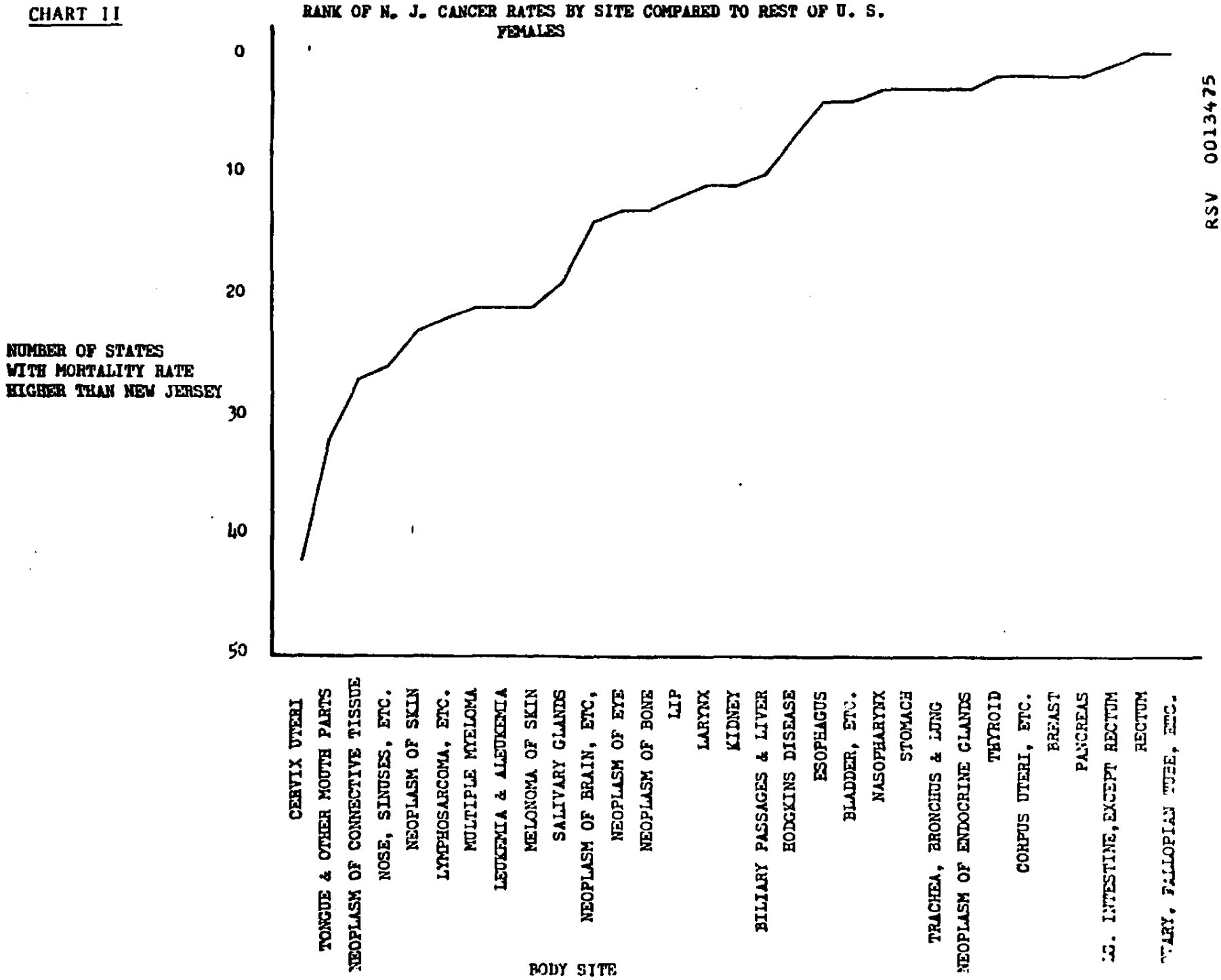
Comparison of New Jersey and United States Cancer Mortality Rates by Cancer Site: 1950-1969

Body Site	Mortality Rate/100,000 N.J.		U.S.		Upper Decile Rate - All States		Number of States With Mortality Rate Higher Than N.J.	
	M	F	M	F	M	F	M	F
LIP	.27	.02	.33	.03	.62	.10	39	12
SALIVARY GLANDS	.46	.24	.44	.22	.74	.36	12	19
NASOPHARYNX	.42	.15	.38	.12	.56	.23	6	3
TONGUE & OTHER MOUTH PARTS	5.52	.95	4.21	1.05	8.91	1.86	5	32
ESOPHAGUS	5.73	1.23	4.10	1.03	7.17	1.34	3	4
STOMACH	18.92	9.82	15.22	7.70	20.77	11.79	2	3
LARGE INTESTINE EXCEPT RECTUM	22.13	21.12	16.54	16.25	22.64	21.31	1	1
RECTUM	11.65	6.80	7.65	4.82	11.73	6.80	1	0
BILIARY PASSAGES & LIVER	5.93	5.77	5.16	5.34	6.05	6.80	3	10
PANCREAS	10.42	6.34	9.63	5.83	11.77	6.91	3	2
NOSE, NASAL CAVITIES, MIDDLE EAR & ACC. SINUSES	.42	.22	.43	.23	.70	.30	25	26
LARYNX	3.39	.27	2.54	.24	5.00	.50	4	11
TRACHEA, BRONCHUS & LUNG	46.83	7.22	37.98	6.29	51.97	7.96	2	3
BREAST	.37	30.61	.28	25.51	.43	31.53	2	2
CERVIX UTERI	---	6.29	---	7.79	---	11.76	---	42
CORPUS UTERI OTHER PARTS OF UTERUS	---	7.37	---	6.13	---	7.56	---	2
OVARY, FALLOPIAN TUBE & BROAD LIGAMENT	---	10.03	---	8.57	---	10.03	---	0
PROSTATE	17.77	---	17.84	---	21.12	---	26	---

Body Site	Mortality Rate/100,000 N.J.		Mortality Rate/100,000 U.S.		Upper Decile Rate - All States		Number of States With Mortality Rate Higher Than N.J.	
	M	F	M	F	M	F	M	F
TESTIS	.78	---	.83	---	1.11	---	30	---
KIDNEY	4.40	2.15	3.86	1.99	4.60	3.19	6	11
BLADDER & OTHER URINARY ORGANS	9.68	2.91	6.78	2.39	9.68	3.27	0	4
MELANOMA OF SKIN	1.59	1.11	1.55	1.11	2.29	1.72	19	21
NEOPLASM OF SKIN	1.28	.62	1.51	.71	3.15	1.49	33	23
NEOPLASM OF EYE	.22	.21	.23	.19	.34	.28	26	13
NEOPLASM OF BRAIN & OTHER PARTS OF NERVOUS SYSTEM	4.54	3.06	4.42	2.91	5.40	3.33	16	14
NEOPLASM OF THYROID	.48	.81	.43	.69	.67	.88	5	2
NEOPLASM OF OTHER ENDOCRINE GLANDS	.33	.26	.31	.20	.59	.36	10	3
NEOPLASM OF BONE	1.58	.91	1.35	.85	1.63	1.21	6	13
NEOPLASM OF CONNECTIVE TISSUE	.63	.45	.65	.48	.84	.62	20	27
HODGKIN'S DISEASE	2.41	1.58	2.29	1.32	3.00	1.89	13	7
LYMPHOSARCOMA & RETICULOSARCOMA, OTHER LYMPHOMAS	4.93	3.16	4.89	3.25	5.48	3.66	14	22
MULTIPLE MYELOMA	1.59	1.24	1.76	1.24	2.35	1.69	38	21
LEUKEMIA & ALEUKEMIA	8.74	5.73	8.81	5.74	10.00	6.58	22	21
ALL CANCERS	205.01	147.92	174.04	130.10	178.06	136.32	0	1

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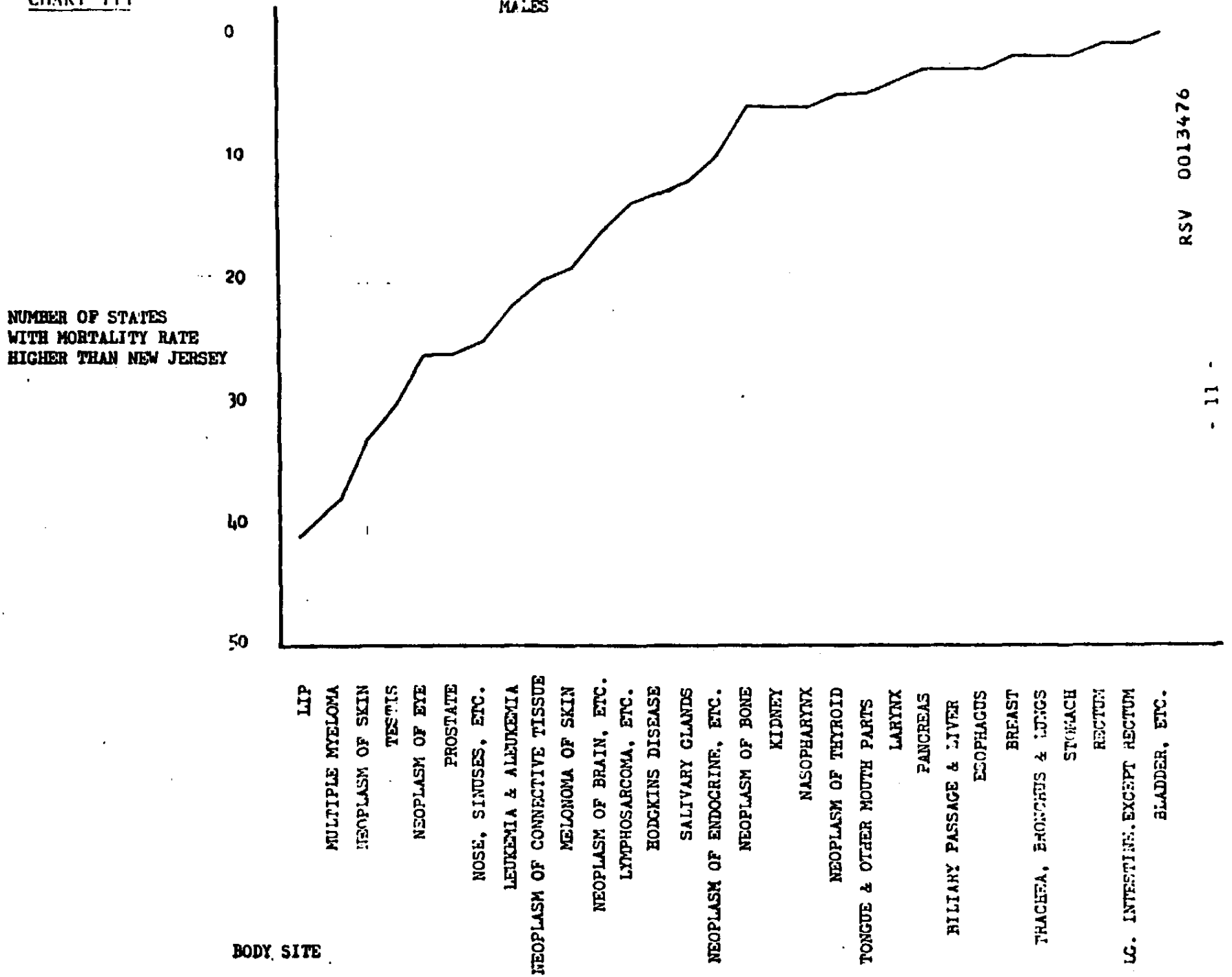
CHART II



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CHART III

RANK OF N. J. CANCER RATES BY SITE COMPARED TO REST OF U. S.
MALES



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APPENDIX 1-B

Cancer Mortality Rates 1950-1969

This Table presents the mortality rate for each specific cancer for each N.J. county. This rate is the number of deaths per 100,000 population, and is age-adjusted.

At the bottom of the table are the rates for the entire state, as well as the entire country, and the ratio between the two. This ratio gives a rough idea how much above or below the N.J. rate is compared to the national rate.

The accompanying chart presents these ratios graphically. It may be observed that for most types of cancer the ratio is greater than 1.0, indicating that the New Jersey rate is higher than the rate for the U.S.

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COUNTY	LIP 140		SALIVARY GLANDS 142		NASO-PHARYNX 146		TONGUE, FLOOR OF MOUTH, MESO-PHARYNX UNSPEC-IFIED 141-143 144-145 148		ESOPH-AGUS 150	
	M	F	M	F	M	F	M	F	M	F
ATLANTIC	.2	.0	.8	.0	.6	.0	4.8	1.6	5.3	1.3
BERGEN	.2	.0	.4	.2	.4	.1	5.4	.9	5.5	1.1
BURLINGTON	.5	-	.4	.1	.4	.1	5.5	.6	4.4	.7
CAMDEN	.4	-	.6	.3	.3	.1	5.7	.8	4.5	1.2
CAPE MAY	-	-	-	.3	-	.1	3.3	1.2	5.0	1.0
CUMBERLAND	.4	.1	.1	.1	.5	-	4.3	.8	4.3	1.0
ESSEX	.3	.0	.5	.3	.4	.2	6.1	.9	5.9	1.4
GLOUCESTER	.1	.1	.3	.3	.4	.3	4.0	.6	4.4	1.0
HUDSON	.3	-	.5	.3	.5	.2	7.8	1.1	8.2	1.7
HUNTERDON	.3	-	.2	-	.2	.3	5.1	.6	3.4	.5
MERCER	.2	-	.5	.4	.4	.3	6.6	1.0	6.6	1.1
MIDDLESEX	.3	-	.3	.3	.5	.3	5.5	.9	7.9	1.3
MONMOUTH	.2	.0	.4	.2	.4	.2	4.5	1.1	4.1	1.3
MORRIS	.1	.0	.4	.1	.5	-	3.6	.9	4.2	.9
OCEAN	.4	.1	.4	.2	.5	.1	3.0	1.2	4.5	1.3
PASSAIC	.4	.0	.5	.1	.7	.3	6.2	1.2	6.2	1.5
SALEM	.2	-	.2	.2	.6	-	2.4	.8	4.5	.8
SOMERSET	.1	-	.5	.2	.1	.2	3.8	.8	5.2	1.1
SUSSEX	.8	-	.2	.8	.4	.2	2.9	.7	5.2	1.0
UNION	.3	.0	.5	.2	.2	.1	5.3	1.1	5.2	1.1
WARREN	.1	-	.6	.1	.9	-	4.5	.4	5.4	.6
N.J.	.27	.02	.46	.24	.42	.15	5.52	.95	5.73	1.23
U.S.	.33	.03	.44	.22	.38	.12	4.21	1.05	4.10	1.03
RATIO NJ/US	.82	.67	1.05	1.09	1.10	1.25	1.31	.91	1.40	1.19

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COUNTY	STOMACH 151		LARGE INTESTINE 153		RECTUM 154		LIVER & BILIARY PASSAGES 155		PANCREAS 157	
	M	F	M	F	M	F	M	F	M	F
ATLANTIC	17.7	8.3	21.7	21.5	10.7	7.4	6.5	5.5	10.6	6.1
BERGEN	18.5	10.3	21.8	20.6	11.7	6.7	5.8	5.5	10.0	6.5
BURLINGTON	16.0	8.0	22.3	22.4	9.7	5.4	5.1	5.9	8.6	5.7
CAMDEN	17.4	8.2	23.9	23.3	10.8	6.9	5.2	6.2	10.0	6.3
CAPE MAY	17.9	8.1	21.9	18.4	9.9	5.3	5.9	4.8	12.0	6.2
CUMBERLAND	17.3	8.0	18.0	21.1	7.2	5.9	4.4	5.9	8.4	7.1
ESSEX	18.5	10.1	24.8	21.7	13.3	7.3	6.7	5.4	10.7	6.5
GLOUCESTER	16.0	6.9	19.0	20.8	9.4	6.8	5.6	6.4	10.9	5.3
HUDSON	23.1	12.1	23.9	21.3	14.1	8.0	7.1	7.0	10.3	6.4
HUNTERDON	17.5	8.0	14.8	22.4	11.5	7.2	5.1	6.4	9.1	5.5
MERCER	20.2	9.1	22.8	22.0	12.3	6.6	5.6	5.6	11.6	5.5
MIDDLESEX	22.9	12.1	23.4	20.4	11.2	6.4	6.8	6.6	11.0	6.8
MONMOUTH	17.0	8.8	22.8	22.2	9.8	6.2	4.7	4.9	10.7	6.5
MORRIS	15.5	7.4	18.3	19.9	9.6	6.2	5.6	4.5	10.0	4.8
OCEAN	15.0	7.9	19.0	20.6	8.8	5.9	5.5	6.4	8.3	5.1
PASSAIC	21.9	11.6	21.0	20.3	12.9	7.3	5.9	5.6	11.9	6.5
SALEM	15.1	6.6	16.1	20.3	8.2	6.1	4.3	6.4	10.3	7.7
SOMERSET	18.1	8.6	17.8	18.4	9.0	5.3	4.5	4.7	10.5	6.1
SUSSEX	15.2	9.1	16.2	17.4	11.7	6.8	4.9	4.6	7.4	6.2
UNION	19.1	10.6	22.2	20.9	11.8	6.4	5.8	5.8	10.7	6.9
WARREN	14.4	7.5	23.7	18.1	12.9	6.5	4.8	6.1	10.1	8.5
N.J.	18.92	9.82	22.13	21.12	11.65	6.80	5.93	5.77	10.42	6.34
U.S.	15.22	7.70	16.54	16.25	7.65	4.82	5.16	5.34	9.63	5.83
RATIO NJ/US	1.24	1.28	1.35	1.30	1.52	1.41	1.16	1.08	1.08	1.09

RSV 0013479

COUNTY	NOSE, NASAL CAVITIES, MIDDLE EAR, & ACCESSORY SINUSES 160		LARYNX 161		TRACHEA BRONCHUS LUNG 162-163		BREAST 170		CERVIX UTERI 171
	M	F	M	F	M	F	M	F	F
ATLANTIC	.5	.4	3.6	.3	42.5	8.1	.4	27.2	7.2
BERGEN	.3	.3	3.2	.1	46.4	7.0	.5	32.7	4.8
BURLINGTON	.5	.1	2.8	.1	41.8	6.5	.5	27.9	8.1
CAMDEN	.5	.1	3.4	.3	50.4	7.4	.3	29.6	6.6
CAPE MAY	.6	.2	2.6	.2	45.4	7.7	.6	26.2	9.2
CUMBERLAND	.5	.4	2.0	.1	42.9	7.2	.4	24.0	12.1
ESSEX	.3	.2	3.8	.3	44.7	7.2	.5	33.0	6.3
GLOUCESTER	.7	.2	2.2	.3	42.4	6.9	.7	28.8	7.2
HUDSON	.5	.2	4.4	.4	57.4	7.7	.3	30.7	6.1
HUNTERDON	1.2	.3	2.1	.5	38.7	6.7	.7	28.4	5.8
MERCER	.3	.2	3.2	.3	44.7	7.6	.5	26.5	7.3
MIDDLESEX	.5	.1	4.3	.2	54.5	6.6	.2	30.4	6.5
MONMOUTH	.3	.2	3.0	.3	48.6	8.5	.5	30.2	6.9
MORRIS	.3	.1	2.8	.3	41.1	6.1	.3	30.7	5.6
OCEAN	.3	.2	3.3	.2	48.6	7.3	.2	28.1	6.3
PASSAIC	.4	.3	3.5	.2	46.3	7.2	.1	29.1	5.8
SALEM	.4	-	1.9	.8	39.8	7.0	.2	30.7	10.3
SOMERSET	.6	.2	2.9	.4	44.2	8.6	.1	28.3	4.5
SUSSEX	.2	.3	3.4	.2	40.8	6.7	-	24.3	9.0
UNION	.5	.2	2.8	.3	42.0	6.9	.3	34.4	5.5
WARREN	.6	.3	3.2	.6	41.6	6.0	.3	26.6	9.8
N.J.	.42	.22	3.39	.27	46.83	7.22	.37	30.61	6.29
U.S.	.43	.23	2.54	.24	37.98	6.29	.28	25.51	7.79
RATIO NJ/US	.98	.96	1.33	1.12	1.23	1.15	1.32	1.20	.81

RSV 0013480

COUNTY	CORPUS UTERI, UTERUS, CHORIONEP-ITHELIOMA 172-173 174 M	F	OVARY, FALLOPIAN TUBE, BROAD LIGAMENT 175 M	F	PROSTATE 177 M	F	TESTIS 178 M	F	KIDNEY 180 M	F
ATLANTIC	8.2		8.9		16.2		.6		3.7	1.4
BERGEN	6.4		10.3		18.6		.8		4.6	2.5
BURLINGTON	7.6		9.1		19.2		.6		3.8	1.8
CAMDEN	8.5		8.3		17.5		.8		4.8	2.1
CAPE MAY	9.6		11.4		17.1		.1		3.3	2.0
CUMBERLAND	8.7		8.3		20.2		.4		3.7	.7
ESSEX	7.2		11.4		19.0		.9		4.5	2.1
GLOUCESTER	10.6		8.2		20.8		1.1		4.6	1.1
HUDSON	8.2		9.3		16.4		.7		4.0	2.1
HUNTERDON	6.5		11.6		17.6		.4		4.2	1.7
MERCER	7.2		9.9		17.3		.4		4.4	2.2
MIDDLESEX	6.9		8.8		17.9		.6		4.7	2.4
MONMOUTH	7.7		9.8		18.1		.7		4.6	1.8
MORRIS	6.6		10.1		15.6		1.0		4.3	2.2
OCEAN	7.1		8.7		15.0		.5		3.4	1.5
PASSAIC	6.6		10.7		16.6		.9		4.6	2.8
SALEM	9.1		8.5		21.0		1.8		4.0	2.1
SOMERSET	6.5		9.6		15.7		.8		4.3	2.4
SUSSEX	8.1		11.3		17.8		.4		3.9	4.1
UNION	6.7		11.3		19.3		.8		5.2	2.3
WARREN	9.1		11.1		15.7		2.4		3.7	2.4
N.J.	7.37		10.03		17.77		.78		4.40	2.15
U.S.	6.13		8.57		17.84		.83		3.86	1.99
RATIO NJ/US	1.20		1.17		1.0		.94		1.14	1.08

RSV 0013481

COUNTY	BLADDER & OTHER URINARY 181		MELANOMA SKIN 190		SKIN-OTHER 191		EYE 192		BRAIN & OTHER PARTS OF NERVOUS SYSTEM 193	
	M	F	M	F	M	F	M	F	M	F
ATLANTIC	8.7	3.2	1.9	1.3	1.6	.5	.2	.2	4.7	3.3
BERGEN	8.9	3.1	1.7	1.2	1.0	.5	.1	.2	5.4	3.1
BURLINGTON	9.2	2.8	1.3	1.0	.8	.3	.6	.2	4.6	3.3
CAMDEN	9.3	3.4	2.0	1.3	1.5	.6	.2	.2	4.0	3.0
CAPE MAY	11.5	3.5	.8	1.8	2.0	.9	.3	-	5.7	3.5
CUMBERLAND	9.0	2.3	1.3	.7	1.8	1.1	.2	.3	4.2	1.9
ESSEX	10.9	2.6	1.5	1.0	1.3	.7	.2	.1	4.8	3.4
GLOUCESTER	9.2	3.7	1.5	.9	1.3	.9	.1	.4	4.8	2.6
HUDSON	10.3	3.2	1.3	.9	1.1	.7	.3	.2	4.2	2.6
HUNTERDON	11.0	3.1	1.9	.4	1.1	.9	-	.3	3.2	2.1
MERCER	9.2	3.1	1.7	1.0	1.3	.5	.1	.2	4.4	2.6
MIDDLESEX	8.4	2.8	1.8	1.2	1.8	.6	.2	.3	3.9	3.8
MONMOUTH	10.4	2.6	1.5	1.1	1.1	.7	.2	.3	4.5	2.3
MORRIS	8.1	2.5	1.8	1.1	1.3	.7	.2	.2	4.4	3.7
OCEAN	11.2	3.2	1.5	1.2	1.2	.3	.2	.3	4.1	2.8
PASSAIC	9.0	2.7	1.7	1.0	1.4	.6	.2	.2	4.7	3.0
SALEM	16.1	3.6	2.6	1.2	2.1	.6	-	-	4.3	1.7
SOMERSET	7.9	3.0	1.9	.7	1.5	.3	.6	.2	4.7	3.5
SUSSEX	10.9	1.4	1.8	2.3	1.4	1.3	.6	-	4.5	2.5
UNION	9.9	2.8	1.4	1.2	1.1	.5	.2	.3	4.5	3.3
WARREN	7.9	3.2	1.2	1.4	1.7	1.0	.3	-	5.0	2.8
N.J.	9.68	2.91	1.59	1.11	1.28	.62	.22	.21	4.54	3.06
U.S.	6.78	2.39	1.55	1.11	1.51	.71	.23	.19	4.42	2.91
RATIO NJ/US	1.43	1.22	1.03	1.00	.85	.87	.96	1.11	1.02	1.05

RSV 0013482

COUNTY	THYROID GLAND 194		OTHER ENDOCRINE 195		BONE INC. JAW 196		CONNECTIVE TISSUE 197		HODGKINS DISEASE 201	
	M	F	M	F	M	F	M	F	M	F
ATLANTIC	.5	.8	.2	.2	1.0	.9	.6	.3	2.1	1.3
BERGEN	.5	.7	.3	.2	1.6	.9	.5	.4	2.5	1.7
BURLINGTON	.5	.7	.2	.3	1.4	1.1	.6	.4	2.2	1.6
CAMDEN	.6	.7	.3	.2	1.4	1.1	.9	.4	2.8	1.3
CAPE MAY	.3	1.3	.6	.5	.5	1.1	.3	.6	1.4	.6
CUMBERLAND	.8	.5	.2	.2	1.4	.7	.5	.5	1.5	1.7
ESSEX	.4	1.0	.5	.3	1.8	1.1	.8	.5	2.8	2.1
GLOUCESTER	.5	.9	.2	.1	1.7	1.0	.4	.9	2.8	1.6
HUDSON	.4	.7	.3	.2	2.2	.8	.5	.4	2.4	1.4
HUNTERDON	.3	1.0	-	.6	1.2	.6	.5	.7	1.8	1.1
MERCER	.5	1.4	.2	.2	1.6	1.1	.4	.5	2.5	1.5
MIDDLESEX	.5	.9	.3	.2	1.7	.8	.5	.4	2.2	1.8
MONMOUTH	.6	.7	.2	.3	1.3	1.0	.8	.6	2.2	1.3
MORRIS	.6	.5	.7	.4	1.3	.5	.6	.4	1.8	1.2
OCEAN	.5	.3	.2	.4	1.7	1.2	.6	.5	2.0	.9
PASSAIC	.3	.9	.2	.3	1.7	.8	.6	.4	2.2	1.7
SALEM	.4	.6	.4	.2	.9	2.2	.2	.6	2.5	.6
SOMERSET	.4	.4	.5	.2	.7	.4	.8	.4	1.9	1.2
SUSSEX	1.0	.3	.4	.4	1.0	.6	.6	.2	3.9	1.7
UNION	.5	.9	.4	.3	1.5	.8	.7	.4	2.8	1.8
WARREN	.5	.7	.5	.9	1.6	.8	1.2	.4	2.6	1.5
N.J.	.48	.81	.33	.26	1.58	.91	.63	.45	2.41	1.58
U.S.	.43	.69	.31	.20	1.35	.85	.65	.48	2.29	1.32
RATIO NJ/US	1.11	1.18	1.06	1.30	1.17	1.07	.97	.94	1.05	1.20

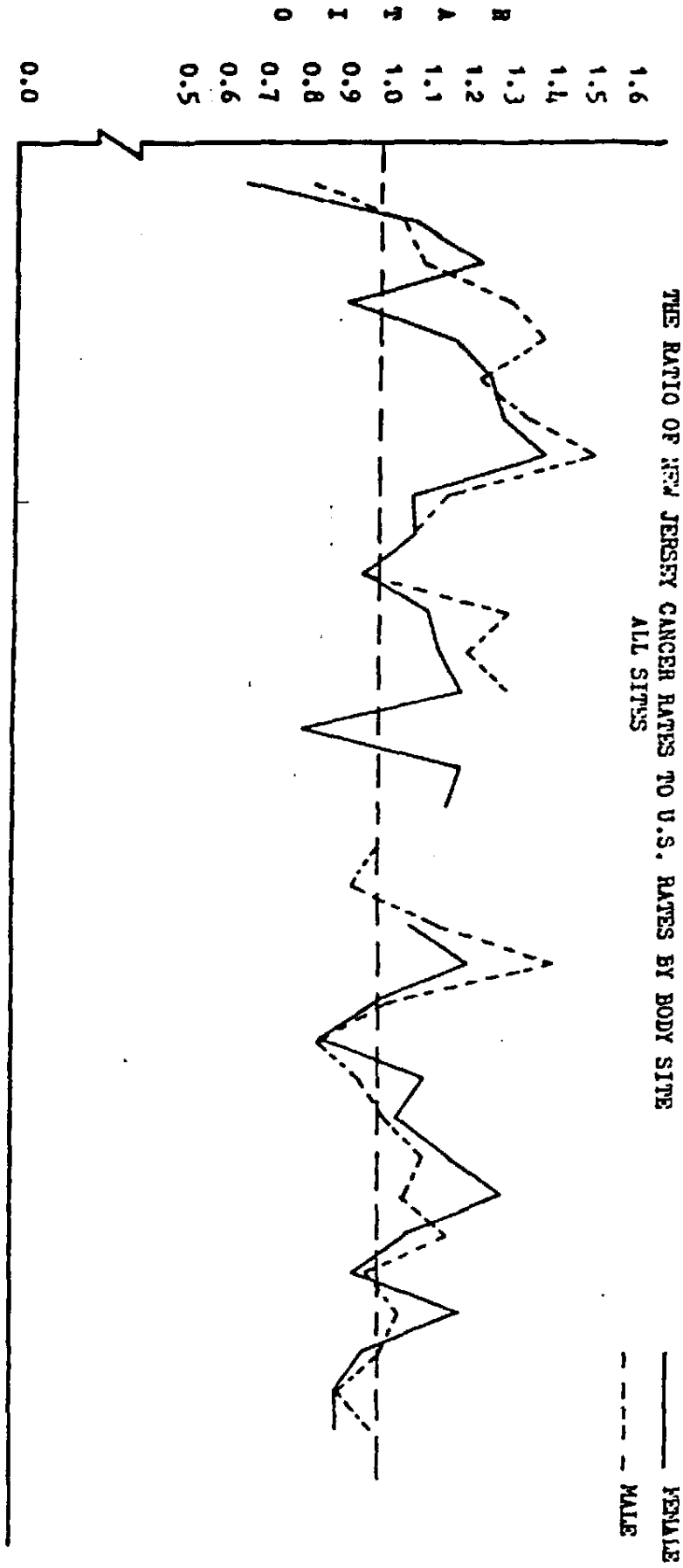
RSV 0013483

COUNTY	LYPHOSARCOMA, RETICULO-SARCOMA, MYCOSIS FUNGOIDES 200-202 205 M	F	MULTIPLE MYELOMA 203 M	F	LUKEMIA & ALEUKEMIA 204 M	F	ALL ICD'S NOT PREV. LISTED M	F	ALL 140-205 M	F
ATLANTIC	4.5	2.9	1.6	1.0	8.4	5.3	13.0	10.7	195.3	145.2
BERGEN	5.2	3.9	1.6	1.3	8.8	6.1	9.8	8.5	202.1	148.1
BURLINGTON	4.4	2.7	1.9	1.5	8.7	5.0	10.1	9.0	188.5	139.9
CAMDEN	3.7	2.6	1.7	1.1	8.1	5.8	11.8	10.7	204.7	148.4
CAPE MAY	4.3	2.5	1.8	.7	8.9	5.1	10.8	8.8	194.7	142.9
CUMBERLAND	5.6	3.5	1.3	.9	8.3	4.7	10.2	9.3	181.4	140.0
ESSEX	5.4	3.4	1.6	1.2	9.8	6.3	12.5	9.3	215.1	154.5
GLOUCESTER	4.4	2.7	2.1	1.5	9.0	4.7	10.6	8.0	191.1	141.8
HUDSON	4.5	2.7	1.2	1.1	9.0	5.1	14.8	10.6	231.8	153.5
HUNTERDON	4.1	2.8	2.0	.7	8.3	7.5	8.2	8.3	175.7	143.3
MERCER	6.0	3.2	1.2	1.1	7.8	6.1	10.8	10.0	205.4	145.2
MIDDLESEX	4.7	3.4	1.9	1.5	7.9	5.1	12.3	9.6	220.8	149.2
MONMOUTH	4.6	3.2	2.4	1.1	8.7	5.8	10.2	10.0	199.0	147.6
MORRIS	5.4	3.4	1.7	1.3	8.6	5.3	9.5	7.9	179.2	135.5
OCEAN	4.2	3.1	1.1	.9	7.8	5.9	12.7	8.3	185.5	137.2
PASSAIC	5.2	2.9	1.3	1.3	9.2	5.7	11.7	9.1	209.5	147.8
SALEM	5.5	2.9	.9	1.0	9.5	6.1	9.4	8.0	185.9	146.6
SOMERSET	3.8	2.4	1.7	1.9	7.5	6.0	10.9	9.1	182.8	135.7
SUSSEX	5.4	1.9	1.7	1.3	7.5	6.6	9.0	9.1	180.8	140.5
UNION	6.0	3.4	1.6	1.5	9.1	5.8	12.0	8.1	203.4	151.6
WARREN	2.8	3.9	1.8	1.7	7.6	5.9	9.9	10.0	189.3	147.7
N.J.	4.93	3.16	1.59	1.24	8.74	5.73	11.60	9.29	205.01	147.92
U.S.	4.89	3.25	1.76	1.24	8.81	5.74	12.64	9.89	174.04	130.10
RATIO NJ/US	1.01	.97	.90	1.00	.99	1.00	.92	.94	1.18	1.14

RSV 0013484

CHART IV

THE RATIO OF NEW JERSEY CANCER RATES TO U.S. RATES BY BODY SITE
ALL SITES



BODY SITE

LIP
SALIVARY GLANDS
NASOPHARYNX
TONGUE AND OTHER MOUTH PARTS
ESOPHAGUS
STOMACH
LARGE INTESTINE, EXCEPT RECTUM
RECTUM
BILIARY PASSAGES AND LIVER
PANCREAS
NOSE, NASAL CAVITIES, SINUSES, ETC.
LARYNX
TRACHEA, BRONCHUS AND LUNGS
BREAST
CERVIX UTERI
CORPUS UTERI, ETC.
OVARY, FALLOPIAN TUBE, ETC.
PROSTATE
TESTIS
KIDNEY
BLADDER & OTHER URINARY ORGANS
MELANOMA OF SKIN
NEOPLASM OF SKIN
NEOPLASM OF EYE
NEOPLASM OF BRAIN, ETC.
NEOPLASM OF THYROID
NEOPLASM OF OTHER ENDOCRINE GLANDS
NEOPLASM OF BONE
NEOPLASM OF CONNECTIVE TISSUE
HODGKINS DISEASE
LYMPHOSARCOMA & RETICULOSARCOMA, ETC.
MULTIPLE MYELOMA
LEUKEMIA & ALEUKEMIA

RSV 0013485

APPENDIX 1-C

Cancer Mortality Rates 1950-1969, All Cancers

County	NON-WHITE M	NON-WHITE F	WHITE M	WHITE F
Atlantic	220.1	150.8	195.3	145.2
Bergen	281.4	192.2	202.1	148.1
Burlington	216.3	141.0	188.5	139.9
Camden	228.0	177.1	204.7	148.4
Cape May	184.2	176.8	194.7	142.9
Cumberland	155.8	144.2	181.4	140.0
Essex	219.2	154.6	215.1	154.5
Gloucester	183.1	146.0	191.1	141.8
Hudson	289.7	197.3	231.8	153.5
Hunterdon	297.1	303.2	175.7	143.3
Mercer	203.5	150.1	205.4	145.2
Middlesex	279.0	208.2	220.8	149.2
Monmouth	225.4	135.3	199.0	147.6
Morris	248.6	170.1	179.2	135.5
Ocean	265.3	231.6	185.5	137.2
Passaic	271.5	182.7	209.5	147.8
Salem	232.0	161.9	185.9	146.6
Somerset	226.9	228.5	182.8	135.7
Sussex	913.3	465.4	180.8	140.5
Union	252.2	166.3	203.4	151.6
Warren	274.2	187.7	189.3	147.7
N.J.	230.33	163.41	205.01	147.92
U.S.	184.28	139.18	174.04	130.10
Ration NJ/US	1.25	1.17	1.18	1.14

RSV 0013486

Part II. Suggested Program for the Department of Environmental Protection

The Department of Environmental Protection (DEP) should devote more effort to determining and defining the dangers which New Jersey residents face as a result of exposure to carcinogens and other environmental contaminants. One important step would be to begin to inventory the presence and patterns of use of toxic chemicals in the environment. Another need is to expand DEP efforts in measuring the concentrations of carcinogenic and other exotic pollutants that may be present in the air, drinking water supplies, fish and other wildlife, surface and ground waters, and sediments.

Such a program would have the following elements:

A. Toxic Substances Inventory.

At present, DEP has permit systems which collect data on the emissions of certain relatively common pollutants into the air and water. These existing systems should be expanded to provide information on the emissions of all potentially carcinogenic and other toxic substances. The Solid Waste Administration is developing an equivalent system which would allow it to collect information at a level of detail similar to that obtainable through the air and water permit systems; this system will allow collection of data regarding emissions, treatment and disposal of solid and liquid toxic wastes.

A department-wide inventory would provide information as to the toxic chemicals currently in use in New Jersey, their amounts, and their emissions, as well as their ultimate pathways into the environment. This information will allow priorities to be established for monitoring and regulation. It will also help establish the criteria upon which regulations may be based, including, for example, toxicity, persistence in the environment, and amounts used in the state.

B. Monitoring.

There are a number of different ways in which the contamination of the environment may be monitored. Today, as in the past, DEP and other state and federal agencies monitor the environment on a routine basis for the presence of certain pollutants, the so-called "classical" pollutants. Examples of these pollutants are sulfur dioxide and carbon monoxide in air and sewage and phosphates in water.

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These monitoring systems have provided useful information on the overall pollution of these various media, and have enabled us to determine whether current efforts at cleansing the environment of these "classical pollutants" have been successful or not.

A second type of monitoring, which is much more difficult, complex, and time-consuming, is the monitoring of certain organic and inorganic materials in the environment. This monitoring is characterized by difficult and complex analytical procedures, by higher costs, and by the fact that relatively few samples can be taken and analyzed at any given time. It is exemplified by the recent preliminary assessment performed by the EPA to determine the concentrations of certain selected carcinogenic materials in drinking water which we mentioned in Part I, and by DEP's special monitoring for PCBs (polychlorinated biphenyls) in New Jersey's fish, shellfish, and sediments.

Historically, this more difficult type of monitoring has been performed only to a very limited extent in New Jersey; it should be expanded. Some specific areas in which additional monitoring should be performed are:

Air

1. Polynuclear aromatic hydrocarbons
2. Chlorinated hydrocarbons
3. Carcinogens in the vicinity of certain types of industrial facilities
4. Arsenic
5. Heavy metals (for example, mercury and lead)

Water, including drinking water, surface water, ground water, and leachate from landfills

1. Known or suspected carcinogens, e.g., chloroform, carbon tetrachloride
2. Chlorinated hydrocarbons, including PCBs and selected pesticides
3. Heavy metals

Fish, Wildlife and Sediments

1. Chlorinated hydrocarbons
2. Heavy metals
3. Organic compounds suspected of being carcinogenic and known to accumulate in biological organisms.

RSV 0013488

Finally, a third type of monitoring may be used as a tool to find toxic and carcinogenic materials in the environment which have not been previously discovered or even looked for. This is the most time-consuming and sophisticated of all monitoring, in that many analyses must be performed using many different techniques on even a single sample. The problems associated with the identification of each of the materials present are substantial, especially in view of the fact that many "exotic" contaminants are present in very small amounts. This type of work is on the border line between monitoring and pure research. It is characterized by the very few samples that are taken, by the sophisticated equipment required to perform the analyses, by the time required to carry out each analysis and by the high cost per analysis. This effort could be most useful, for example, to:

1. Determine the production of leachate from landfills and the subsequent contamination of ground and surface waters;

2. Determine interactions among various chemicals present in the environment;

3. Determine the formation of toxic or carcinogenic materials from non-toxic precursors in the environment; and

4. Determine the degradation of substances in the environment to form toxic or carcinogenic compounds.

The two areas of highest priority which should be monitored by this means are the determination of leachate production from landfills that have accepted toxic and hazardous chemical wastes in the past, and the determination of the extent to which ground waters in New Jersey have been contaminated by improper disposal of toxic materials. (This includes the contamination of groundwater resulting from improper housekeeping around industrial facilities, accidental spills of toxic materials, etc.)

C. The Treatment and Disposal of Toxic Wastes.

The treatment and disposal of toxic, hazardous, and carcinogenic wastes presents a serious problem for New Jersey. With its large concentration of industry, New Jersey produces a large quantity of such wastes annually, and has, in addition, accepted for disposal large quantities of such wastes from other states. Historically, these wastes have not been treated before disposal; they have been disposed of either through ocean dumping or in sanitary landfills. In the past, little was done to prevent these materials from being washed out of the landfills into the ground and surface waters of the state. Over the past five years some attempts have been made to control and limit the disposal of these types of wastes. However, the leachate resulting from wastes deposited many years ago will continue to be a problem into the future.

RSV 0013489

In order to avoid creating more problems in the future, a program should be established for the environmentally safe treatment and disposal of these types of wastes. Such a program would logically be a part of the overall solid waste management program being developed by the Solid Waste Administration. One of the first steps is to determine what kind of wastes are produced, how they are transported, and where they are treated and disposed; this is part of the inventory described above. This information will enable the Solid Waste Administration to establish priorities for its programs, and will allow judgements as to which materials should not be disposed of onto the land.

There are a number of toxic substances for which sufficient information is available now, so that a determination may be made as to the unsuitability of their disposal on land. In order for these wastes to be disposed of properly, however, alternative treatment or disposal methods must be made available. This will require the development of criteria to define environmentally sound disposal procedures. The first priority in this area is the development of performance criteria for incinerators that are or may be used to dispose of toxic and hazardous organic materials. These criteria must insure that the materials are burned completely, and that no toxic substances enter the air through the stack or into the water via water used for cleaning ("scrubbing") the exhaust gases. The presence of one or more such facilities in the State of New Jersey would provide a necessary alternative to help insure that environmentally dangerous chemicals may be safely treated. An example of the type of wastes which should be incinerated, rather than being disposed of by any other means, is the family of persistent chlorinated hydrocarbons, including pesticides, PCBs, and other similar materials.

Another problem is the disposal of sludges produced in sewage treatment facilities. These sludges often contain toxic substances from the effluents of industrial facilities that feed into the sewage plant; the sludges are presently disposed of in the ocean. The federal policy for disposal of such sludges calls for the phasing out of ocean dumping by 1981; as a result, alternative methods will have to be found. At present, several studies of local, state and regional scope are underway which are attempting to identify sound alternative methods. It will be necessary to carefully monitor the development of these alternate programs, as well as the facilities themselves, to insure that toxic substances present in the sludges are not inadvertantly released into the environment.

D. Reduction of Water-borne Discharges of Toxic Substances and the Protection of Drinking Water Supplies

RSV 0013490

The current system by which effluents into surface waters are controlled does not take into account many toxic and carcinogenic substances. In fact, even basic data as to the exact nature and composition of these effluents is lacking. The toxic substances inventory discussed above should provide the information necessary for development of a program to limit such toxic effluents. It will still be necessary, however, to monitor and analyze these effluents in order to insure that standards are not exceeded.

Many of the toxic substances currently emitted into the waters of New Jersey or those that have been emitted in the past will remain available in the surface or ground waters in their present toxic form for many years to come. It is therefore necessary to establish a monitoring program that will test potable water for the presence of such materials. Current systems for potable water treatment do not remove many of these substances from the water, and as a result, these substances can be ingested through the use of water for drinking, cooking, etc. The program that is developed here should include the establishment of effluent emission standards. These standards must limit the amount of the toxic substances that may be emitted into the environment, as well as limit the amounts discharged into sewage treatment facilities. New means should be developed for the pretreatment of effluents before discharge, as well as for proper disposal of the resulting wastes.

E. Reduction of Air-borne Discharges of Toxic Substances

The industries of New Jersey emit a large number of substances into the atmosphere. In addition to the ambient air monitoring discussed above, methods and procedures should be developed by which the stack effluents may be tested. This is needed to identify the substances that are being emitted, as well as to insure compliance with any standards that may be promulgated to limit such emissions.

F. Relation to Federal Laws and Activities.

The federal government currently has authority to limit the introduction of toxic substances into the environment through a variety of laws. Many of these laws provide the framework in which state regulatory programs function. Programs developed in New Jersey will have to strive to avoid duplication of federal roles and activities and to supplement and complement federal regulatory action.

RSV 0013491

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RSV 0013492

APPENDIX 2

New Jersey Cancer Mortality Rates 1950-1969

The maps in this appendix show the cancer mortality rates for selected body sites for white males and females in New Jersey.

Body sites chosen for these maps were those associated with the highest cancer mortality rates in the state; had counties which had death rates statistically higher than counties in the rest of the United States and where the mortality rate of the state was greater than the country. Specifically the criteria were:

1. mortality rate greater than 5 per 10,000
2. at least one county in the upper 10 percent of all counties in the U.S.
3. a mortality ratio of New Jersey to United States exceeding 1.0.

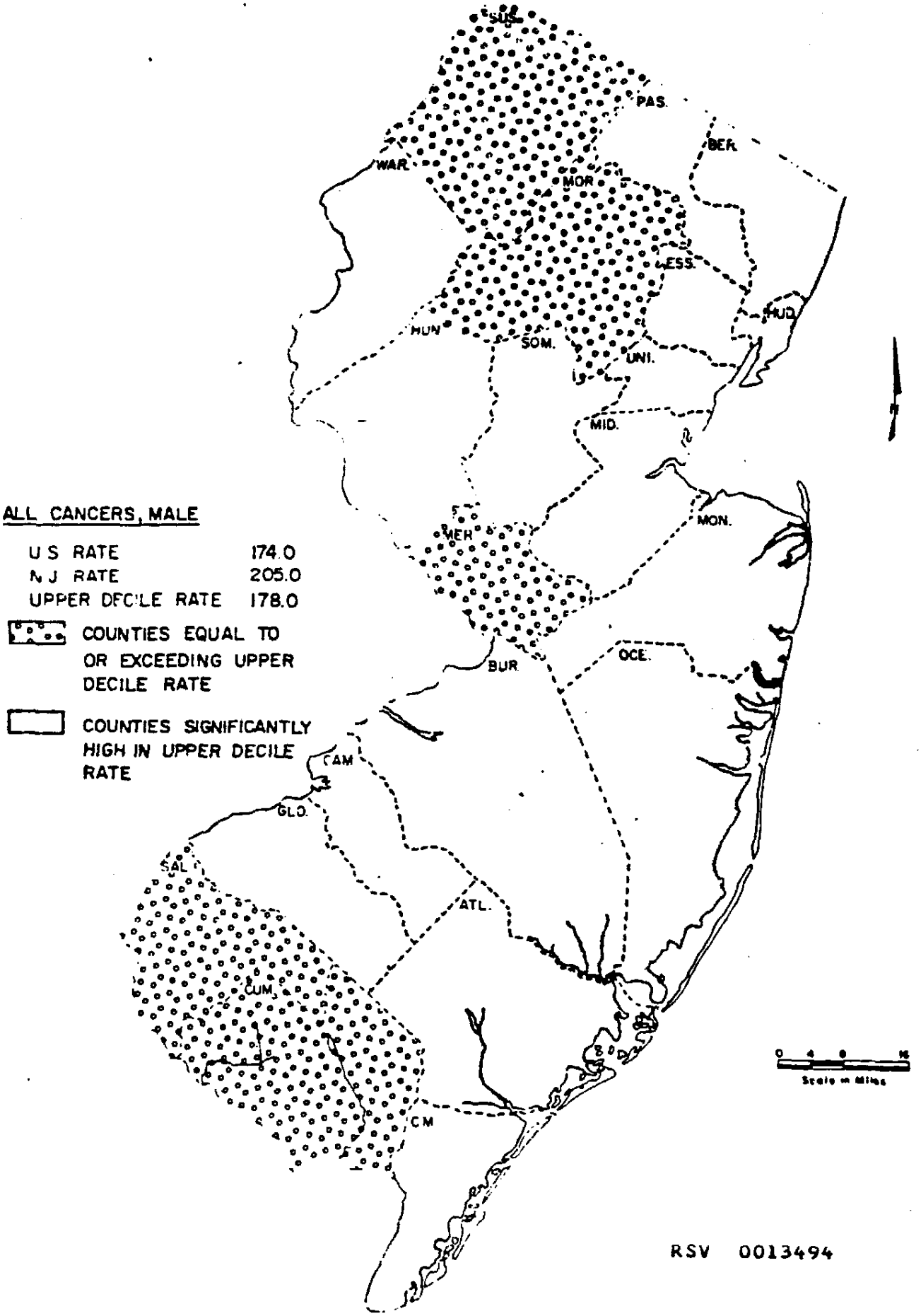
The sites which conformed to all of these criteria were:

All Malignant Neoplasms, Male and Female
Stomach, Male and Female
Large Intestine, Male and Female
Rectum, Male and Female
Pancreas, Female
Trachea, Bronchus and Lung, Male and Female
Breast - Female
Corpus Uteri
Ovary
Bladder Male (Female bladder is included for comparison although the N.J. rate is lower than 5)
Leukemia, Female

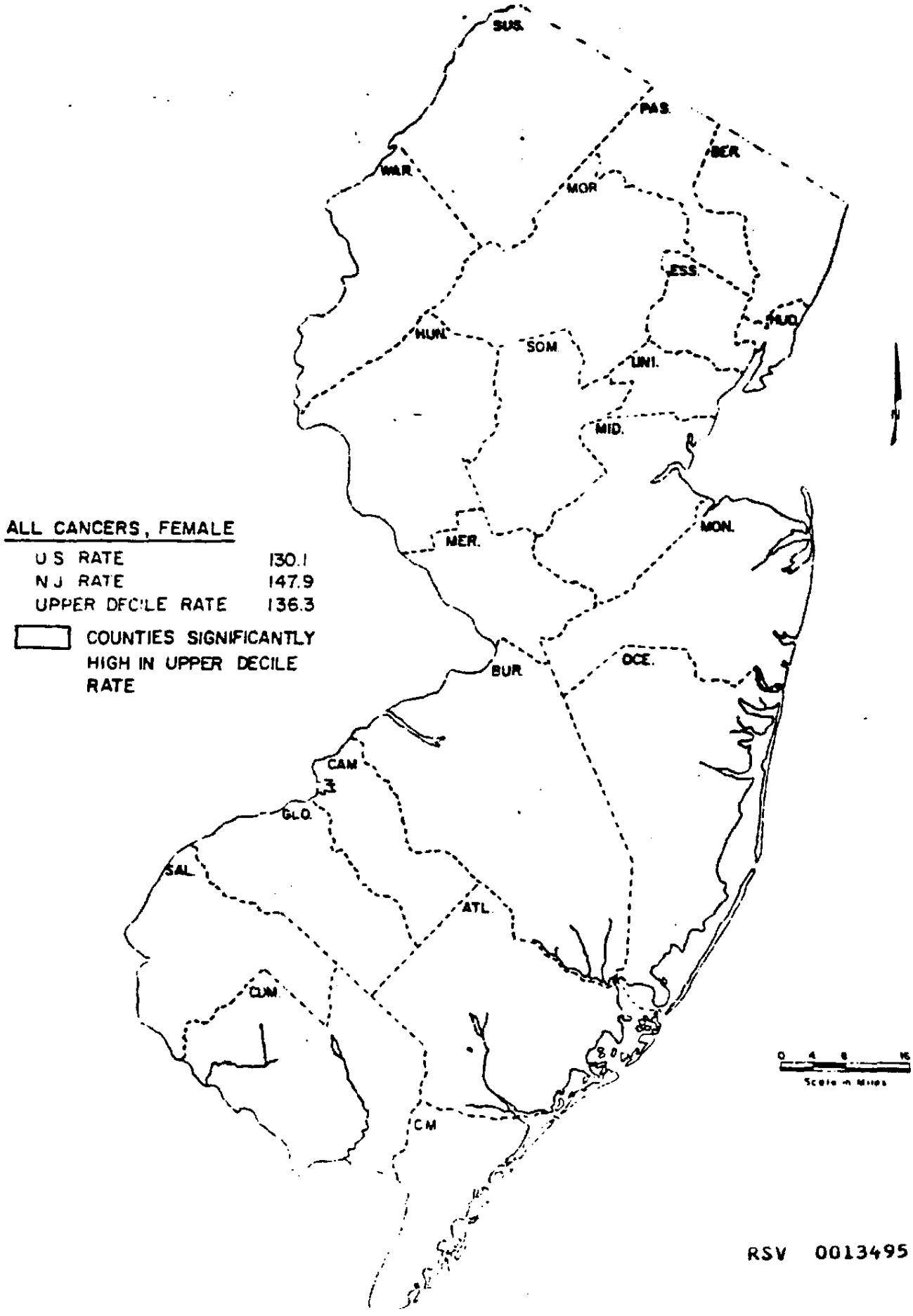
The counties shown with circles are those whose mortality rates were in the upper decile for all counties in the U.S. The upper decile is composed of the 10 percent of all counties in the U.S. that had the highest mortality rates nationally.

The counties shaded are those that had a mortality rate significantly higher than the U.S. rate. Due to difficulties in reading the maps in the Cancer Atlas, not all counties that are significantly high are shaded. Revisions will be made as additional information is received from the National Cancer Institute.

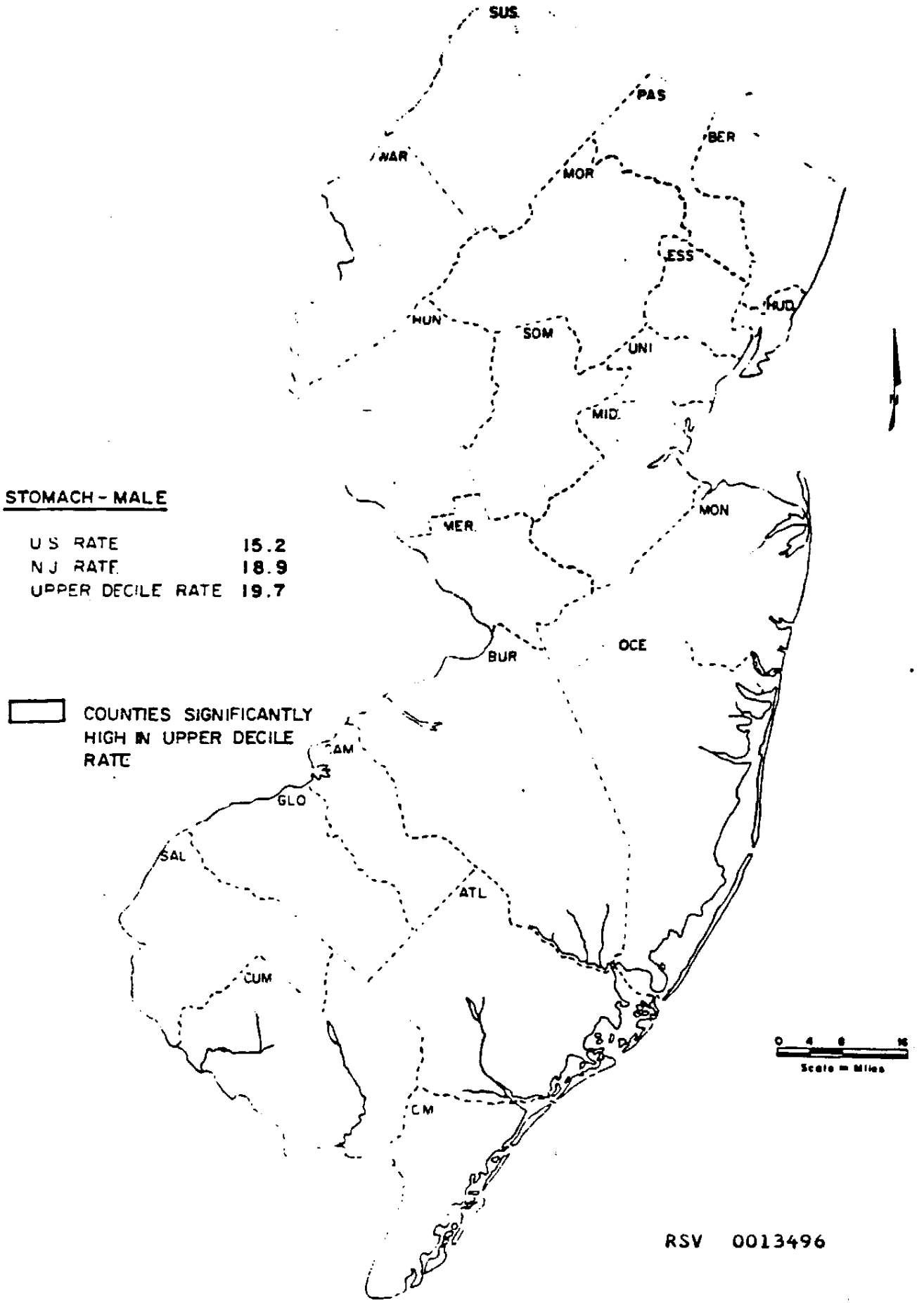
RSV 0013493


ALL CANCERS, MALE
U S RATE 174.0
N J RATE 205.0
UPPER DECILE RATE 178.0
COUNTIES EQUAL TO OR EXCEEDING UPPER DECILE RATE
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
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Scale in Miles

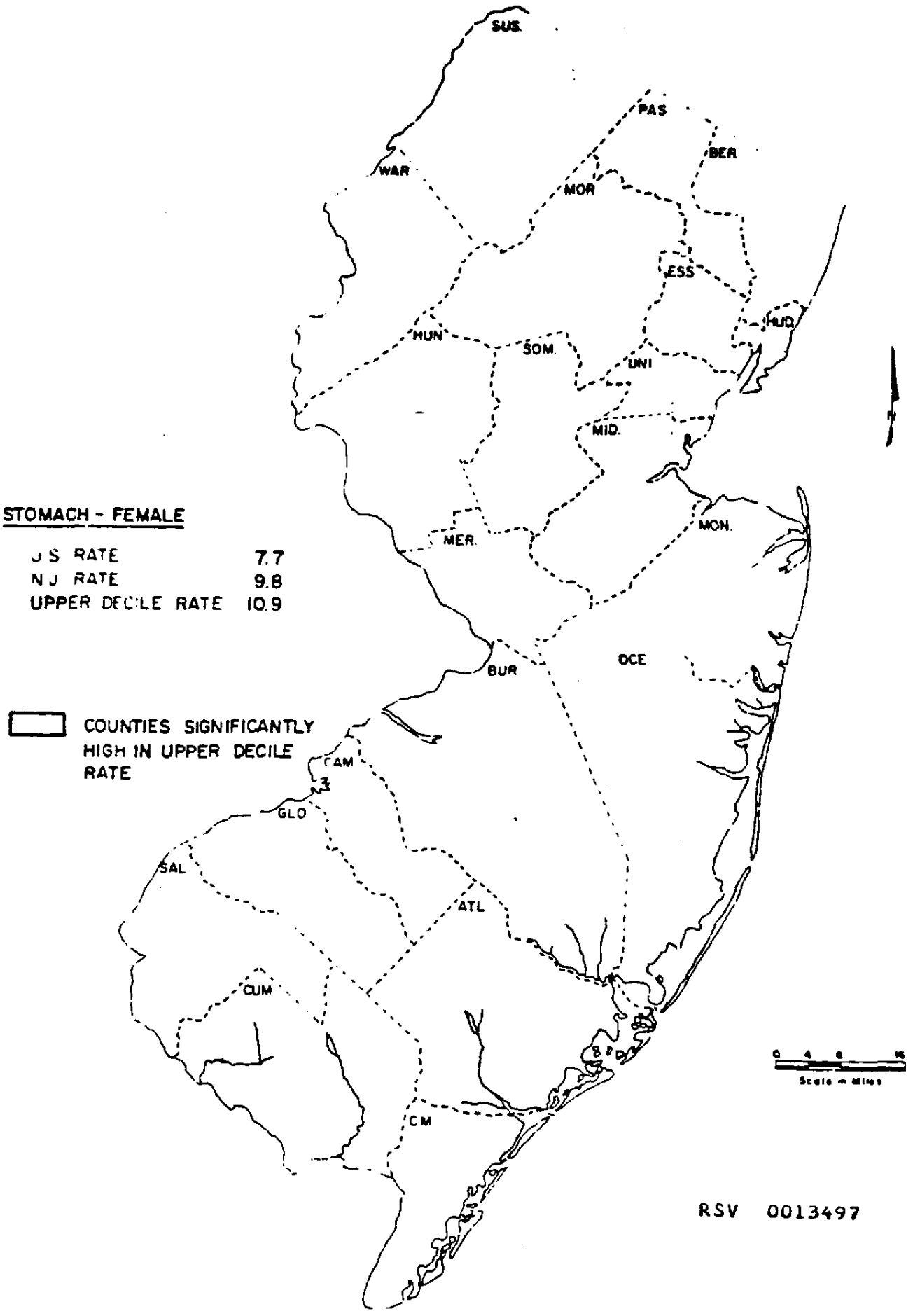
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ALL CANCERS, FEMALE
U S RATE 130.1
N J RATE 147.9
UPPER DECILE RATE 136.3
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0 4 8 16
Scale in Miles

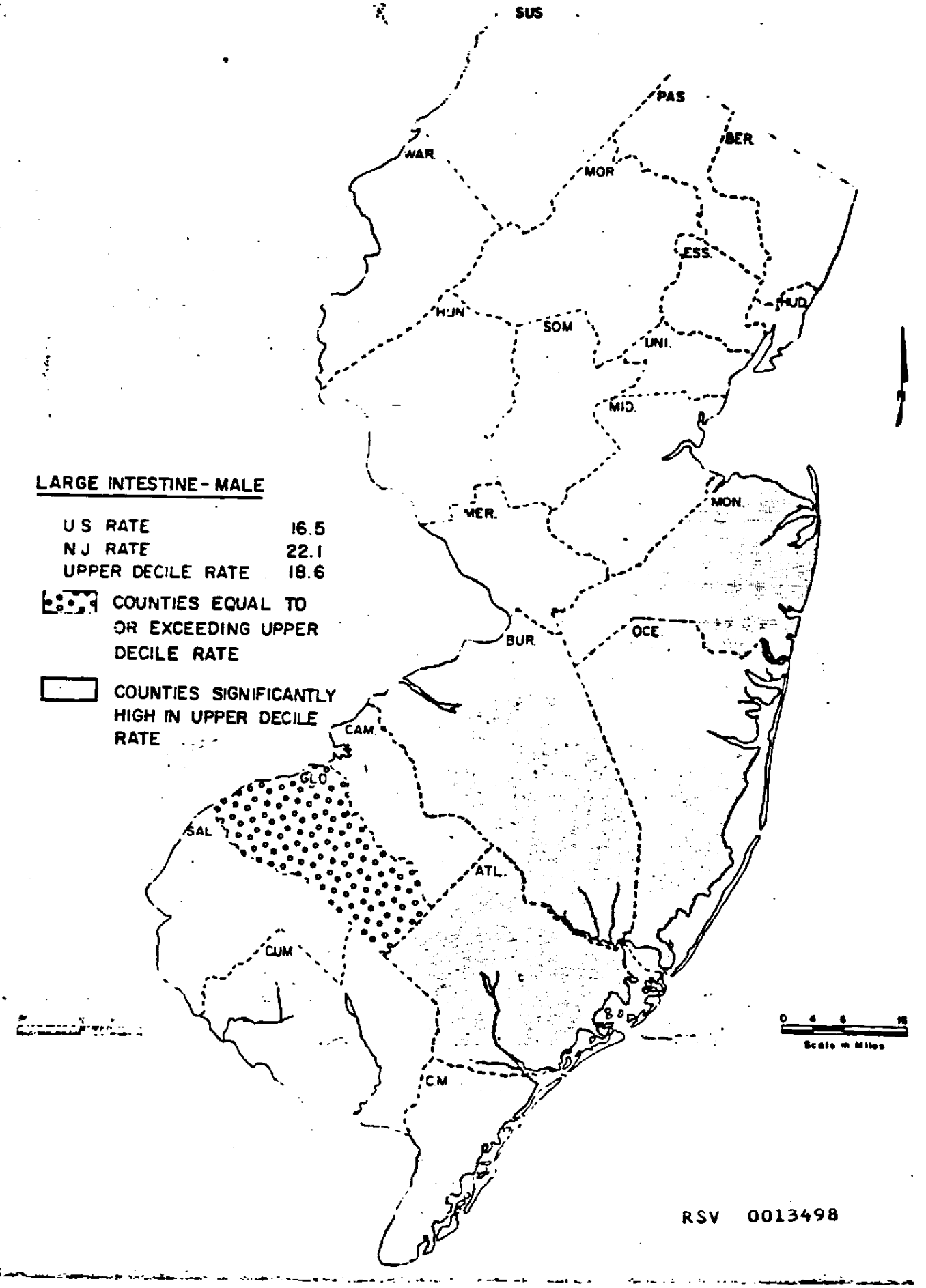
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STOMACH - MALE
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0 4 8 16
Scale in Miles

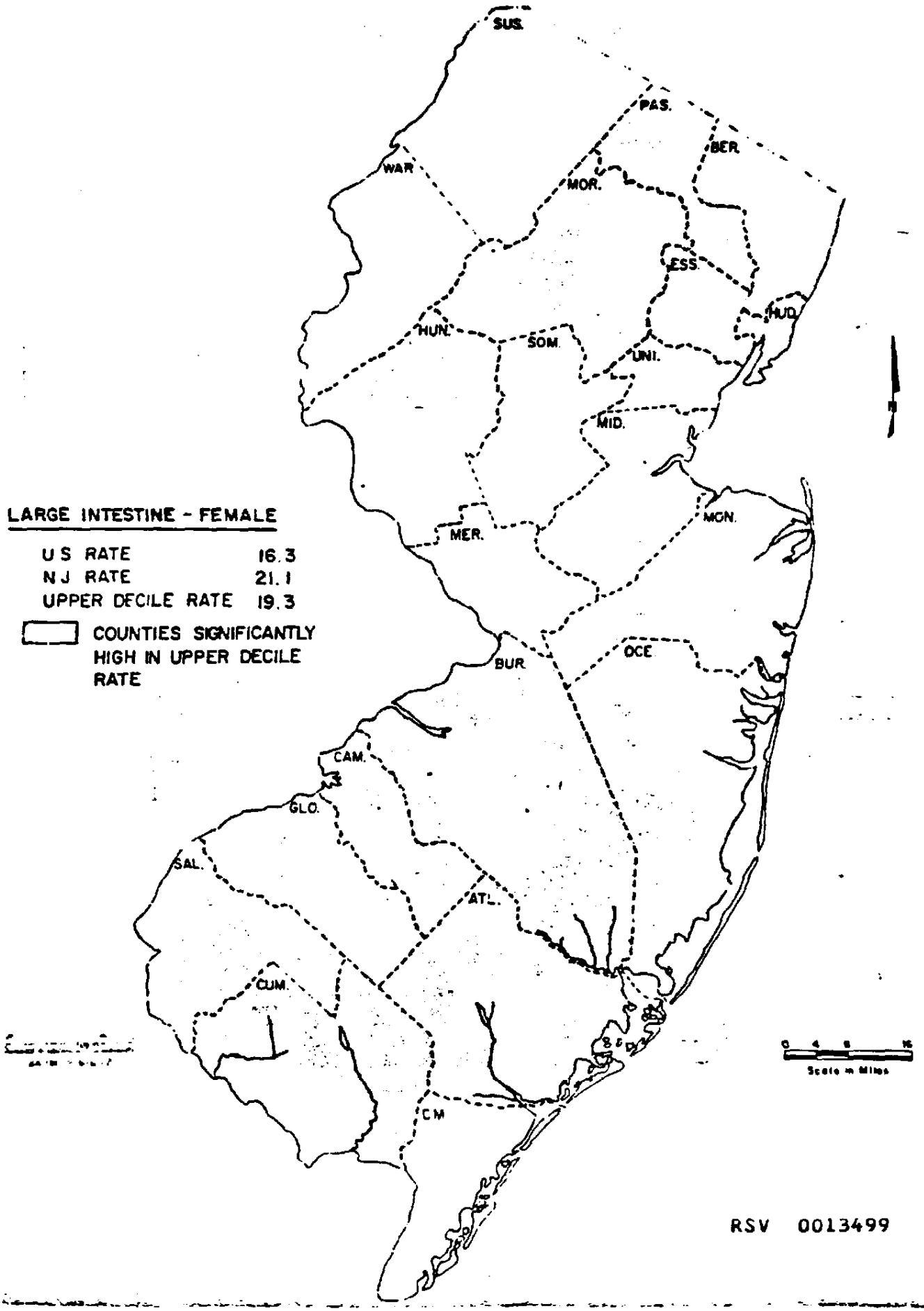
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STOMACH - FEMALE
U S RATE 7.7
N J RATE 9.8
UPPER DECILE RATE 10.9
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
0 4 8 16
Scale in Miles

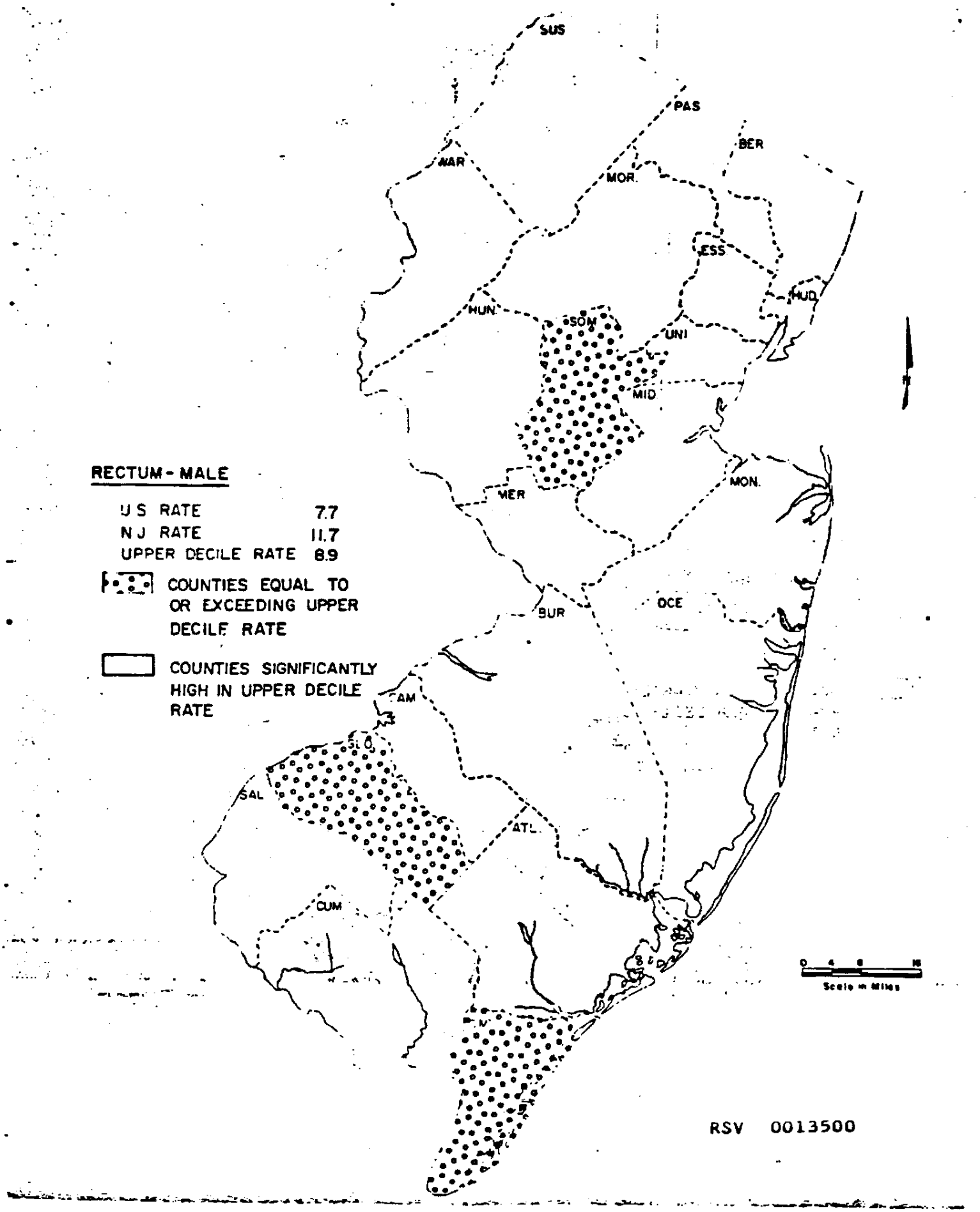
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LARGE INTESTINE - MALE
U S RATE 16.5
N J RATE 22.1
UPPER DECILE RATE 18.6
COUNTIES EQUAL TO OR EXCEEDING UPPER DECILE RATE
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
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Scale in Miles

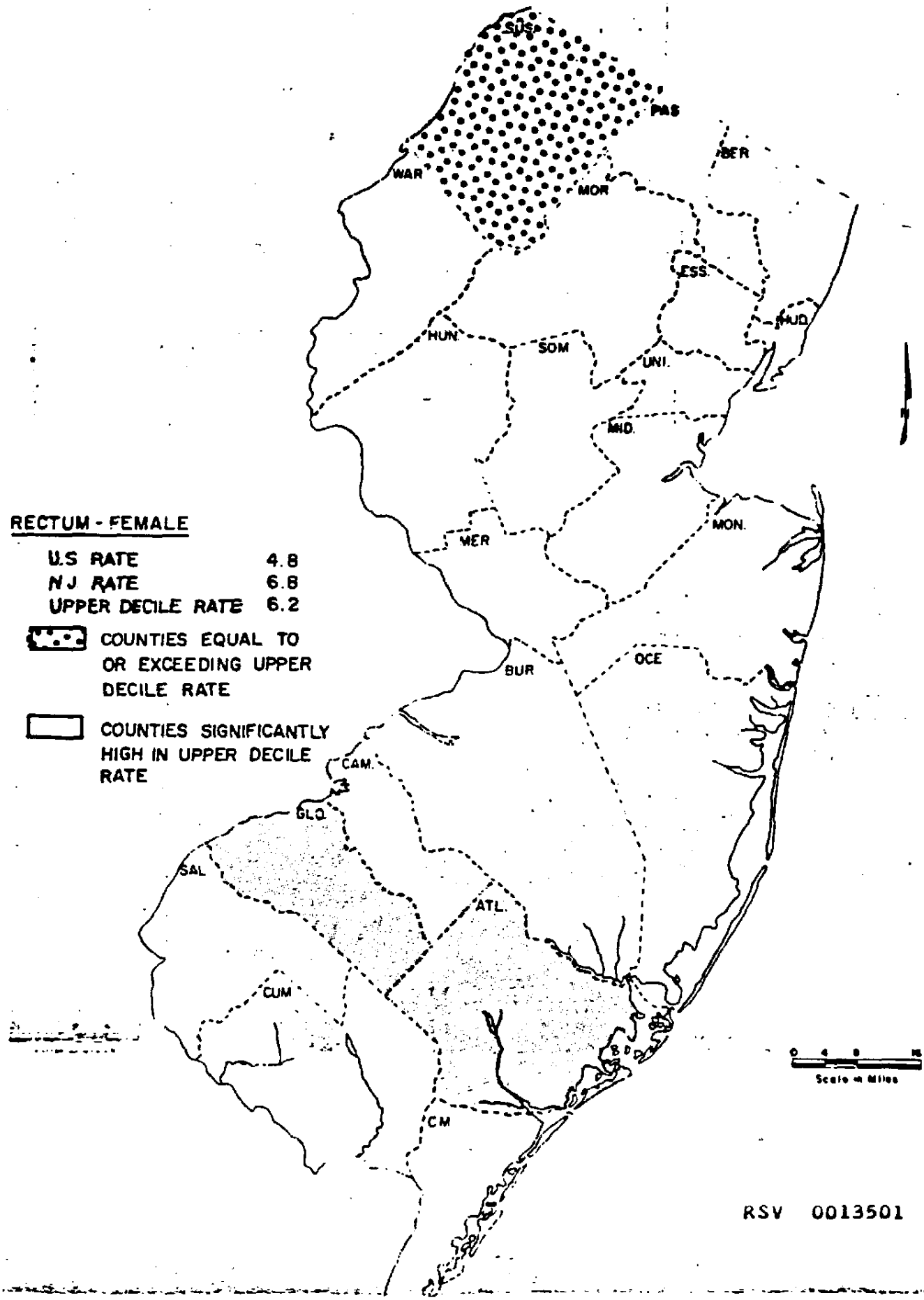
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LARGE INTESTINE - FEMALE
U S RATE 16.3
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COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
0 4 8 16
Scale in Miles

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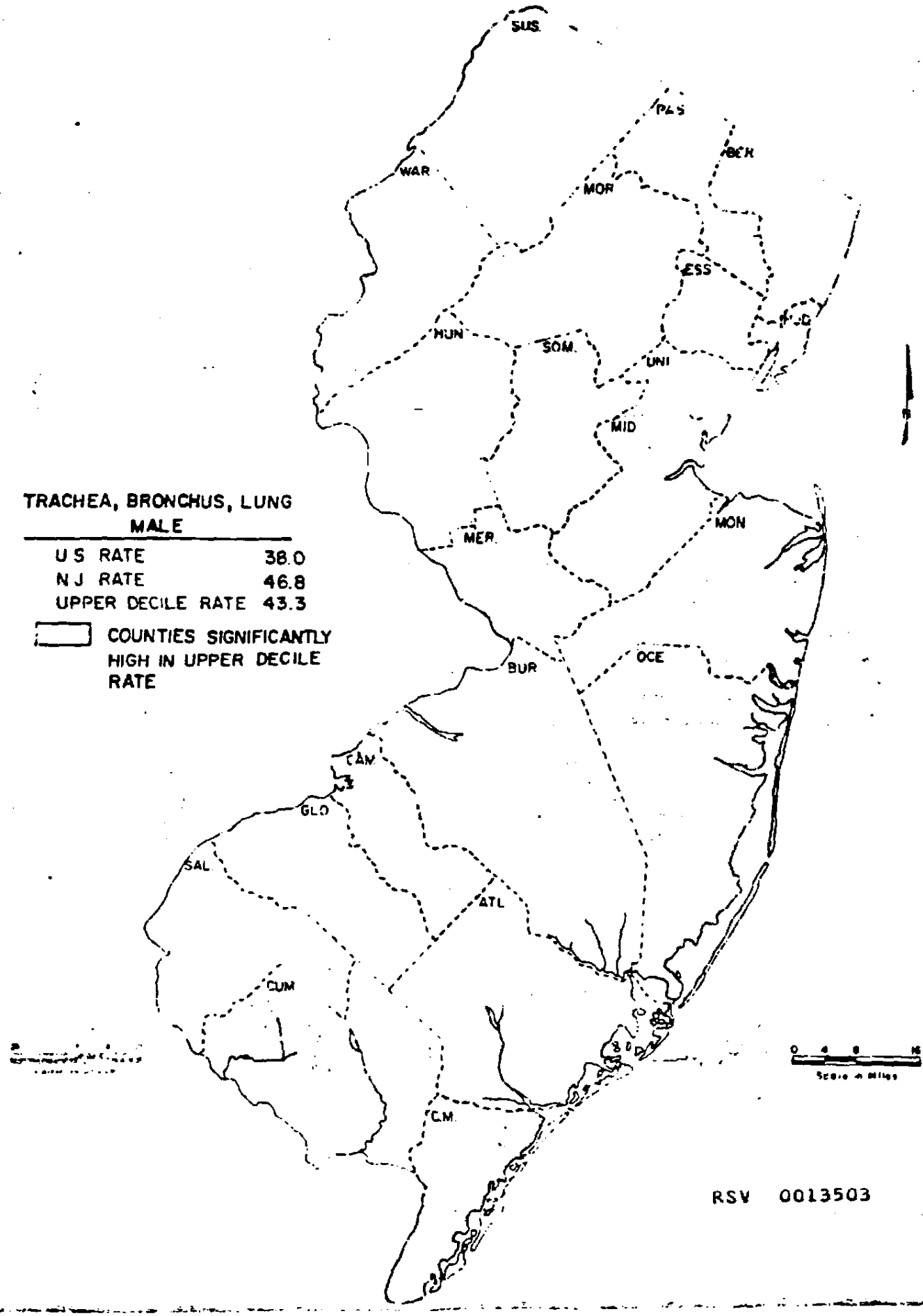

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RECTUM - MALE
U S RATE 7.7
N J RATE 11.7
UPPER DECILE RATE 8.9
COUNTIES EQUAL TO OR EXCEEDING UPPER DECILE RATE
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
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Scale in Miles

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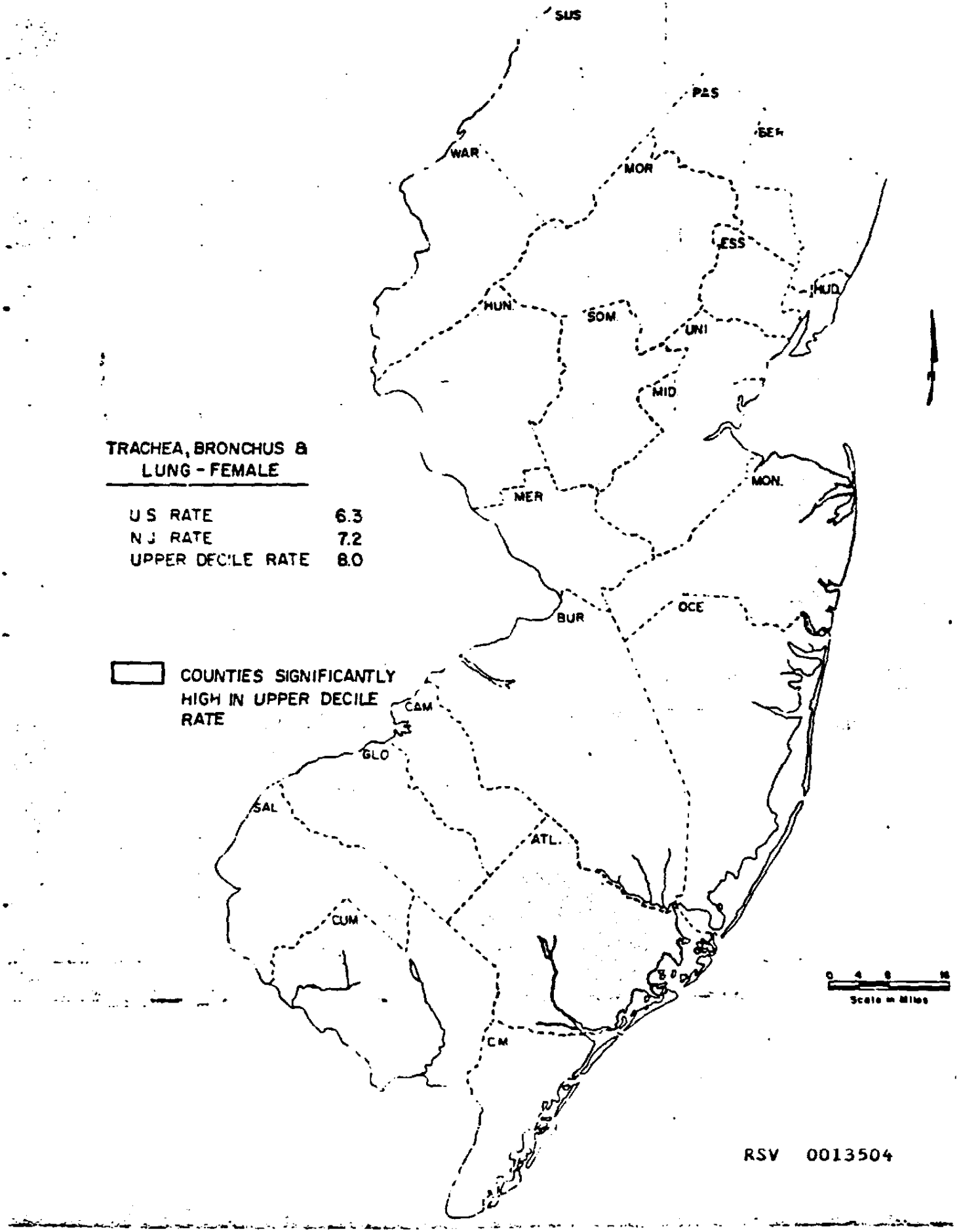

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RECTUM - FEMALE
U.S RATE 4.8
NJ RATE 6.8
UPPER DECILE RATE 6.2
COUNTIES EQUAL TO OR EXCEEDING UPPER DECILE RATE
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
Scale in Miles
RSV 0013501

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PANCREAS – FEMALE
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N.J. RATE 6.3
UPPER DECILE RATE 8.0
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
Scale in Miles

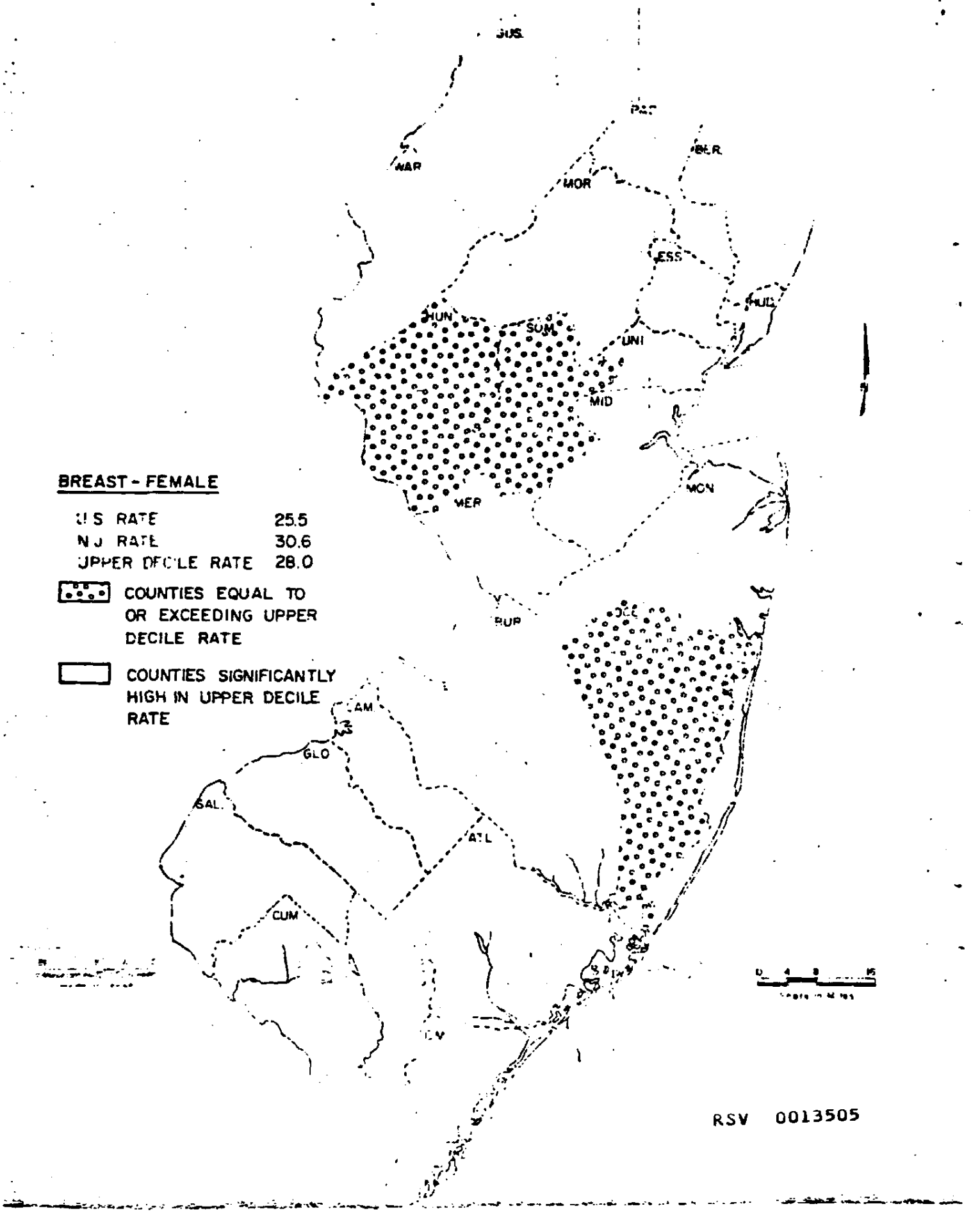
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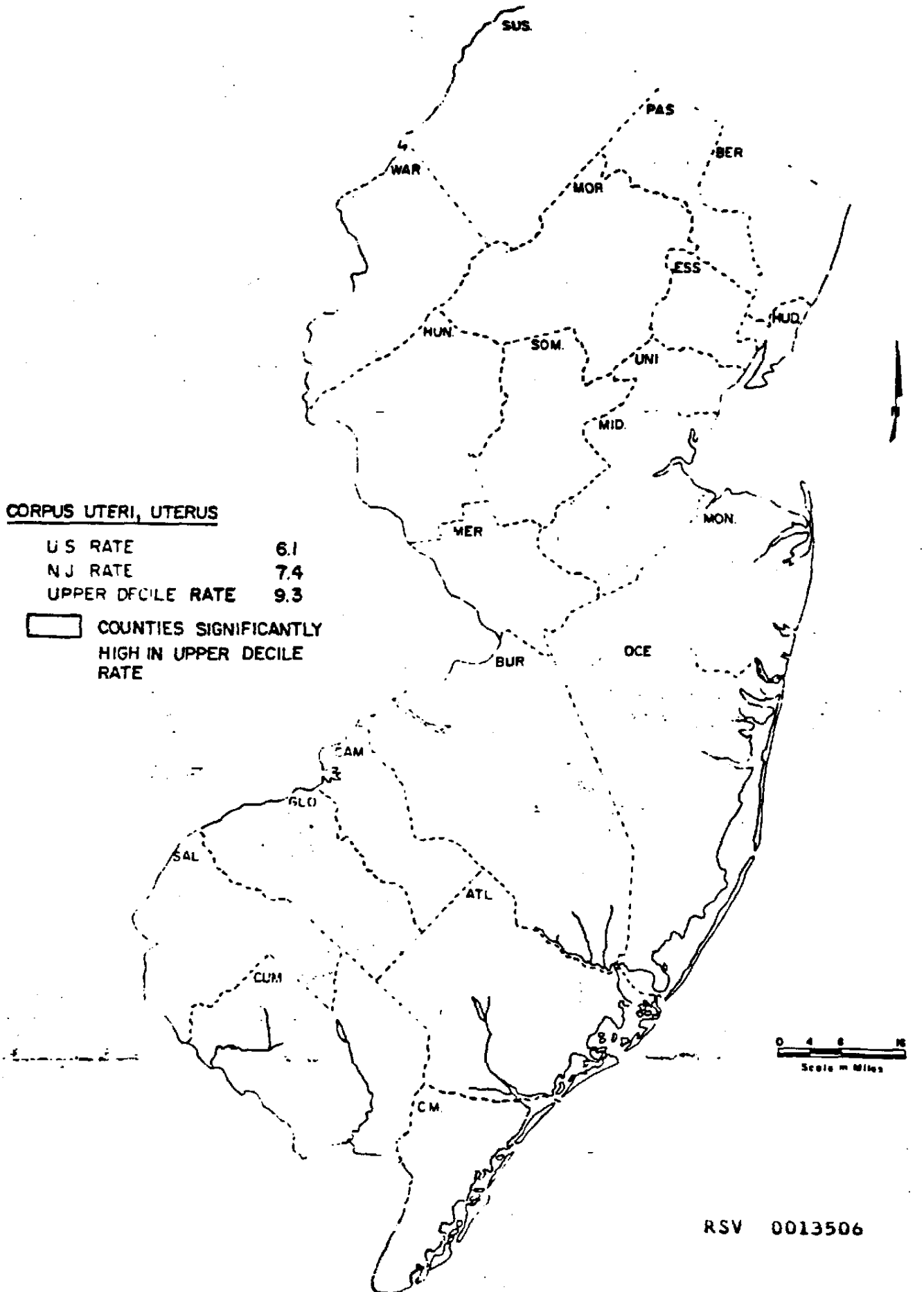

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TRACHEA, BRONCHUS, LUNG
MALE
U S RATE 38.0
N J RATE 46.8
UPPER DECILE RATE 43.3
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Scale in Miles

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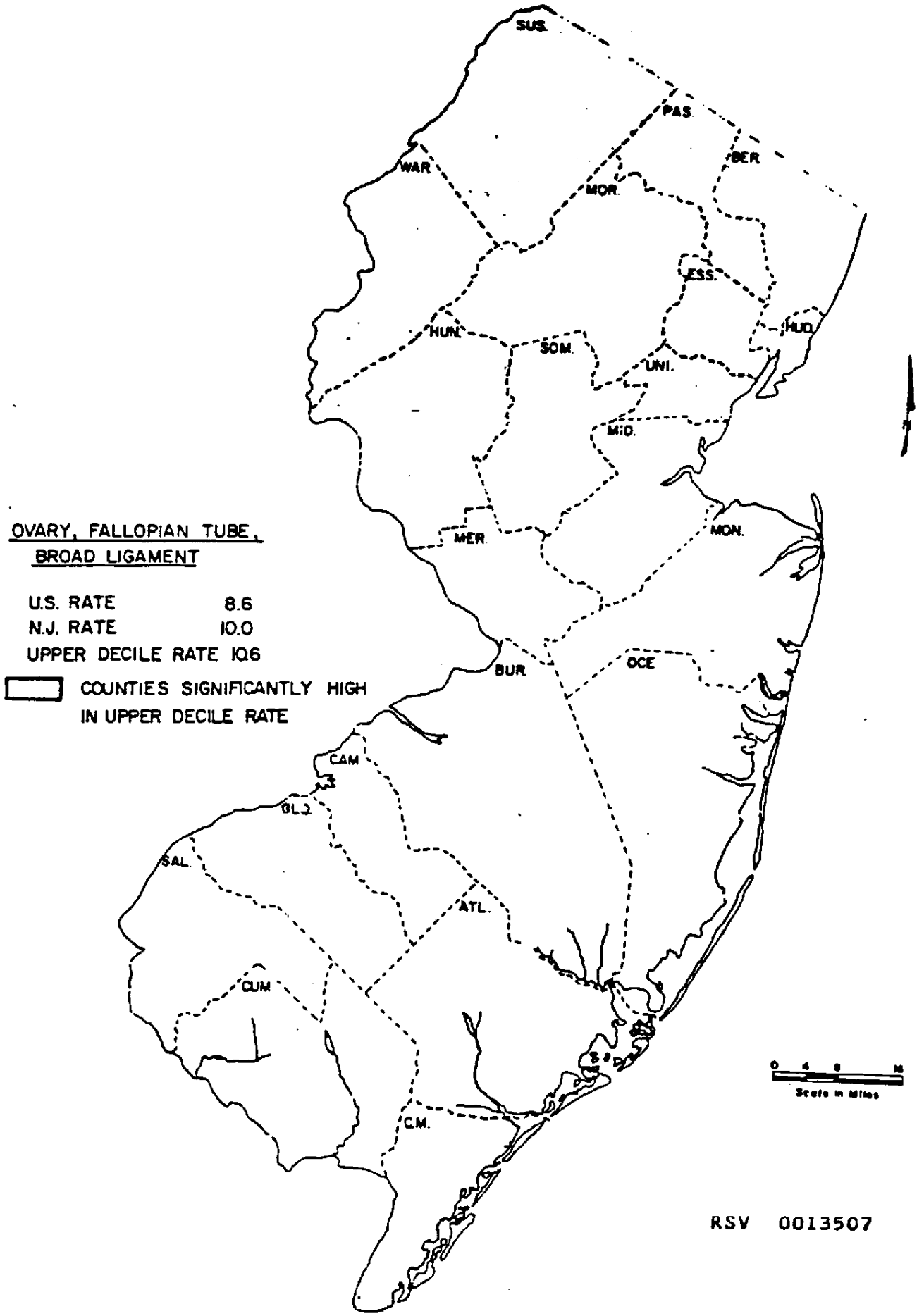

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TRACHEA, BRONCHUS & LUNG - FEMALE
U S RATE 6.3
N J RATE 7.2
UPPER DECILE RATE 8.0
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
Scale in Miles

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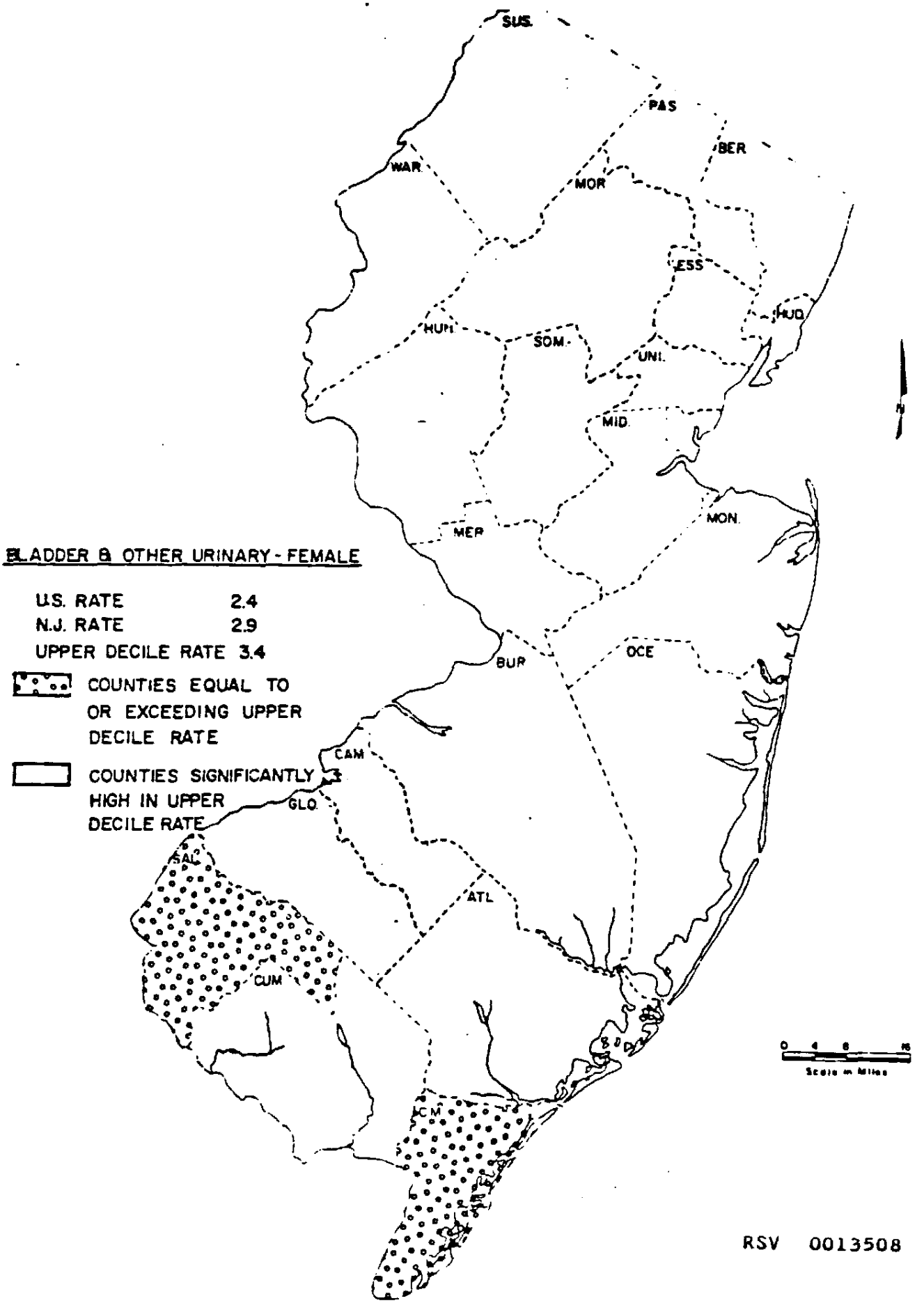

BREAST - FEMALE
U S RATE 25.5
N J RATE 30.6
UPPER DECILE RATE 28.0
COUNTIES EQUAL TO OR EXCEEDING UPPER DECILE RATE
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
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CORPUS UTERI, UTERUS
U S RATE 6.1
N J RATE 7.4
UPPER DECILE RATE 9.3
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
0 4 8 16
Scale in Miles

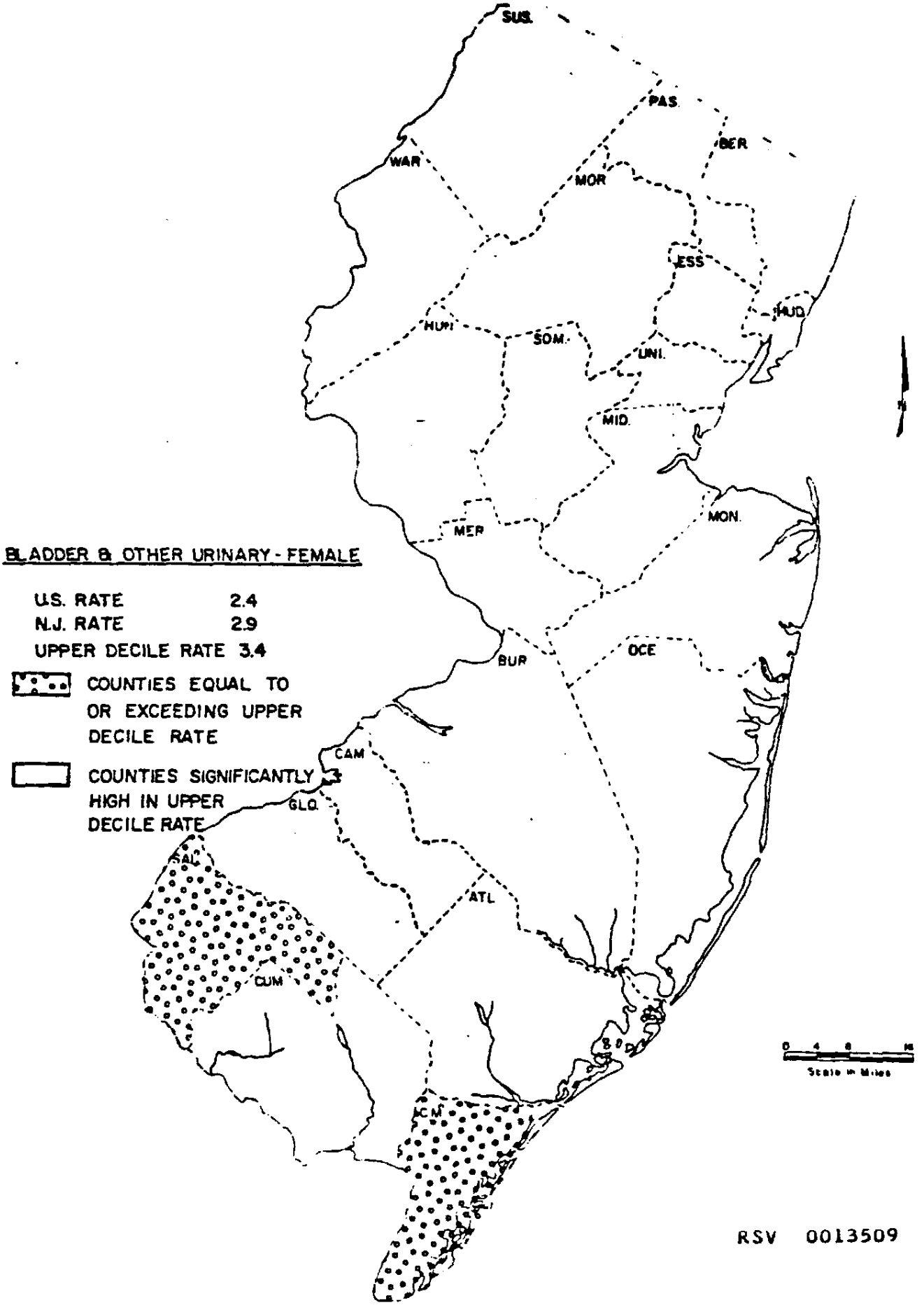
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C.M.
OVARY, FALLOPIAN TUBE, BROAD LIGAMENT
U.S. RATE 8.6
N.J. RATE 10.0
UPPER DECILE RATE 10.6
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
0 4 8 16
Scale in Miles

RSV 0013507


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BLADDER & OTHER URINARY - FEMALE
U.S. RATE 2.4
N.J. RATE 2.9
UPPER DECILE RATE 3.4
COUNTIES EQUAL TO OR EXCEEDING UPPER DECILE RATE
COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE
Scale in Miles

RSV 0013508



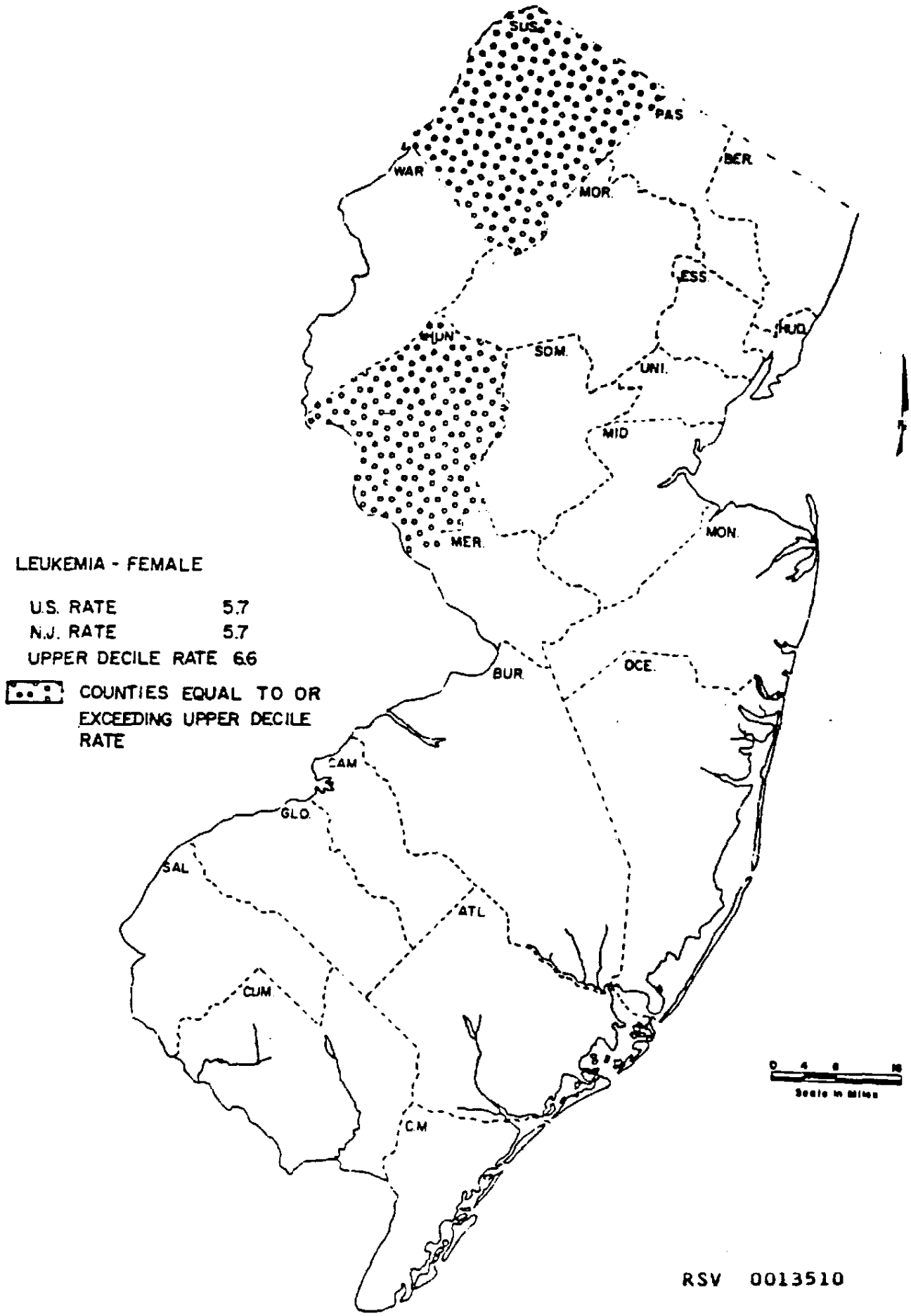
BLADDER & OTHER URINARY - FEMALE

U.S. RATE 2.4
N.J. RATE 2.9
UPPER DECILE RATE 3.4

COUNTIES EQUAL TO OR EXCEEDING UPPER DECILE RATE

COUNTIES SIGNIFICANTLY HIGH IN UPPER DECILE RATE

RSV 0013509


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LEUKEMIA - FEMALE
U.S. RATE 5.7
N.J. RATE 5.7
UPPER DECILE RATE 6.6
COUNTIES EQUAL TO OR EXCEEDING UPPER DECILE RATE
Scale in Miles

RSV 0013510

Appendix 3.

FACT SHEET: ATLAS OF CANCER MORTALITY FOR U.S. COUNTIES: 1950-1969

U.S. Department of Health, Education, and Welfare
National Institutes of Health
National Cancer Institute
Bethesda, Maryland 20014

The National Cancer Institute (NCI) has published an Atlas of Cancer Mortality for U.S. Counties: 1950-1969 showing geographic variation in cancer death rates across the U.S. for 35 anatomic sites of cancer.

NCI scientists believe the Atlas provides clues to occupational and other environmental factors that contribute to cancer causation. The Atlas can be used to identify communities or areas of the U.S. where additional studies may pinpoint these factors.

Authors of the Atlas are Thomas J. Mason, Ph.D., Frank W. McKay, Robert Hoover, M.D., William J. Blot, Ph.D., and Joseph F. Fraumeni, Jr., M.D. of NCI's Epidemiology Branch.

The Atlas contains maps of 16 common cancer sites on a county-by-county basis. The other 19 sites, for which fewer deaths occurred during 1950-1969, are mapped by state economic area (SEA). The SEA is a unit defined by the U.S. Bureau of the Census as a single county or group of counties with similar economic and social characteristics. SEAs usually are intermediate in size between counties and states.

The maps are based on average annual cancer death rates (deaths per 100,000 population) computed after tabulation of cancer deaths in the U.S. during 1950-1969. This information, obtained from data provided by HEW's National Center for Health Statistics, is based on death certificates. The rates were computed separately for whites and

nonwhites and for males and females. The data were adjusted to correspond to the distribution by age of the U.S. population in 1960. The resulting 729-page volume of cancer death rates was published by NCI in 1974.

The new maps of cancer mortality show geographic patterns separately for males and females and describe cancer only in whites. The smaller numbers of nonwhites in the U.S. make modification of the mapping technique necessary to assure reliable results. Another mapping study now under way will examine in detail geographic differences in cancer death rates for nonwhites.

The maps are followed by summary tables for each cancer site, listing a percentile ranking of both mortality rates and numbers of deaths. Using these tables, together with the volume of county-by-county tabulations, a reader may assess the relative impact of a particular cancer for any county. Other tables in the Atlas list death rates for each cancer site for consecutive five-year age groups.

NCI scientists suggest the chief value of the maps will be to stimulate scientists and other health professionals to conduct studies of intriguing cancer patterns in their own locales. Similar geographic patterns for both males and females for a particular cancer suggest that common environmental factors may contribute to causation; markedly different patterns for the sexes suggest effects of occupational factors.

For some sites such as melanoma (a rare form of skin cancer), the NCI scientists found predictable geographic patterns. Melanoma deaths occurred predominantly in the southern U.S. In areas of the Southwest bordering Mexico, rates were somewhat lower. Scientists have known for many years that sunlight is a major cause of skin cancer, and that darker-skinned persons

are less susceptible.

Cancers of the colon and rectum, believed to be related to diet, were found in both sexes at above average rates in the Northeast (New Jersey, southern New York, Connecticut, Rhode Island, Massachusetts) and in urban areas along the Great Lakes (Buffalo, Cleveland, Detroit, Chicago, Milwaukee). Low rates were found in the southern and central parts of the U.S. Surprisingly, breast cancer showed a similar pattern, suggesting that this disease may have an environmental factor in common with cancers of the large intestine.

High rates in the Northeast for cancers of the esophagus, larynx, mouth and throat, and bladder were limited to males, suggesting the influence of occupational factors. In a correlation study, the NCI scientists identified high rates of cancers of the lung, liver and bladder in counties with significant employment in the chemical industry. Additional studies are needed to clarify any occupational risks.

It is nearly certain, the NCI scientists believe, that industrial exposures have produced the striking geographic concentrations of bladder cancer deaths in males in the East.

The NCI scientists also found above-average lung cancer death rates in counties where a significant percentage of the work force is engaged in smelting and refining of copper, lead and zinc ores. Arsenic, a known human cancer-producing agent, is an airborne byproduct of the smelting operation for these ores. Above-average rates were found for females as well as males in these counties, suggesting spread of an occupational risk to the surrounding community.

High mortality rates for lung cancer were found along the Gulf Coast from Texas to the Florida Panhandle. Of Louisiana's 64 counties, 38

are in the highest 10 percent of all U.S. counties ranked for rates of male lung cancer mortality. Thirteen Louisiana counties are in the top one percent, as are an additional seven counties along the Gulf Coast and along the Atlantic Coast from northern Florida to Charleston, South Carolina. This pattern suggests that environmental factors, in addition to cigarette smoking, may be contributing to lung cancer deaths in these predominantly rural and seaport areas.

High rates of stomach cancer were found in the North Central States (the Dakotas, Minnesota, Wisconsin, upper Michigan). NCI scientists believe ethnic factors are responsible for this pattern. The high rates in both sexes correspond closely with the geographic concentration of persons with ancestors from Austria, the Soviet Union, and Scandinavia. Stomach cancer rates in these countries are also higher than the U.S. average.

The NCI scientists noted an unexpected concentration of above-average rates for cancers of the lip and mouth/throat among women in the South. There were no clearly discernible patterns in the U.S. for several forms of cancer, including cancers of the pancreas, brain, salivary gland, nose, and sinuses.

The authors of the mapping study caution that the maps should not be used alone to ascribe cancer mortality to hazards in specific areas. For example, mortality rates in specific areas sometimes may reflect environmental exposures from entirely different parts of the United States due to population movements such as retirement. However, the geographic patterns are compatible with known risk factors for several cancer sites, a fact that gives the scientists confidence in the geography of cancers for which little has been known.

NCI scientists already have used the county-by-county mortality data and mapping study as an initial indicator of possible public health hazards, and have found no unusual cancer mortality patterns so far among persons residing where drinking water is contaminated by asbestos or where homes are built on radioactive tailings from uranium mines. However, continued monitoring of the mortality patterns in these areas is necessary.

NCI scientists will continue to refine the data and mapping techniques. New studies will follow up leads suggested by national patterns for specific forms of cancer. Among these will be studies concerning the effect of migrations of persons from one part of the U.S. to another.

Results of a separate mapping study of geographic differences among nonwhites, when completed, will be used to compare differences among whites and nonwhites. An in-depth analysis of the effects of socioeconomic status, urbanization, and latitude on cancer death rates will complement the current studies of the U.S. county data.

# # #

June 1975

CANCER AND CANCER RESEARCH IN NEW JERSEY

Appendix 4.

A Report by the Research Committee of the Cancer Institute of New Jersey, March 1976*

Recent headlines quote from an NIH report[1] that New Jersey citizens have the highest cancer rates in the United States for many of the most frequently fatal types of cancer, including cancer of the bladder, breast, large intestine, rectum, and lung. This has been known for a number of years, but has attracted relatively little publicity or official response in New Jersey. Statistical analysis as shown in Table 1 indicates that New Jersey has 3.5 percent of the U.S. population, but 4 percent of new cancer cases which makes the incidence of cancer 14 percent higher in New Jersey than in the United States. The estimated hospital costs of cancer care to New Jersey residents were 75 million in 1972.

Nationally, cancer is the disease most feared by the American people and in response to these concerns Congress established the National Cancer Program in 1971 to provide the National Cancer Institute (NCI) more funds for cancer research and control. Funding of the effort at NCI has increased from $181 million in 1970 to over $600 million in 1975, but New Jersey has qualified for relatively little of this (see Table 2).

* Membership: Lewis L. Coriell, Chairman, Institute for Medical Research; Charlotte Avers, Douglass College; Alan Conney, Hoffman-LaRoche; Eric Hirschberg, CMDNJ; Arnold Levine, Princeton University; Donald Norris, Rutgers Medical College; Robert J. Robinson, Rutgers Medical College; Arnold Rubin, CMDNJ; R. Walter Schlesinger, Rutgers Medical College; Robert W. Simpson, Waksman Institute; William A. Strohl, Rutgers Medical College; Thomas R. Walters, CMDNJ

TABLE 1

COMPARISON OF CANCER STATISTICS IN N.J. AND THE U.S.

	U.S.	N.J.	N.J. AS % OF U.S.
POPULATION (2)	212,812,000	7,414,000	3.5
NEW CANCER CASES (3)	655,000	26,000	4.0
CANCER DEATHS (3)	355,000	14,000	3.9
INCIDENCE PER 100,000 POPULATION	308	351	114.0
MORTALITY PER 100,000 POPULATION	167	189	113.2

COST OF HOSPITAL CARE FOR N.J. CANCER PATIENTS (4) $75 MILLION

ESTIMATED TOTAL COSTS INCLUDING DOCTOR BILLS, OPD THERAPY, LOST WAGES, INSURANCE, INDIRECT COSTS (5,6) $750 MILLION TO 1.0 BILLION

TABLE 2

CANCER RESEARCH GRANTS AND FELLOWSHIPS AWARDED DURING FY 1975

STATE	POPULATION (7)	AWARDED BY ACS			AWARDED BY NCI		
		TOTAL $	PER CAPITA $	N.J. AS 100%	TOTAL $	PER CAPITA $	N.J. AS 100%
CONNECTICUT	3,095,000	$1,109,224	.36	900	$ 10,827,000	3.50	336
MARYLAND	4,098,000	$ 615,996	.15	375	$ 75,211,000	18.35	1760
MASSACHUSETTS	5,828,000	$1,636,282	.28	700	$ 42,361,000	7.27	699
NEW JERSEY	7,316,000	$ 307,725	.04	100	$ 7,598,000	1.04	100
NEW YORK	18,120,000	$4,757,318	.26	650	$107,251,000	5.92	569
PENNSYLVANIA	11,827,000	$1,358,661	.12	300	$ 34,788,000	2.94	283
VIRGINIA	4,967,000	$ 484,146	.10	250	$ 17,156,000	3.45	332

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Citizen concern for this situation led to founding of the non-profit Cancer Institute of New Jersey (CINJ) on September 20, 1974. Among its purposes are --- "improving existing levels of early detection, treatment, research and education in --- cancer."

CINJ applied for a planning grant from NCI to develop a plan for attacking New Jersey's cancer problem in a coordinated manner including the most modern methods of cancer research, patient care, and cancer control. The application was approved in December 1975, and funded in August 1976.

The Research Committee of CINJ at its first organizational meeting on January 30, 1976 sought to identify explanations for New Jersey's lack of participation in the national cancer research programs on the theory that if reasons could be identified they would form the basis for corrective measures in the cancer plan to be developed for New Jersey.

This brief report is the first attempt to respond to this issue and to identify the causes and suggest solutions for New Jersey's failure to take advantage of federal programs to support cancer research and control. To do this New Jersey's performance will be compared to other states in its geographic region.

New Jersey is in the heart of the industrialized eastern seaboard. It is 46th in size among the states, but has the densest population per square mile, 1,016 in 1975[8], and is increasing at the rate of 1.3 percent per year (Table 3). It has the 4th highest per capita income among the states and is the most highly urbanized (Table 4). In spite of its density of wealth, population, and need for cancer research, New Jersey has not been able to attract much of the federal funds made available through the National Cancer Institute.

TABLE 3 [8]

NEW JERSEY POPULATION

	1975	Est. 1980	Est. 1985
Total population	7,644,720	8,095,000	8,596,500
Population/sq. mile	1.016	1.076	1.143

Historically, New Jersey has lacked the most advanced and complex medical care facilities. Residents who required these services have had to seek them in institutions in New York City and Philadelphia. This served the more affluent citizens of New Jersey who could afford to travel to another state for medical care but retarded provision of high quality care for the average citizen.

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One of the reasons is that for many years New Jersey provided no centers of excellence for training physicians and has therefore had to export its medical students to other states for medical

education. These centers of excellence in education and training are usually the nucleus of centers of excellence of patient care. In recent years as the costs of medical education have increased many states have refused to accept New Jersey applicants to medical school because a considerable portion of the expense is borne by the receiving state. The net result is that many New Jersey students cannot get into American medical schools and many of those who do succeed never come back to New Jersey - but set up practice in the area where they receive their undergraduate and residency training. There are a number of cumulative effects of these policies, for example:

1. A shortage of medical manpower: A survey in 1975 shows that the national average ratio of M.D.'s per 100,000 population is 159, whereas in New Jersey it is 126, and in South Jersey it is 91.5[8]. Dr. Stanley Bergen, President of CMDNJ has estimated that there is a continuing need for 100 new physicians a year in South Jersey to gradually correct the deficit and replace physicians who die or leave the area. As of January 1975 only a small percentage of all M.D.'s practicing in New Jersey had received their medical education in New Jersey: 6.2% in Area I; 7.3% in Area II; 5.7% in Area III; and 3.8% in Area IV.

2. A shortage of fulltime physicians in hospitals: There are few medical school type hospitals in New Jersey where American medical school graduates prefer to take their internship and residency training. Therefore, most New Jersey hospitals do not attract American trained internes and residents and must settle for

TABLE 4

RANK OF NEW JERSEY IN RELATION TO OTHER STATES 1974-1975

	Population Density (9).		Income/Capita (10)
State	Per Sq. Mile	Rank	Rank
Connecticut	624	4	2
Maryland	397	5	10
Massachusetts	727	3	12
New Jersey	953	1	4
New York	381	6	6
Pennsylvania	262	8	18
Virginia	117	15	23
United States	58		

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graduates of foreign schools or do without residents (Table 5).

3. A shortage of research: With few fulltime teachers, residents, and practitioners in most New Jersey hospitals it is obvious that there is little time for research, research training, or research facilities devoted to cancer or to any other disease for which present knowledge is insufficient (Table 6). Note that in New Jersey medical schools there are 1.4 students enrolled for advanced degrees per 100,000 population whereas in neighboring states it varies from 5.9 to 15.3 with an average of 9.2/100,000 or 657 percent more than in New Jersey. Figures are not available at this writing for students enrolled for advanced science degrees in universities. However New Jersey has only one major university so its position relative to neighboring states is not likely to be changed when this data becomes available.

4. A suboptimal quality of care: The ultimate result of the first 3 points is suboptimal care for the citizens of New Jersey. Other cumulative effects could be listed but the ones above are sufficient to indicate that there are chronic deficiencies in the quality and availability of patient care and health delivery in New Jersey. The high incidence of tuberculosis and infant mortality in New Jersey's urban centers, the above average incidence of cancer deaths, the necessity of seeking care in Philadelphia or New York are unresolved concerns. The costs of hospitalization and treatment in Philadelphia are approximately double those in South Jersey. A comparison made in 1973 shows the following average costs of procedures in Philadelphia vs. South Jersey (Table 7).

TABLE 5

INTERNE AND RESIDENT HOUSE STAFF IN NEW JERSEY HOSPITALS 1974-1975 (11)

STATE	TOTAL # HOUSE STAFF	# GRADUATES OF FOREIGN SCHOOLS	PERCENTAGE OF HOUSE STAFF GRADUATES OF FOREIGN SCHOOLS
CONNECTICUT	1,171	494	42
MARYLAND	1,956	754	38
MASSACHUSETTS	2,742	689	25
NEW JERSEY	1,743	1,289	74
NEW YORK	10,004	5,485	54
PENNSYLVANIA	3,869	1,255	32
VIRGINIA	1,133	191	17

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TABLE 6

GRADUATE TRAINING IN BASIC SCIENCE DEPARTMENTS OF MEDICAL SCHOOLS (12)

STATE	# STUDENTS ENROLLED FOR ADVANCED TRAINING			TOTAL STUDENTS	
	MASTERS	PH.D.	POST-DOCTORAL	#	#/100,000 POPULATION
CONNECTICUT	179	200	94	473	15.3
MARYLAND	105	202	78	385	9.4
MASSACHUSETTS	25	241	191	457	8.0
NEW JERSEY	11	73	19	103	1.4
NEW YORK	245	1,117	157	1,519	8.4
PENNSYLVANIA	183	578	196	957	8.0
VIRGINIA	58	204	25	287	5.9

DOES NOT INCLUDE GRADUATE TRAINING IN UNIVERSITIES.

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TABLE 7

AVERAGE COSTS IN $ (13)

	PHILA.	SOUTHERN NEW JERSEY	HIGHER % OF COST IN PHILA.
AVERAGE PER DIEM COST	$157	$ 85	83%
AVERAGE CARDIAC CATHETERIZATION	$700	$226	209%
AVERAGE DIALYSIS	$370	$138	168%
AVERAGE OPERATING ROOM CHARGE/HOUR	$245	$137	78%

In addition to the greater costs of medical care in Philadelphia for South Jersey residents one must add the expense and inconveniences of travel for patients and visitors, parking, and personal safety. The difference in costs is not accounted for by the types of tertiary care provided in Philadelphia. Most types of tertiary care are now provided in South Jersey and care by tertiary care physicians is not necessarily more expensive than care by primary care physicians as shown by a survey in Ohio.(14) In 1960 twenty-two percent of hospital beds in Philadelphia were occupied by South Jersey residents while in 1973 it was seven percent.

Cancer in New Jersey

New Jersey has some of the highest cancer death rates in the United States for the more frequently fatal cancers, including bladder, breast, large intestine, rectum, and lung as shown by a national survey.(1)

> "Of the 21 counties in New Jersey, 18 have bladder cancer rates in the highest decile of male rates for all U.S. counties. Indeed, the rate for Salem County New Jersey (16.1 per 100,000 population) ranks highest among all American counties with a white population of at least 10,000."

> "Of the 21 counties in New Jersey, the rates for colon cancer are in the highest decile nationally for 15

counties in males, and 16 counties in females. For rectal cancer, the corresponding numbers are 18 and 15."

Medical Education in New Jersey

The above deficiencies of health care among others led to the establishment of a private medical school at Seton Hall in 1954 which later was taken over by the State in 1965 and moved to Newark where it became the New Jersey College of Medicine and Dentistry. In 1962 the state opened another medical school at Rutgers called Rutgers Medical School. In 1970 both medical schools and the dental school were put under one Board of Trustees called the College of Medicine and Dentistry of New Jersey (CMDNJ) which now has several divisions: New Jersey Medical School (Newark); Rutgers Medical School (Piscataway); New Jersey Dental School (Jersey City); Graduate School for Biomedical Sciences (Newark); and another medical education program being developed in South Jersey. The latter school will be a branch of the parent CMDNJ and will provide the 3rd and 4th years of clinical training at existing Cooper Medical Center and other community hospitals. A Veteran's Hospital to serve Delaware Valley has been authorized at the site to complete a regional medical complex of teaching hospitals in South Jersey.

The primary function of a medical school is to train future physicians and the multiplicity of specialized skills required to deliver modern medical care means that a medical school differs from a community hospital in one important fact. It must have a nucleus of fulltime physicians who devote time to

training medical students. Time, which is not available to the busy practitioner. The 4 essential elements which a medical school must provide for thorough and efficient education of physicians are: 1)experience in patient care, 2)formal education, 3)research and 4)community outreach programs. These in turn require: --

1) Instruction in the care of sick patients requires that a medical school must have use of sufficient hospital beds and outpatient facilities to meet its teaching requirements.

2) Formal education requires classrooms and laboratories for lectures, demonstrations, examinations and discussion.

3) Time for research is essential to attract scholarly faculty who will study unsolved medical problems and keep abreast of recent advances at other institutions around the world. A medical school should have access to all advanced medical knowledge and be able to interpret it to the students and provide it to patients in its region.

4) Outreach programs to maintain and improve standards of community health care delivery are the fourth cornerstone for a modern medical school. It includes regional planning, ongoing postgraduate education of physicians,

cooperative programs with community hospitals for training internes and residents, refresher courses, coordination of health and welfare services, referral channels for regional hospitals, and research to develop better methods for community delivery of health care.

All these four elements are necessary to create a center of excellence for health care which we call a medical school. All four have been deficient in New Jersey in the past, and even today the medical school budgets provide inadequate clinical facilities at Rutgers, and inadequate support for research at all units - the most important single item in attracting and holding a scholarly faculty. The key to excellence in a medical school is the quality of the faculty. Modern medicine is advancing so fast that obsolescence occurs in a few years among practitioners unless they attend refresher sessions, and occurs even in the medical school unless a portion of the faculty can devote some time to research and scholarly pursuits that keep them abreast of new methods of diagnosis, treatment, prevention and education in their own specialty.

Research is another name for acquisition of knowledge and scientific research implies a systematic accumulation of verifiable facts in an effort to solve problems for which no known explanation exists. In biological science modern research dates from the time of Louis Pasteur who demonstrated over 100 years ago the techniques and benefits of the controlled experiment to learn about the cause, diagnosis and prevention of infectious diseases. Application of these techniques has eliminated most infectious diseases in the Western World and stands as the out-

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It did not happen suddenly. 60+ years of basic research after Pasteur showed the way were required before enough knowledge was accumulated to eliminate the epidemics of contagious and infectious diseases which formerly kept the death rate about equal to the birth rate. Infectious diseases were controlled, one by one, when sufficient knowledge was accumulated to break the cycle of infection at some vulnerable point. In some cases it was through vaccination, in others it was through sanitation, mosquito control, antibiotics, or chemotherapy. In all cases it involved study of the normal life cycle of an external germ or parasite capable of causing disease.

Control of cancer is infinitely more difficult than control of infectious diseases and will also require a prolonged period of basic research to accumulate the knowledge base necessary for control. The problem in cancer is loss or malfunction of normal control mechanisms that regulate interacting life processes between cells. Among mechanisms already implicated are: genetic susceptibility, viruses, radiation, chemical mutagens, immune surveillance, hormonal and neuronal messages, and the feedback messages that serve to integrate the actions of each cell with the whole body. Study of these chemical changes and controls within and between cells is called molecular biology and is the thrust of modern research which will eventually lead to better control of the diseases that now account for most serious illnesses and death - including arteriosclerosis, hypertension, heart disease, stroke, cancer, genetic diseases, diabetes, arthritis and mental diseases.

Is Research Cost Effective?

Western culture is built on research which continues to develop better ways of providing food, clothing, housing, transportation, labor saving machinery, energy sources, manufactured items of all kinds and even improved plants and animals. It has made the West the industrial leader of the world with the highest standard of living. Industries in the U.S. that maintain a vigorous research effort are perennial leaders in profits. Medical research is also highly cost effective and a dramatic example is poliomyelitis. In the middle of this century treatment of poliomyelitis was costing the country nearly 100 million dollars a year and we were building whole hospitals for the rehabilitation of people with paralyzed muscles. The March of Dimes invested a small percentage of this, 5 million a year, in basic research and as a result poliomyelitis is now prevented by a few cents worth of vaccine. Measles, Rh disease and rubella are now controlled by similar procedures. The rubella epidemic of 1964 left 30,000 victims with deafness, blindness, cardiac and behavioural defects that have already cost the country 2 billion dollars. Penicillin and other antibiotics permit home and office treatment of 99 percent of patients with infectious diseases due to bacteria and as a result contagious disease hospitals once maintained by every city and/or state are all closed for want of patients.

Can these triumphs of industrial and medical research be repeated for cancer? The answer is - of course - if we permit research to continue. How long will it take? The answer - until we accumulate sufficient knowledge to understand how to interrupt or reverse the cancer process. What then is the nature of scientific research and how can it be fostered?

Nature of Scientific Research

It seeks to explain biological phenomena by creating a hypothesis to explain observed facts and then tests the hypothesis by a controlled experiment rather than by debate. If the results are consistent and repeatable a new truth has been established which can be incorporated into a new hypothesis. In this manner research progresses in stepwise fashion, by small steps which frequently do not lead straight to the original goal. It is moved by curiosity and enthusiasm of those seeking explanations for an unresolved problem. Small advances are contributed by many investigators and real breakthroughs require prolonged effort and accumulation of many bits of data. New ideas and new hypothesis rarely occur to anyone not actively working on a problem and it is from interpretation of the results of one experiment that the questions to be answered by the next experiment are formulated. Hence the need for freedom to pursue the results wherever they may lead.

Kornberg has likened the flow of science to a river which flows always toward the sea[15] "Like rivers, the pace and dimensions of scientific movement vary enormously. But shallow or deep, broad or narrow, sluggish or swift, the movement is inexorably forward." "In contrast, the support of science throughout history rises and falls like the tides."

It has been a century since Pasteur established the pattern that scientific medicine has followed since. The benefits in better health are too numerous to list and have reached their greatest potential in the last quarter century with the advent of molecular biology which is developing chemical explanations for health and disease at the cellular level. Continued research is the only way that partially treated diseases can be moved into the high technology group where they can be prevented or cured. To stop research and put all effort into better delivery of what is already known would accomplish relatively little. How much would we gain in control or prevention of cardiovascular disease? Hypertension, stroke? Heart attacks? Cancer? Mental sickness? Arthritis? Kidney disease? Genetic disease, Sickle cell disease? Multiple sclerosis? Diabetes? For all these diseases we have only palliative, expensive treatment of end results but no prevention or cure such as is available for poliomyelitis or strep throat. Of course we should try to make available all that is known but keep in mind that it is the portion of funds spent on research that will lead to prevention or cure.

Advancement of knowledge through scientific research has amply demonstrated that it is the most efficient process yet devised by man for solving problems as shown by its applications in western culture. If there are problems raised by some of the solutions and if there are remaining unsolved problems and questions it seems imperative that science continue using the methods which have been proved in the past. In short, the scientific community has a clear responsibility to continue. The cost benefit ratio is favorable, and there is no valid alternative. Basic research has a long time scale, the source of innovative ideas is the individual investigator, and the cumulative efforts of many disciplines and facilities are needed for modern research. What then, are the basic requirements for a vigorous research program vs. cancer or any other unsolved medical problem?

Requirements

The requirements for productive biomedical research are well established by experience, widely recognized in the scientific community and are in fact the basis for evaluating grant proposals by all granting agencies that support cancer research. By far the most important is to identify a competent investigator to lead and conduct the research project. All other requirements are secondary and supportive. They include adequate physical facilities and equipment, stable support, freedom of inquiry, intellectual environment and competent administration.

Competent investigator: Competence is judged by past record of productivity, originality, ability to conceive new ideas, get them funded, carry through the research, publish the results and defend it before peers who then are able to confirm the new discoveries by repeating the studies with the same end results. All these qualities are found wherever innovative research is being produced. Modern cancer research involves use of techniques from many disciplines such as patient care, chemistry, pharmacology, physics, microbiology and cell culture. It is therefore most efficiently conducted if several investigators combine their various skills to create a critical mass of technical disciplines. Excellence in research is dependent on excellence and competence of the principal investigator who directs the research program.

Facilities and equipment: Each scientific discipline has special requirements for laboratory space, animal space, incubators, microscopes, supplies and technical instruments, refrigerators, etc. In many cases expensive equipment and space can be shared by several investigators which is another economy in having a critical mass of investigators working in one institution.

Stable support: The most critical element of support is for stable funding of the salary of the principal investigator so that he or she can feel secure in building a research team, applying for grant support to assemble all the equipment, supplies, personnel, animals and/or patient contacts for a successful research effort. Successful investigators bring in much additional supplementary funding so underwriting their salary is an excellent investment financially as well as for all the secondary benefits that follow a successful research program. Stability of support for the principal investigator is stressed because exceptionally competent investigators are in demand, have their choice of many job opportunities, and because biomedical research requires prolonged effort. Pasteur's advice to his students was effective 100 years ago, repeatedly confirmed during the intervening years and still appropriate here : "Allow me to give you an advice that I have always tried to follow and which consists of staying the longest possible time in one subject. Everywhere, I believe, the secret of success lies in prolonged efforts. With perseverence in research one finishes by acquiring what I would like to call the instinct for truth."

Freedom of inquiry: Having gathered the facilities, supplies, support and innovative investigator for a research project it is of utmost importance to let the investigator conduct the research in his own way. We are seeking new and innovative explanations for

cancer never before articulated and this is the specialty of the investigator chosen to lead the project.

Intellectual environment for inquiry: Where a group of investigators in an institution are conducting research on related problems, an esprit de corps and appreciation of scholarship develops which is infectious and promotes exchange of ideas, criticism, stimulation, cooperation and collaboration, the sum of which is greater than its parts.

Competent administration: The role of management in research is to bring together all the above elements and facilitate their interaction in the most productive manner. It involves measures to foster discussion and intercommunication, optimal use of facilities and equipment, cooperative planning, efficient housekeeping, maintenance of facilities, procurement of supplies and equipment, fiscal management, allocation of space, appointment of investigators and coordination with other departments, institutions or agencies. General fiscal accountability is a function of research administration. Competent research administration will have an appreciation of the requirements for innovative research and will avoid trying to direct the conduct of research. That is the function of the principal investigators.

Cancer Research in New Jersey

By applying the above guidelines to New Jersey it is not difficult to see why New Jersey has received so little of cancer research funds from NCI and other sources. New Jersey has few competent cancer investigators with time, facilities and equipment for research. It is not training adequate numbers of new investigators nor providing stable support to attract those trained elsewhere.

To correct this situation and thereby receive its fair share of cancer research funds from federal and other sources New Jersey would have to correct the deficiencies that exist in the state. Namely, provide stable funds to attract cancer investigators and provide nuclei of facilities and services where productive research can be conducted to help solve New Jersey's cancer problems. The health benefits for cancer victims of such a move are obvious and the ripple effects in other health related fields are also predictable. Would it be cost effective to spend New Jersey funds to encourage development of centers of excellence for cancer research? By referring to Table 2 we can see that New Jersey received $7,598,000 from the NCI in 1975 whereas the 6 neighboring states received on the average 4.93 times as much on a per capita basis. If New Jersey were to attract its proportionate share of the NCI cancer grants and contracts it would have recieved in 1975 $37,458,000 instead of $7,598,000, a loss of 30 million dollars in one year.

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A logical conclusion after review of all these facts is that New Jersey can ill afford to continue its present policies in this matter. Corrective measures should be adopted promptly and funded adequately

to make up for lost ground with the object of making New Jersey competitive in cancer research within the next five years. Where can New Jersey get the necessary funds? Several possible sources come to mind and a few will be mentioned.

Suggested Plans

1. Cigarette smoking is one of the major causes of lung cancer and a small tax on cigarettes would serve the dual purpose discouraging cigarette smoking, and helping to bring New Jersey's cancer research capability up to the regional average. A special 1 cent tax per package would provide a fund of approximately 8 million dollars a year which should be earmarked for cancer research in New Jersey. The present cigarette taxes in New Jersey and neighboring states are shown on Table 8. Half the fund should be allocated in designated portions to the one cancer research institute now functioning in New Jersey and to each of the three branches of the medical-dental school of New Jersey in North, Central and South Jersey and to the state university. These institutions have the personnel, experience dedication and commitment to use the funds in an optimal manner. These awards should be specified for cancer research - with special emphasis on providing stable support for competent investigators and any supportive facilities and services required, at the discretion of the scientific management of those institutions. Strict yearly accounting should be required and carry-over of unspent funds should be permitted to encourage management to make long range plans for staffing, facilities and program development. This would leave half the fund for funding approved grant applications and cancer control programs from these and other institutions within the state and to encourage awards from NCI and other federal sources.

TABLE 8

CIGARETTE TAXES PER PACK, 1976 (16)

CONNECTICUT	21¢
MARYLAND	10¢
MASSACHUSETTS	21¢
NEW JERSEY	19¢
NEW YORK	15¢ (PLUS 8¢ MORE N.Y.C.)
PENNSYLVANIA	18¢
VIRGINIA	2.5¢

NOTE: NEW JERSEY'S CIGARETTE TAX OF 19¢ PER PACKAGE GENERATED AN INCOME OF $167,000,658 IN 1975.

Committed taxes are unconstitutional in New Jersey, but the feasability of an appropriation of seed money for cancer research and a cigarette tax of identical size should be explored. It would be a sound investment of public funds and politically attractive, given a public fully aware of New Jersey's cancer problems and the shortage of staff and facilities for cancer research.

Kentucky established such a program in 1970, funded by a 5 cent specific cigarette tax on every cigarette package sold in the state. A Kentucky Tobacco Research Board was created by the legislature to supervise expenditure of the funds on the university campuses at Lexington and Louisville and elsewhere(17).

Such a massive program is not now necessary in New Jersey. A modest 1 cent per package will be adequate at this time. If care is taken in setting up the governing board to distribute the funds it can be assured that awards are made on scientific merit, which will guarantee that New Jersey will help solve its cancer problems and lay the groundwork for solution of many other unsolved medical problems which are of concern to residents of the state and the nation.

II. Other sources of funding are possible although the cigarette tax and/or a liquor tax have much appeal. Epidemiologic studies clearly implicate alcohol and tabacco in the causation of cancer of the esophagus.

III. Another potential source of funding for cancer research is voluntary contributions from industry in New Jersey. At least two large segments of industry could profit directly and indirectly from better cancer research and control in New Jersey, the chemical-pharmaceutical industry and the insurance industry. Contributions to non-profit cancer research would be tax exempt.

Funds from industry could supplement and match the NCI planning grant award to CINJ in its developmental stages and help to bridge the gap until some stable state funding is established and thereafter to provide seed money for new approaches not covered by the stable funding sources.

All the foregoing proposed renaissance for cancer research in New Jersey is not an impossible pie-in-the-sky pipedream, but rather a practical, achievable goal - which would have broad public support and predictable benefits in better health care and greatly increased federal funding to help attend New Jersey's cancer problems.

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16. Monthly State Cigarette Tax Report, Feb. 12, 1976. Tobacco Tax Council, 5407 Patterson Ave., Richmond, Va. 23226.

17. Kentucky Tobacco Research Board, 1975 Annual Review.

18. A Comparison of State and Local Tax Effort in the District of Columbia and the Fifty States. Feb. 1975. Office of Fiscal Planning and Research, Dept. of Finance and Revenue, Room 4126, Municipal Center, 300 Indiana Avenue, N.W., Washington, D.C. 20001.

Appendix 5.

State of New Jersey
Department of Environmental Protection
P.O. Box 1390
Trenton, N.J. 08625
609 292 2885

November 5, 1976

Honorable John M. Skevin
Senator
370 Kinderkamack Road
Oradell, New Jersey 07649

Dear Senator Skevin:

Thank you for the kind and thoughtful words in your letter of September 14. I assure you that the assistance and guidance that you requested will be available to you and the other members of the Senate Commission to Study the Incidence of Cancer in New Jersey whenever you deem it necessary.

I agree with you that the State of New Jersey cannot sit back and do nothing about the high rates of cancer in our state. Following the mandate of Governor Byrne's Executive Order No. 40, and working in concert with the other members of the Cabinet Committee on Cancer Control, we are already developing and implementing monitoring and regulatory activities related to this department's responsibilities for air, water and solid waste.

In your letter you state that you are "drafting legislation which would list suspected carcinogens and set limits beyond which their emission into the air would be illegal". Legislation directed to the goal of reducing the exposure of New Jersey residents would be a clear signal to the people of New Jersey of the concern with which the legislature views the issue of cancer in New Jersey.

You requested that I indicate to you any suggestions or recommendations concerning your proposal. While I support your goal and intent, I offer the following comments for your consideration regarding your specific proposal:

1. Legislation which lists specific substances and establishes emission limits requires that a specific technical change in any one of the detailed specifications would require action by the Legislature. I suggest that the legislation empower the implementing department to determine the substances to be controlled as well as the specific emission limitations. The department would

therefore have the flexibility to respond to scientific and technological advances by changing the substances listed or the emission standards.

2. The legislation should not be confined to known or suspected carcinogens, but should be sufficiently flexible to include other substances that adversely affect public health, such as substances that are mutagenic or teratogenic (cause genetic changes or birth defects).

3. I suggest that the legislation not be confined to emissions into the air, but include emissions into any part of the environment including water and land.

4. Giving a company sixty days to stop all emissions does not allow sufficient time to purchase and install control equipment, place it into operation, or to introduce necessary process changes.

5. The substances to be controlled have different physical and chemical properties, are emitted into different media such as air and water, and as a result, widely differing technologies must be used to control emissions. In some cases, the control technology may not be fully developed yet. As a result, certain types of emissions may be controlled more quickly and readily than others. It may therefore be more effective to:

a. establish a goal of maximum reduction of the emissions of carcinogenic substances into the environment;

b. reduce to zero the emissions of certain substances known to be carcinogenic to humans, and

c. require the use of best available control technology for the control of the emissions of other carcinogenic, toxic and hazardous pollutants.

In your letter you also requested that I submit to the Commission our needs for equipment, testing facilities and manpower. You suggested that we may want to look at our needs in terms of a short-term (start-up) program and a long term program.

The short term needs of the DEP cancer program have already been defined in our budget requests. For this fiscal year, I requested $2,300,000, of which $500,000 was appropriated; I have since submitted a request for a supplemental appropriation of $1.8 million.

We will utilize our current appropriation of $500,000 for:

a. chemical monitoring of air, water, sediments and biological organisms,

b. biological testing of air and water samples for cancer-causing potential,

c. survey of the production, use and emission of cancer-causing agents in New Jersey, and

d. regulatory planning and implementation including feasibility and costs of reducing human exposure to carcinogens.

The short term continuation and expansion of the program that we have started this year can be accomplished with the resources I have requested including the $1.8 million supplemental appropriation.

For the long term, additional resources will be needed. The resources that we will need include: sampling and analytical equipment, office and field personnel, ancillary and support equipment, and facilities to house the equipment and personnel. A brief description of the programs and resources follows:

1. One of the primary requirements for the development and implementation of regulatory programs is the ability to determine the presence of carcinogenic substances in the environment, and to monitor the effluents of specific facilities. This requires people and equipment to gather the samples in the field, as well as to chemically analyze the samples taken. Each of the media to be analyzed necessitates different sampling equipment, and appropriately trained personnel.

Analysis of the samples obtained is difficult technically, time consuming and expensive. Complex and sophisticated equipment is required and must be run by experienced analytical chemists.

2. In order to define the extent of carcinogenic emissions into the environment, it is necessary to determine the spatial density of use and emissions. Such an assessment is labor intensive, and requires the development of the necessary data handling and storage systems.

5. In order to develop regulatory programs, it is also necessary to determine and understand the deleterious health effects resulting from the presence of toxic substances in the environment. Much of the basic research in this area is currently being done by agencies of the federal government and agencies affiliated with the U.N. This information must be evaluated in light of usage and emissions in New Jersey.

The list of suspected carcinogens published by the National Institute of Occupational Safety and Health contains about 1500 substances. The list contains all substances for which any information exists that they may be carcinogenic. The International Agency for Research on Cancer of the World Health Organization has reviewed the information available about these substances and has published its results in a nine-volume work.

These publications and others, are being utilized to prepare a draft list of substances of concern to New Jersey. I will send a copy of this list to you and the other members of the Commission as soon as the draft is ready.

4. Some relatively non-toxic substances emitted into the environment may react with other substances to form carcinogens; nitrosamines are one such example. Chemical analyses and theoretical chemical evaluations are required to determine the possible reactions that may occur. Personnel with advanced chemical training are required for this effort, as is a data processing capability.

5. To determine the extent of the regulatory programs required, we must determine the community exposures to carcinogenic substances and the resulting risks. This determination will entail the meshing and correlation of information from chemical and biological monitoring, industrial surveys, effluent data and data on health effects. This requires personnel with scientific expertise in toxicology, physiology, chemistry, biometrics and data processing.

6. The ultimate goal of the DEP cancer program is the reduction or elimination of the emissions of carcinogens into the environment, and to thereby decrease human exposure and risk. Development of such regulatory programs is complex, and must take a number of factors into account, including:

1. the information available on health effects of the substance;

2. the presence or absence of these substances in the environment;

3. the amounts of these substances emitted into the environment by various routes;

4. the availability of control technology;

5. the cost of installation and utilization of such technology;

6. the benefit to be derived

This, too, will require personnel able to evaluate such data and information in order to develop the necessary programs.

Finally, during the coming year, we will be requesting legislation to enable us to accept from the federal government implementation and enforcement of federal regulations related to emissions of toxic substances and reduction of exposure to these substances. This legislation will deal largely with emissions into water and the maintenance of water quality. An example of such legislation is S-1222 introduced by Senator McGahn, which will enable us to accept enforcement responsibility for the National Pollution Discharge Elimination System. We are currently developing and defining other such legislation and will confer with you and the other members of the Commission concerning our needs in the near future.

Faithfully,

David J. Bardin
Commissioner

RSV 0013550

Appendix 6.

STATE OF NEW JERSEY
DEPARTMENT OF HEALTH
TRENTON, N. J. [illegible]

JOANNE E. FINLEY, M.D., M.P.H.
[illegible]

September 8, 1976

Honorable John M. Skevin
Chairman, Senate Commission on Cancer
370 Kinderkamack Road
Oradell, New Jersey 07649

Dear Senator Skevin:

I have your important and meaningful letter of August 13 in which you repeat your determination to do all you can as a Legislator to reduce New Jersey's alarming cancer death rate. I certainly believe you are on the right track when you seek to emphasize action which is aimed at reducing--or preferably eradicating human exposures to carcinogenic agents. This is the essence of prevention. I hold the deeply felt belief that many cancers, once they are established in the human body, are relatively incurable, or certainly foreshorten life and its quality. Therefore, preventive measures hold far more hope for a significant impact on this costly and painful problem, than does the present national policy of almost total emphasis on more and more dollars for equipment and facilities for mere treatment and palliation.

Martin T. Zanna, M.D., Acting Director, Chronic Diseases Control Service, New Jersey Department of Health, will appear before your Commission on September 10, 1976 at 10:30 A.M. to offer testimony in my behalf. I regret that an important meeting with the New Jersey Hospital Association which I have been unable to change, prevents my personal appearance. However, Dr. Zanna and I have discussed his testimony and it will be completely consistent with the views, policies and expertise of this Department.

In the meantime, I have attached some answers to the questions you have raised in your letter.

Most sincerely,

Joanne E. Finley, M.D., M.P.H.
State Commissioner of Health

JEF:mlt

RSV 0013551

STATE OF NEW JERSEY
DEPARTMENT OF HEALTH
TRENTON, N.J.

JOANNE E. FINLEY, M.D. M.P.H.

September 8, 1976

TO: The Senate Commission on Cancer
Senator John M. Skevin, Chairman

FROM: Joanne E. Finley, M.D., M.P.H.
State Commissioner of Health

SUBJECT: Questions and Answers on Environmental Carcinogenesis

1) What chemicals are suspected as carcinogens? Where is there a list? How complete is it?

The National Institute of Occupational Safety and Health (NIOSH) has produced a rather limited list of 14 chemical substances for which there is enough evidence of potential carcinogenesis for OSHA to set standards covering the necessary and required protection of workers and the occupational environment. NIOSH is an arm of the Department of Health, Education, and Welfare, administratively within the Center for Disease Control. Its responsibility under the Occupational Safety and Health Act is, among other things, to use expert input to sift data on chemical and other toxic substances. Where the evidence of human hazard meets acceptable scientific criteria, NIOSH proposes standards for the protection of those exposed to these substances. The emphasis of protection is on workers because their exposure is the most intense, and of the longest duration. In the case of cancer, animal and epidemiologic research proves the degree and duration of exposure to be of major importance in the likelihood of developing cancer.

OSHA is an arm of the Department of Labor, and it is responsible for the promulgation of the standards NIOSH recommends, and for their enforcement.

The list of 14 substances, and some indication of the industries or processes in which they are used, appears below:

KNOWN (OR PROBABLE) CARCINOGENS AND THEIR USES

AGENT	USES
4-Nitrobiphenyl	Analytical standard
Alpha Naphthylamine	In manufacture of herbicides, dyestuffs, food colors and color film, paint, plastics, rubber and petroleum products.
4, 4-Methylene bis (2 - chloroaniline)	Curing agent for epoxy and other polyurethane resins.
Methyl chloromethyl ether	In manufacture of ion exchange resins, textiles, and drugs.
3, 3-Dichlorobenzidine	In manufacture of printing inks, dyes, plastics and crayons.
Bis-chloromethyl ether	In manufacture of ion exchange resins.
Beta Naphythylamine	In manufacture of dyes and pesticides, in photography, and as a chemical reagent.
Benzidine	In production of dyes, rubber and plastics, printing ink, fire proofing and in medical laboratories.
4-Aminodiphenyl	As an antioxidant in rubber manufacture and as an intermediate chemical in dye production.
Ethyleneimine	In paper and textile industries in herbicides, resins, drugs, and jet fuel.
Beta Propiolactone	In plastic manufacture.
2-Acetylaminofluorene	Potentially as a herbicide
4-Dimethylaminoazobenzene	As a dye.
N-Nitrosodimethylamine	As an industrial solvent and in synthesis of rocket fuel.

It should certainly be clear that 14 substances do not comprise a complete list. NIOSH actually has published a list of over 12,000 toxic substances many of which are felt to be potentially capable of inducing cancer. But the wheels for turning out relative scientific certainty grind slowly. Also OSHA has been slow to promulgate many standards which NIOSH has drawn. Therefore, for a State to succeed in intelligent control of all carcinogenic chemicals, a national process must be speeded up.

It should also be stressed that chemical agents believed to cause cancer, are placed on these various lists after they are in use, and after much testing and research has taken place. There is no mechanism at work in this country at this time to list a substance, or forbid its use, or emission before it is introduced into the environment. The federal Toxic Substances Control Act would correct this to some extent.

2) What makes a pollutant suspicious? What tests are conducted? How sure are we the chemical is dangerous?

The main agency for conducting tests, directly, or through contracts with reputable researchers, is the National Institute for Environmental Health Sciences (NIEHS), also a part of HEW. Their criteria documents are made available to both EPA and NIOSH.

The testing may be based on three different, but related methods. There must be strong epidemiologic evidence. Animal studies are used to confirm epidemiologic hypotheses by substantiating the dangers to living organisms very similar to man. Relatively quick screening tests using bacterial systems (mutagenesis testing) can also assay a substance's carcinogenic potential.

All the tests must be statistically significant to the highest degree. In other words, all variables other than the exposure to the substance, that could explain the cancer's occurrence consistently, must be ruled out.

This is a scientific method for associating a cause and effect, and it is a reliable method. If anything, scientific research is so cautious, that we can be said to lean over backwards to prove a danger before a substance gets on a list. We may, therefore, be missing many substances that do indeed harm mankind.

3) What technology is available for controlling air pollution? Are there efficient control devices? Are they cost/effective?

EPA and the New Jersey Department of Environmental Protection would know the technology for air pollution control.

However, we again wish to stress that those who work inside of industrial plants with these substances are at the greatest risk of cancer. The New Jersey Department of Health has copies of all applicable OSHA standards. We are therefore aware of the recommended devices and methods for protecting the internal environment and the workers in it. Many of these methods and devices appear to be less costly--and more effective--than the technology of air pollution control.

JEF:mlt

Appendix 7.

State of New Jersey

DEPARTMENT OF ENVIRONMENTAL PROTECTION
JOHN FITCH PLAZA, P. O. BOX 1390, TRENTON, N. J. 08625

SELECTED ENVIRONMENTAL CARCINOGENS

- DRAFT #1

Group I List.....Page 1
Group II List.....Page 39

October, 1976

Program on Cancer and the Environment

Peter W. Preuss, Ph.D.
Glenn Paulson, Ph.D.

RSV 0013556

Acknowledgments

David Schrier and Paul White contributed greatly to the preparation of this list, the formulation of the use-dose rating concept, and the compilation and review of the available scientific information about Cancer and the Environment which stands behind this document.

Without the care and effort of Helen Grover, this document would not have been completed in a timely manner. Ms. Grover's efforts, coupled with those of Linda Watson and Debbie Giberson have ensured the widest possible public review of this publication.

INTRODUCTION

One of the major goals in the initial phase of the Department of Environmental Protection program on Cancer and the Environment has been the selection of a number of cancer-causing chemicals (carcinogens) of potential concern to the residents of New Jersey. These chemicals, compiled in this document, will be the basis for a survey of the use of these carcinogens and for future environmental monitoring activities.

There are many sources of information about cancer and carcinogenesis, ranging from original research reports to review documents. This list was not compiled from the primary scientific literature and journals, but rather from reviews prepared by national and international scientific organizations.

The main compilation of known and suspected carcinogens used (Suspected Carcinogens" A Subfile of the NIOSH Toxic Substances List) (7), contains information concerning more than 1500 substances. This list contains a much smaller number of substances. It is limited to those that may result in non-occupational environmental exposure and that are used or produced by industrial facilities. Thus, a number of potentially important classes of compounds are excluded. Examples of excluded classes include: carcinogens that may be formed through secondary reactions in the environment, (such as certain nitrosamines); chlorinated hydrocarbons, food additives and drugs; and the substances produced in industrial or fuel-burning processes, (such as the polycylic aromatic hydrocarbons produced during the combustion of fuel).

Three major criteria were used for selecting the substances included here:

1. The substance had to be a proven carcinogen in either animals or man, and, in addition,

2. The substance had to be of wide-spread use, or,

3. The substance had to be a particularly potent carcinogen

The list is broken down into two groups according to what is known about usage of the substances. The first group consists of those substances which available information indicates are used in quantities greater than 200,000 kilograms (kg) per year (about 440,000 pounds per year) in the U.S. The second group consists of those substances which are used in lower quantities or for which usage information is not readily available.

The list was prepared using a variety of information sources. The sources most used were:

1. The nine volume Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man published by the International Agency for Research on Cancer (IARC), an agency of the World Health Organization (1). Wherever possible, summaries from these volumes are included in the list and are identified as IARC.

This review was prepared by an expert committee which evaluated the available literature in order to determine whether the current state of research allowed a conclusion as to whether any given chemical was carcinogenic. The IARC evaluation is accepted as definitive by many U.S. agencies dealing with carcinogens. For instance, the National Cancer Institute states, "These monographs constitute the most authoritative source available on carcinogenicity widely distributed to governments and research agencies." Both the Occupational Safety and Health Administration and the National Institute of Occupational Safety and Health have used the monographs to help define their priorities in reviewing chemicals for regulation.

In addition to evaluating the evidence concerning human and animal carcinogenicity, the IARC reviews present a summary of what is known about the patterns of use for each chemical and frequently give an estimate for total U.S. production. These documents also discuss the environmental occurrence of the chemicals, although this information is frequently sparse.

2. Suspected Carcinogens: A Subfile of the NIOSH Toxic Substances List is a comprehensive listing of carcinogenic chemicals (7)

This subfile contains over 1500 suspected carcinogens and gives the dosages at which each listed chemical has produced tumors. However, unlike the IARC Monographs, the subfile does not evaluate the studies quoted. The NIOSH Subfile was used for dosage information in some of the summaries included in the list. This subfile is identified in the list as "Suspected Carcinogens".

3. NIOSH frequently publishes "alerts" about chemicals that it has found to pose an occupational hazard. These include a careful review of the dangerous properties (carcinogenic or otherwise) of the chemical, as well as a detailed discussion of what is known about the usage of the chemical. These alerts also provided information used in the preparation of the list (2). These alerts are identified in the list as "Hazard Reviews".

4. The National Cancer Institute publishes bulletins summarizing information about compounds it has studied; see , for example, reference 8. Dosage information for this list was sometimes taken from these reports.

5. NIOSH Criteria documents, detailed studies of the occupational hazards posed by compounds, were used when available for a few of the chemicals on the list; see, for example, reference 5.

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The most intrinsically difficult part of this effort was the attempt to evaluate the carcinogenic potency of these substances. The information required was the dosage that is carcinogenic to humans. Since this is available for only a limited number of chemicals, we have attempted to derive some indication of the potency from the data obtained from animal experiments.

It is difficult to extrapolate results from experiments on animals to humans. Further, it is not possible to readily devise a uniform scale of potency in animals. Chemicals have been tested in a wide range of animal species and in a wide range of strains within a given species. In addition, chemicals have been tested by a wide range of routes of administration, (i.e., orally, by inhalation, etc.) and effective doses by these different routes can vary greatly.

Because of these variations, no linear scale which ranks carcinogenicity can be very accurate. Given all these problems, we have chosen to use the smallest total dose that has been found to be carcinogenic in any species by any route as an indicator. This quantity is given for each of the chemicals on our list (if available). We also list the lowest doses that are carcinogenic in any species by the oral and inhalation routes, since these are the predominant means of human exposure to environmental chemicals. These dosages permit some judgement to be made as to those chemicals that may be of particular concern.

A further indication of the potential risk posed by a chemical is given by a scale that we have devised, the use-dose rating. This rating is calculated by dividing the quantity of the chemical produced in the U.S. (expressed in millions of kg) by the smallest carcinogenic dose of the chemical in any species by any route (expressed in grams). Thus, a chemical that has a production of one million kg and is carcinogenic at a dose of one gram has a use-dose rating of 1. Like the carcinogenic dose itself, the use-dose rating is not a firm linear rating of the chemicals, but rather is an indicator to aid in the determination of substances of concern.

This list is not intended as a setting of priorities; rather it is a first attempt at locating the environmental carcinogens of greatest concern. We expect that the list will be expanded as more information becomes available.

Finally, careful consideration must be given to the groups of carcinogens excluded from this list: those that are not produced industrially; those formed by reactions of chemicals in the environment; and those produced inadvertantly by man's activities. These

RSV 0013560

include, for example, the polycyclic and heterocyclic hydrocarbons which occur when organic matter is burned, nitrosamines, and certain chlorinated hydrocarbons.

This list will be utilized as a major component of two other efforts: a carcinogen use survey and a chemical monitoring program. The survey, to be done in conjunction with the Department of Labor and Industry, will provide information concerning the use, production and emission into the environment of the substances on the list. The chemical monitoring program will provide information as to the ambient concentrations of these substances in the environment as well as the concentrations emitted.

RSV 0013561

GROUP I

Production or Usage Greater than 200,000 kg/year

AMITROLE
ARSENIC and COMPOUNDS
ASBESTOS
AURAMINE
BENZENE
*BENZIDINE
BERYLLIUM and COMPOUNDS
CADMIUM and COMPOUNDS
CARBON TETRACHLORIDE
CHLOROFORM
CHLOROPRENE
CHROMIUM and COMPOUNDS
CHRYSOIDINE
[redacted]
ortho and para-DICHLOROBENZENE
*3,3'-DICHLOROBENZIDINE
3,3'-DIMETHOXYBENZIDINE
3,3'-DIMETHYLBENZIDINE
1,1-DIMETHYLHYDRAZINE
ETHYLENE DIBROMIDE
*ETHYLENEIMINE (AZIRIDINE)
HYDRAZINE
LEAD and COMPOUNDS
*4,4'-METHYLENE BIS(2-CHLOROANILINE)
4,4'-METHYLENE DIANILINE
*alpha-NAPHTHYLAMINE
*beta-NAPHTHYLAMINE
NICKEL and COMPOUNDS
POLYCHLORINATED BIPHENYLS (PCBs)
SUDAN I (Solvent Yellow 14)
THIOUREA
TRICHLOROETHYLENE
VINYL CHLORIDE MONOMER

*Regulated by OSHA as a carcinogen

AMITROLE 3-Amino-1,2,4-triazole

Usage

The major commercial use for amitrole in the U.S., has been as a herbicide. It is also used as a photograhic reagent and ingredient in some hair preparations. A cancellation order for use on crops was issued in 1971 by the Department of Agriculture. It is not produced in the U.S. Imports in the first nine months of 1973 were 406,000 kg.

Dosage Levels

Oral-rat-100 ppm in diet showed a 55% thyroid adenoma incidence; total dose - 2.0 g.

Use Dose Rating

0.2

IARC Summary

Animal data

Amitrole induced thyroid and liver tumors in both mice and rats following oral and/or subcutaneous administration. An increased incidence of liver-cell tumors in the trout has also been reported following oral administration, but this cannot be considered as conclusive until additional studies using properly controlled diets are reported. Limited skin-painting studies in mice gave no evidence of skin carcinogenicity.

Human data

A single, small cohort study raises the suspicion that amitrole may be carcinogenic to man, but the findings cannot be regarded as conclusive.

References:

IARC 7:31
Dangerous Properties of Industrial Materials

RSV 0013563

ARSENIC AND COMPOUNDS

Usage

In 1971, U.S. consumption of arsenic metal was 0.49 million kg. Almost all of this was imported from Sweden. Total production of arsenic pentoxide in the U.S. for 1971 was 0.6 to 0.7 million kg. U.S. production of arsenic trioxide in 1971 was 15 million kg, while imports of calcium arsenate amounted to 0.43 million kg.

Dosage Levels

IARC states that animal studies are either inadequate or gave negative results.

Special Comments

Human data point to a causal relationship between skin cancer and arsenic exposure.

IARC Summary

Animal data

Many studies have given essentially negative results, but most of them are not referred to in this monograph because of inadequacies in the experimental design (e.g., too few animals, too short a duration, poor survival, too low a level of exposure).

Adequate oral studies on arsenic trioxide in the mouse and on lead arsenate, calcium arsenate, sodium arsenate, arsenic trioxide and sodium arsenite in the rat gave negative results.

The studies designed to detect cocarcinogenicity to mouse skin by potassium arsenite, sodium arsenate or arsenic trioxide gave negative results.

The two recent preliminary reports suggesting possible carcinogenic effects in mice exposed to sodium arsenate, potassium arsenite and arsenic trioxide by subcutaneous, intravenous, oral and transplacental routes are difficult to interpret on the basis of the findings presented, and the results await confirmation.

Human data

The available studies point consistently to a causal relationship between skin cancer and heavy exposure to inorganic arsenic in drugs, in drinking-water with a high arsenic content, or in the occupational environment.

The risk of lung cancer is clearly increased in certain smelter workers who inhale high levels of arsenic trioxide. However, the causative role of arsenic is uncertain, since the influence of other constituents of the working atmosphere cannot

RSV 0013564

be determined. An increased relative frequency of deaths from lung cancer has been found in other occupational groups exposed to high levels of inorganic arsenic compounds (e.g., sheep-dip workers, certain mining and vineyard workers).

Cases of lung cancer occurring after the medicinal use of inorganic arsenic compounds, and of liver haemangioendothelioma following various kinds of exposure to arsenic have been reported, but these may be chance associations.

No evidence exists that other forms of cancer occur excessively with heavy arsenic exposure.

References:

IARC 2:48
Criteria Document - arsenic

ASBESTOS

Usage

Asbestos has been used for thousands of years. Most present day applications of asbestos are in the building industry to strengthen cement and plastics. It is also used for heat insulation and sound absorption. It is essential in brake shoes and clutch plates. Asbestos cloths are used extensively for fire protection. Chrysotile accounts for over 95% of asbestos used. Other forms of commercial importance are anthophyllite, amosite and crocidolite. The world production of asbestos now exceeds 3.6×10^9 kg. per annum. U.S. consumption in 1974 was 7.4×10^8 kg.

Dosage Levels

Inhalation - rat - 86 mg/m^3 for 30 hrs. a week for 18 months caused lung carcinomas from chrysotile.

Intrapleural administration - rat - 20 mg - mesotheliomas.

Use Dose Rating

37,000

Special Comments

In humans, mesotheliomas have occurred after short exposures to asbestos and in those exposed at home to dusty clothing or to a neighboring source of asbestos air pollution.

IARC Summary

Animal data

Injection of asbestos into the pleural cavity has demonstrated that all major commercial forms can produce mesotheliomas. Experiments suggest that this is probably not due to contaminants such as oils and waxes or heavy metals. It is more likely that the size and shape of the particles are the main factors. Thin, long fibres (less than 0.5 um diameter and 10 um in length) seem to be most active in producing tumors. Fine glass fibres of similar diameter can also produce mesotheliomas. The carcinogenicity decreases as the materials are pulverized. Inhalation experiments in rats, guinea-pigs and monkeys can produce fibrotic lesions in the lung and pleura similar to those found in man. By inhalation, mesotheliomas and lung cancer carcinomas have been produced in a small proportion of rats exposed to the four commercial types of asbestos.

Human data

There is substantial evidence that the risk of lung carcinoma and mesothelioma is small in workers in chrysotile mines and mills, and the same is possibly true for amosite. Some crocidolite mining areas and mills have been associated with a higher risk of mesothelioma. Communities in the neighborhood of these mines have had, in some instances, an appreciable exposure to asbestos dust. Mesotheliomas have been observed in these populations.

Industrial exposures to asbestos have usually been to mixed types of fiber, especially where manufacturing and application are undertaken, for example, textiles, insulation and asbestos cement, and have also occurred in the immediate vicinity. Mesotheliomas have occasionally been diagnosed among families of asbestos workers.

An important excess risk of lung cancer has usually resulted from past heavy exposures. The differences in risk between the several parts of the industry cannot be ascribed to one factor. The type of fiber, past dust levels, the form of dust produced by the process and the length of exposure are all relevant. The risk of lung carcinomas seems to be related to asbestosis.

In manufacturing and application industries mesotheliomas have been caused by exposure to crocidolite, and less frequently to amosite and chrysotile. The period between first exposure and development of tumors is long, usually more than 30 years. The tumors can occur in the absence of other asbestos-related disease.

At the present time, there is no evidence that exposure of the general population to past levels of asbestos dust in the ambient air or in beverages, drinking-water, food or pharmaceutical preparations increased the risk of cancer.

Cigarette smoking enhances the risk of lung carcinoma in asbestos workers to a much greater degree than in the rest of the population.

Reference

IARC 2:17

RSV 0013567

AURAMINE Tetramethyl-p-diamino-imido-benzophenone

Usage

Production is unknown but probably high. It is used in the coloring of paper, cardboard, textiles, leather and in the preparation of solvent yellow 34. Auramine is also used as an antiseptic.

Dosage Levels

Subcutaneously - rat - 0.51 g/kg/21 wks. Total dose - 0.11g.-55% tumor incidence

Oral - mouse - 29g/kg/52 weeks; total dose - 0.73g.; 60% tumor incidence

Use Dose Rating

On the basis of 200,000 kg production in the U.S., 1.8

IARC Summary

Animal data

Auramine is carcinogenic in the mouse and rat. Given orally, it has produced liver tumors in these two species. No tumors were obtained in the only experiment in the dog and in the rabbit. The purity of the auramine used in these experiments is not known.

Human data

One epidemiological study indicates that the open manufacture of auramine presents an occupational bladder cancer risk.

Reference

IARC 1:69

RSV 0013568

BENZENE

Usage

U.S. 1973 production was 4,649 million kg. In 1971, 44.2% was used for production of ethylbenzene, 19.2% for phenol, 15.6% for cyclohexane, 3.9% for maleic anhydride, 3.9% for detergent alkylate, 1.1% for dichlorobenzene, 0.5% for DDT and 8.1% miscellaneous.

Dosage Levels

The animal experiments done so far have not been conclusive. One study suggested carcinogenicity at the following dose: subcutaneously - mouse - 8g/kg; total dose - 0.2g.

Use Dose Rating

Based on 0.2g, 23,250

Special Comments

Benzene is found in air around gasoline stations and other gasoline handlers in concentrations from 0.3 to 6.7 ppm. The average concentration of benzene is ambient air in Los Angeles was found to be 0.015 ppm in 1968. OSHA regulations had set a time weighed average value of 10 ppm and a ceiling concentration of 25 ppm. NIOSH on 8-19-76 recommended a standard of no greater than 1 ppm. Accumulated evidence from clinical as well as epidemiological data is now considered by NIOSH as conclusive that benzene is leukemogenic.

IARC Summary

Animal data

Benzene has been tested only in mice by subcutaneous injection and skin application. The data reported do not permit the conclusion that carcinogenic activity has been demonstrated.

Human data

It is established that exposure to commercial benzene or benzene-containing mixtures may result in damage to the haematopoietic system. A relationship between such exposure and the development of leukemia is suggested by many case reports, and this suggestion is strengthened by a case-control study from Japan.

References:

IARC 7:203
"Update Criteria and Recommendations for a Review Benzene Standard", August, 1976

BENZIDINE

Usage

In 1972, 700,000 kg. were produced in the U.S. It is used in the production of more than 250 dyes.

Dosage Levels

Subcutaneous - mouse - 8g/kg 32-52 weeks; total dose - 0.2g; oral - rat - 5.4 g/kg/60 weeks; total dose - 1.08g.

Use Dose Rating

3.5

Special Comments

Benzidine is regulated by OSHA as a carcinogen. It is a human bladder carcinogen.

IARC Summary

Animal data

Benzidine is carcinogenic in the mouse, rat and hamster, and possibly the dog. Given orally, it has produced bladder carcinoma in the dog after a long latent period and liver tumors in the rat and hamster.

Human data

The epidemiological studies showed that occupational exposure to commercial benzidine alone was strongly associated with bladder cancer. In the same studies, exposure to 2-naphthylamine alone was similarly associated with bladder cancer. A number of case reports from several countries support the relationship between this neoplasm and occupational exposure to benzidine.

References:

IARC 1:80
Hazard Review

BERYLLIUM AND COMPOUNDS

Usage

Beryllium is used as an alloy with copper, aluminum and nickel. It is used in ceramics and vitreous enamel, refractory crucibles, textile fibers, nuclear reactors, space vehicles and rocket motors.

Dosage Levels

Beryllium sulfate - inhalation - rat - 34/ug/m^3/up to 56 weeks
Beryl ore dust - inhalation - rat - 15 mg/m^3/30 hrs/week up to 17 months;
Beryllium fluoride - inhalation - rat - 68 mg/m^3

Special Comments

Rocket exhaust fumes may contain berllium oxide, fluoride and chloride in significant-amounts.

IARC Summary

Animal data

Experimental evidence for the carcinogenicity of beryllium, beryllium salts or beryl exists for three animal species. In particular, beryllium sulfate, beryl ore and bertrandite produce lung tumors in the rat following inhalation exposure; beryllium oxide and beryllium sulfate produce tumors in the monkey following intrabronchial implantation or inhalation; zinc beryllium silicate, beryllium metal and beryllium phosphate produce bone tumors in rabbits following i.v. administration.

Human data

Several epidemiological studies have been carried out on the possible relationship between exposure to beryllium compounds and the occurrence of cancer in man. These studies have not provided evidence of the existence of such a relationship.

References:

IARC 1:18
Suspected Carcinogens

RSV 0013571

CADMIUM AND COMPOUNDS

Usage

U.S. consumption for all forms of cadmium in 1971 was 4.1 million kg. Cadmium and compounds are used in electroplating, plastics stabilizers, pigments, alloys and batteries.

Dosage Levels

cadmium - intramuscular - rat - 70 mg/kg; total dose - 0.014g;
cadmium oxide - subcutaneous - rat - 90 mg/kg; total dose - 0.018g;
cadmium sulfate - subcutaneous - rat - 2 mg/kg; total dose - 0.0004g;
cadmium sulfide - subcutaneous - rat - 90 mg/kg; total dose - 0.018g

IARC Summary

Animal data

Single or repeated s.c. injections of several inorganic cadmium compounds (cadmium chloride, sulphate, sulphide and oxide) result in the development of injection-site sarcomas in the rat. Local tumors were also produced in rats by i.m. injection of cadmium powder and cadmium sulphide.

Interstitial-cell tumors of the testis were found in rats and mice given s.c. injections of soluble cadmium salts (cadmium sulphate and cadmium chloride). The testicular tumors in both species were of interstitial-cell origin and were only seen following testicular atrophy. The putuitary glands of these animals showed castration changes. It is likely, therefore, that the testicular tumors developed as a result of an indirect action of cadmium on the testis. Repeated s.c. injections of cadmium sulphate in the rat failed to produce neoplastic changes in the prostate gland.

The negative results obtained when cadmium acetate was administered orally to rats and mice are not acceptable as evidence of the noncarcinogenicity of this compound since only one low dose level was used.

No conclusions could be drawn from the negative findings obtained in inhalation studies on rats with cadmium oxide and cadmium chloride fumes because of the short duration of the experiments.

Human data

Two studies suggest that occupational exposure to cadmium oxide may increase the risk of prostate cancer in man, but the size of the groups examined was too small to allow definite conclusions to be drawn.

No data are available to suggest that non-occupational exposure to cadmium constitutes a carcinogenic hazard.

References:

IARC 2:74
Suspected Carcinogens

CARBON TETRACHLORIDE

Usage

U.S. production in 1970 amounted to 455 million kg. Of the total production, 69% was used to produce dichlorodifluoromethane, 29% for trichloromethane and the remainder used in grain fumigants, fire extinguishers, solvents and degreasing agents.

Dosage Levels

oral mouse 4.8g/kg; total dose 0.12g

Use Dose Rating

3800

IARC Summary

Animal data

Carbon tetrachloride has produced liver tumors in the mouse, hamster, and rat following several routes of administration, including inhalation and oral ingestion. The results in trout were of borderline significance. There is no evidence of carcinogenicity for organs other than the liver, but most experiments were shorter than the life-span of the animals. No single-dose carcinogenic experiment with long follow-up has been reported.

Human data

Man is exposed to carbon tetrachloride from several sources involving a wide range of doses. No long-term follow-up studies on men exposed to carbon tetrachloride have been reported. The occasional case reports of liver tumors in man following acute intoxication are of doubtful significance, but cannot be disregarded.

References:

IARC 1:53

RSV 0013574

CHLOROFORM

Usage

In 1974 U.S. production was 135 million kg. Most is used in the production of fluorocarbons. Chloroform is extensively used in the drug industry but chloroform containing drugs have been banned by the FDA.

Dosage Levels

oral - mice - 46.4g/kg; total dose - 1.16g

Use Dose Rating

117

Special Comments

There is no known association between chloroform and human cancer. Chloroform has been shown to be carcinogenic by ingestion in laboratory mice and rats in a report released by the National Cancer Institute.

IARC Summary

This summary preceeds the National Cancer Institute study.

Animal data

The carcinogenicity of chloroform has been investigated only in mice in experiments involving a small number of animals at risk. Nevertheless among these the frequency of liver tumors was high. There is no evidence of carcinogenicity for organs other than the liver, but the experiments were shorter than the life-span of the animals. An experiment involving single or a few exposures of newborn mice gave negative results. An assessment of the carcinogenicity of chloroform awaits further experimental evidence.

Human data

Chloroform entails several sources of exposure for humans. No longterm follow-up studies in men exposed to chloroform have been reported.

References:

IARC 1:61
"Current Intelligence Bulletin-Chloroform"

CHLOROPRENE

Usage

Choloroprene is used to produce neoprene. It is estimated that 2,500 U.S. workers are exposed to chloroprene. In 1973 neoprene production was 155 million kg.

Dosage Levels

No dosage levels are available although Russian studies have shown chloroprene to be carcinogenic in rats.

Special Comments

Russian epidemiological studies have shown chloroprene to be associated with skin and lung cancer in humans.

IARC Summary

Not reviewed by IARC.

Reference:

Background Information on Chloroprene

CHROMIUM AND COMPOUNDS

Usage

Production of chromium metal and metal alloys other than with iron was 14 million kg. in the U.S. in 1970. Sodium dichromate is the principal commercial product from which other chromium compounds are made. Most of it is used in pigment production.

Dosage Levels

calcium chromate - implant - rat - 25 mg/kg; total dose - 0.005g;
chromium oxide green (+3) - intratracheal - rat - 100 mg/kg; total dose - 0.02g;
lead chromate - subcutaneously - rat - 150 mg/kg; total dose - 0.003g;
chromium oxide (+6) - implant - rat - 125 mg/kg; total dose - 0.025g.

IARC Summary

Animal data

In many experiments, various chromium compounds have been shown to induce tumors in mice and rats. Calcium chromate has been found to be carcinogenic by several routes of administration, producing epithelial tumors of the lung by intrabronchial implantation and sarcomas by intramuscular and intrapleural administration to rats.

Of the other chromium salts tested in the rat by intramuscular and intrapleural administration, chromic chromate and zinc chromate hydroxide were highly evocative of sarcomas at the site of injection in the rat, while barium chromate, lead chromate, chromic acetate and sodium dichromate were inactive or practically inactive. Strontium chromate was tested only by intramuscular implantation and evoked many local sarcomas.

Studies involving oral administration of chromic acetate in mice and rats which gave negative results were considered inadequate because the level of exposure was low.

Human data

There is an excessive risk of lung cancer among workers in the chromate-producing industry. It is likely that exposure to one or more chromium compounds is responsible, but the identity of this or these is not known.

There is no evidence that non-occupational exposure to chromium constitutes a cancer hazard.

References:

IARC 2:100
"Background Information on Chromium"

RSV 0013577

CHRYSOIDINE, 4-(Phenylazo) - 1,3-benzenediamine, monohydrochloride

Usage

U.S. production in 1972 amounted to 203,000 kg. It is used as a colorant in textiles, paper, leather, inks and wood and biological stains.

Dosage Levels

Oral -mouse-160g/kg; total dose - 4.0g

Use Dose Rating

0.05

IARC Summary

Animal data

Chrysoidine is carcinogenic in mice following its oral administration, producing liver-cell tumors, leukemia and reticulum-cell sarcomas. Tests in rats were too briefly reported to be evaluated.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 8:91

4,4'-DIAMINODIPHENYLMETHANE

Usage

Over 91 million kg are produced in the U.S. annually. About 99% is consumed on site by reaction with phosgene to form isocyanates, the polyisocyanates which are used in the manufacture of polyurethane foams. Approximately 2500 workers in the U.S. are thought to be exposed to this substance.

VOID
Disregard this Page

Dosage Levels

Doses not available

Special Comments

At this time there is no known association with human cancer.

IARC Summary

This substance has not been reviewed by IARC.

Reference:

"Background Information on 4,4' Diaminodiphenylmethane"

ortho and para-DICHLOROBENZENE

Usage

U.S. production of ortho-dichlorobenzene was 2.8 million kg in 1972. Major uses are as a solvent, rust proofing, heat exchanger, coolant, dye intermediate and insecticide.

Production of para-dichlorobenzene in 1972 was 35 million kg. It is mainly used as a space odorant and in moth control.

Dosage Levels

There have been no adequate animal studies to date.

Special Comments

These compounds are not proven carcinogens.

IARC Summary

Animal data

No adequate studies on which to base an evaluation of carcinogenicity were available to the Working Group.

Human data

One report has suggested an association between leukemia and exposure to dichlorobenzenes, but this is insufficient evidence from which to assess the carcinogenic risk of this compound.

Reference:

IARC 7:231

3,3'-DICHLOROBENZIDINE

Usage

U.S. production of this substance in 1971 was 1.6 million kg while imports were 0.658 million kg. It is mainly used as a chemical intermediate in dye production and has some usage as a curing agent for isocyanate containing polymers.

Dosage Levels

oral-rat-22.5 g/kg/52 weeks; total dose - 4.5 g.

Use Dose Rating

0.35

Special Comments

This substance is regualted by OSHA as a carcinogen.

IARC Summary

Animal data

3,3'-Dichlorobenzidine is carcinogenic in the rat following oral and subcutaneous administration and in the hamster after oral administration.

Human data

No epidemiological data are available, but as 3,3'-dichlorobenzidine and benzidine may be made in the same plant, the possibility cannot be excluded that dichlorobenzidine has contributed to the incidence of bladder cancer attributed to benzidine.

References:

IARC 4:49
Hazard Review of 3,3'-Dichlorobenzidine RSV 0013581

3,3'-DIMETHOXYBENZIDINE

Usage

In 1967, U.S. production was 167 thousand kg. Imports in 1971 amounted to 124 thousand kg. It is used principally as a chemical intermediate in the production of dyes and also in the production of ortho-dianisidine diisocyanate.

Dosage Levels

oral - rat - 1.3g/kg/52 weeks; total dose 0.26g

Use Dose Rating

Based on usage of 200,000 kg, 0.77.

IARC Summary

Animal data

3,3'-Dimethoxybenzidine (o-Dianisidine) was shown to have a carcinogenic effect in rats following oral administration. The findings obtained in the hamster by the same route suggest a similar effect.

Human data

No conclusive epidemiological studies have been reported concerning the carcinogenicity of o-dianisidine alone in man.

Reference:

IARC 4:41

RSV 0013582

3,3'-DIMETHYLBENZIDINE (ortho-TOLIDINE)

Usage

U.S. production in 1962 was 110 thousand kg with imports of 70 thousand kg. More recent data is unavailable. More than 95 dyes are derived from this substance.

Dosage Levels

subcutaneous injection - rat - 60 mg/week for 91 weeks; total dose - 5.5g

IARC Summary

Animal data

Purified o-tolidine is a systemic carcinogen in the rat when given subcutaneously. The oral experiment in the rat is of doubtful significance because of the small number of animals involved. In feeding experiments, the commercial product failed to produce tumors in hamsters.

Human data

No epidemiological studies are available.

Reference:

IARC:1:87

1,1-DIMETHYLHYDRAZINE

Usage

Current use of 1,1-dimethylhydrazine in the U.S. is estimated to be less than 500 thousand kg with the major use as a rocket fuel. The substance has some usage as a plant growth regulator and as a chemical intermediate.

Dosage Levels

oral - mouse 4g/kg/60 weeks; total dose - 0.1g

Use Dose Rating

5

Special Comments

It is believed that when this substance undergoes combustion as a rocket fuel, some N-nitrosodimethylamine may be produced.

IARC Summary

Animal data

1,1-Dimethylhydrazine (UDMH) is carcinogenic in mice after oral administration. The observation of a few liver tumors after high oral doses of UDMH occurring in rats after a long latent period does not allow a proper evaluation of the carcinogenic effect in this species.

Human data

No epidemiological data are available to the Working Group.

Reference:

IARC 4:137

RSV 0013584

ETHYLENE DIBROMIDE

Usage

Annual usage in the U.S. is approximately 137 million kg. It is used as an antiknock gasoline additive and as a fumigant. Over 9,000 workers are estimated to be exposed to the substance.

Dosage Levels

oral - mice - 8.6g/kg; total dose - 0.215g

Use Dose Rating

637

Special Comments

To date there is no known association between ethylene dibromide and human cancer.

IARC Summary

This substance has not been reviewed by IARC.

Reference:

"Background Information on Ethylene Dibromide".

ETHYLENEIMINE (AZIRIDINE)

Usage

Currently there is one U.S. producer with an annual estimated production of less than 2.2 million kg. Approximately 50% is polymerized to polyethyleneimine which is used as a flocculent in water treatment and as an intermediate for oil additive compounds, ion exchange resins, adhesives, surfactants and stabilizers.

Dosage Levels

oral - mouse - 4.16g/kg/78 weeks; total dose - 0.104g.

Use Dose Rating

21(on the basis of 2.2 million kg.)

IARC Summary

Animal data

Aziridine is carcinogenic in two strains of mice following its oral administration, producing an increased incidence of liver-cell and pulmonary tumors. Subcutaneous injection of single doses in suckling mice produced an increased incidence of lung tumors in males. In one experiment in rats it increased the incidence of tumors at the injection site following its subcutaneous injection in oil.

Human data

No case reports or epidemiological studies were available to the Working Group.

References:

IARC 9:37
"Hazard Review of Ethyleneimine (Aziridine)"

HYDRAZINE

Usage

In 1966, 10 million kg. were produced in the U.S. Since then production has fallen; in 1971 estimated production was 2.3 million kg. Previous usage for hydrazine has been as a rocket fuel. In 1971 it was estimated that 40% production was as an oxygen scavenger in boiler water treatment and 60% as a chemical intermediate.

Dosage Levels

oral-new born mice- 17mg total dose

IARC Summary

Animal data

Hydrazine or hydrazine salts have been shown to be carcinogenic in mice after oral and intraperitoneal administration, and in rats following oral administration. No tumors were observed in Syrian golden hamsters after oral administration.

Human data

No epidemiological data are available to the Working Group.

Reference:

IARC 4:125

LEAD AND COMPOUNDS

Usage

In 1968 approximately 1.2 billion kg. of lead were consumed in the U.S. The main uses of lead are in manufacture of tetraethyllead as an additive to gasoline, in paints, cables, ammunition and brass, in printing and in can manufacturing.

Dosage Levels

lead acetate - oral - rat - 82g/kg; total dose 16.4g; lead phosphate - subcutaneous - rat - 200 mg/kg; total dose - 40 mg.

Special Comments

The use of lead that is responsible for most human exposure is its addition to gasoline.

IARC Summary

Animal data

Lead acetate is carcinogenic in rats and mice; lead subacetate and lead phosphate are carcinogenic in the rat. Given orally, they produce benign and malignant tumors of the kidney. The observation that exposure of rats to lead subacetate may result in an increased frequency of gliomas needs confirmation, as well as the observation of a high frequency of tumors of the testis, adrenal, thyroid, pituitary and prostate, together with renal tumors, in rats receiving lead acetate. No induction of tumors was reported to occur following exposure to lead arsenate or lead carbonate, but the evidence cannot be held as conclusive.

The pattern of absorption metabolism and storage of lead in the body seems to be similar in all animal species that have been studied. The kidney is a target from the point of view of toxicity in all animal species studied. Renal enlargement and the appearance of intranuclear inclusion bodies in the epithelial cells occur in all laboratory animal species and in man in the same way.

Human data

There is no evidence to suggest that exposure to lead salts causes cancer of any site in man. However, only one epidemiological study of the relationships between exposure to lead and the occurrence of cancer has been reported. It must be noted that the level of human exposure equivalent to the levels of lead acetate producing renal tumors in rats is 810 mg per day (550 mg Pb). This appears to exceed by far the maximum tolerated dose for man.

References:

IARC 1:41
Suspected Carcinogens

4,4'-METHYLENE BIS(2-CHLOROANILINE)

Usage

In 1972 U.S. production was 3.5 million kg. Virtually all is used as a curing agent for isocyanate containing polymers. Approximately 1% is used as a curing agent for epoxy and epoxy-urethane resins.

Dosage Levels

oral - mouse - 80 g/kg; total dose - 2g

Use Dose Rating

1.7

Special Comments

This substance is regulated by OSHA as a carcinogen. It is associated with liver and lung tumors in rats.

IARC Summary

Animal data

4,4'-Methylene bis(2-chloroaniline) is carcinogenic in the mouse and rat after oral administration and produces distant tumors in the rat after subcutaneous administration.

Human data

There are no conclusive epidemiological studies on which an evaluation of the carcinogenic risk can be based.

RSV 0013589

References:

IARC 4:65
Hazard Review, 4,4'-Methylene Bis(2-Chloroaniline)

4,4'-METHYLENEDIANILINE

Usage

Recent U.S. production was estimated by IARC to be approximately 1 million kg annually. Much more is present in an intermediate amine mixture which is used in the production of polymethylene polyphenylisocyanate (of which 52 million kg is produced). This compound is used in the manufacture of rigid polyurethane foam.

Dosage Levels

oral - rat - 6g/kg/72 weeks; total dose - 1.2g.

Use Dose Rating

Considering only the pure compound, 0.63

Special Comments

This chemical is not a definitely established carcinogen. However, its large use warrents its inclusion on this list.

IARC Summary

Animal data

The available experimental evidence in the rat, the only species tested, does not permit a definite conclusion regarding the carcinogenicity of 4,4'-methylenedianiline (DAPM) in this species.

Human data

No epidemiological data are available to the Working Group.

Reference:

IARC 4:79

alpha-NAPTHYLAMINE (1-Napthylamine)

Usage

In 1963 U.S. production of this substance was 500,000 kg. There is no recent production data available. In 1971 imports amounted to 27,000 kg. It is used as an intermediate in dyes, herbicides and antioxidants.

Dosage Levels

subcutaneous - newborn mice - total dose - 300 ug
oral - mouse 11.8g/kg; total dose - 0.29g. These studies were not judged conclusive by IARC.

Special Comments

This substance is regulated as a carcinogen by OSHA. It is strongly associated with bladder cancer in man when used with 4 to 10% beta-napthylamine.

IARC Summary

Animal data

No carcinogenic effect of 1-naphthylamine was observed in the hamster following oral administration. The results obtained after oral and subcutaneous administration to mice are inconclusive. The experiments in dogs demonstrate that 1-naphthylamine, if carcinogenic at all, is less so to the bladder in this species than is the 2-isomer.

Human data

Occupational exposure to commercial 1-naphthylamine containing 4-10% 2-naphthylamine is strongly associated with bladder cancer. A number of case reports from several countries support this association. It is not possible on present evidence to decide whether 1-naphthylamine free from the 2-isomer is carcinogenic to man.

References:

IARC 4:87
"Hazard Review of alpha-Naphthylamine" RSV 0013591

beta-NAPHTHYLAMINE (2-Naphthylamine)

Usage

IARC presents no current information on production of this substance. It is used as an intermediate in production of dyes and antioxidants.

Dosage Levels

stomach tube - mouse - 240 mg/kg/biweekly for 90 weeks caused liver tumors in 50% of the animals; total dose - 1.3g
parenteral - mouse - 18 mg/kg; total dose - 0.004g.

Special Comments

This substance is regulated as a carcinogen by OSHA. According to NIOSH, induction of bladder tumors in workers exposed to beta-naphthylamine is one of the most well established cause and effect relationships in occupational medicine.

IARC Summary

Animal data

2-Naphthylamine is carcinogenic in the mouse, hamster, dog and monkey. Given orally it has produced bladder carcinomas in the dog and monkey, and, at high dosage levels, in the hamster. By this route, it has increased the incidence of hepatomas in the mouse; in the rat and rabbit, it has little, if any, carcinogenic effect.

Human data

Epidemiological studies have shown that occupational exposure to 2-naphthylamine, either alone or when present as an impurity in other compounds, is strongly associated with the occurrence of bladder cancer. There is no doubt that 2-naphthylamine is a human bladder carcinogen.

References:

IARC 4:97
Suspected Carcinogens
Hazard Review of beta-Napthylamine

NICKEL AND COMPOUNDS

Usage

U.S. imports of nickel powder were 3.2 million kg. in 1970. A principal use is as an alloying additive in steel manufacture. Nickel acetate production in 1968 was estimated at 0.3 million kg. It is mainly used as a mordant in the dye industry. Total U.S. production of nickel carbonyl is estimated at less than 7.5 million kg. High purity nickel powder is made from it.

Dosage Levels

nickel powder - inhalation - rat - 15 mg/m^3
nickel carbonyl - intravenous - rat - 157 mg/kg/18 weeks; total dose - 0.031g;
nickel II acetate - intramuscular - rat - 350 mg/kg/43 weeks intermittently; total dose - 0.07g.

Use Dose Rating

nickel oxide - 1360; nickel carbonyl -220

IARC Summary

Animal data

Evidence of tumor induction in the lungs of mice, rats, hamsters or guinea-pigs following inhalation of powdered nickel alone or in combination with sulphur dioxide and powdered limestone is regarded as inconclusive. Two inhalation studies in rats on nickel carbonyl failed to produce conclusive evidence of pulmonary tumor development.

No information on long-term feeding studies was available to the Working Group.

Injection i.m. of nickel powder, nickel subsulphide, nickel oxide or nickelocene into mice or rats may result in the appearance of fibrosarcomas and/or rhabdomyosarcomas. The fact that a variety of nickel compounds produce local tumors suggests that nickel in some form is the active agent. This is also supported by the fact that sarcomas arose around millipore diffusion chambers containing nickel subsulphide. The results of the studies in which nickel powder or nickelocene was injected i.m. into hamsters are regarded as inconclusive.

The observation that repeated i.v. injections of nickel carbonyl induces tumors in rats was also regarded as inconclusive by the Working Group.

No conclusive evidence of tumor formation was found when nickel powder was introduced into the femoral or pleural cavity of rats.

RSV 0013593

Human data

In the past, there has been an excessive risk of cancers of the nasal sinus and lung among nickel refinery workers who inhale nickel containing dusts from crude ores. It is probable that nickel in some form is carcinogenic.

There is no evidence to suggest that non-occupational exposure to nickel constitutes a cancer hazard.

References:

IARC 2:126
Suspected Carcinogens

POLYCHLORINATED BIPHENYLS (PCB's)

Usage

U.S. usage in 1970 was 85 million kg. Production dropped to about 12 million kg by 1972 and has decreased even more since then. PCB's saw a wide variety of uses before 1971. Since then they have been restricted to closed electrical systems.

Dosage Levels

oral - mouse - 18g/kg/44 weeks; total dose 0.46g.

Use Dose Rating

Assuming a usage of 10 million kg., 21.

Special Comments

PCB's have been found in soil, sediment, water and particulate matter. It is a particularly important contaminant in fish, where it sometimes exceeds the FDA limit of 5 ppm. Some fishing has been banned because of this in the Hudson and Great Lakes.

IARC Summary

Animal data

A limited number of PCB's have been tested. Kanechlor 500 and Aroclor 1254 are carcinogenic in mice, inducing benign and malignant liver-cell tumors following oral administration, the only route tested. In rats, Kanechlor 500, 400 and 300 induced multiple hyperplastic liver nodules following oral administration.

Human data

In the absence of epidemiological studies the available case report does not allow an evaluation to be made.

Reference:

IARC 7:261

RSV 0013595

SUDAN I (Solvent Yellow 14) 1-(Phenylazo) -2- naphthalenol

Usage

1972 U.S. production was 270,000 kg. It is used for coloring hydrocarbon solvents, oils, fats, waxes, shoe and floor polish, cellulose ether varnishes, styrene resins, gasoline, soap, and colored smoke.

Dosage Levels

Subcutaneous-mouse, 5.6g/kg; total dose 0.14g

Use Dose Rating

2

IARC Summary

Animal data

Sudan I is carcinogenic in mice following its subcutaneous administration, producing tumors of the liver. It also produced bladder tumors in mice following its implantation into the urinary bladder. Tests by oral administration in mice and rats were negative, but the adequacy of the dose level used could not be assessed.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 8:225

THIOUREA

Usage

In 1964, U.S. use was estimated at 3.6 million kg. Imports in 1973 amounted to 1.7 million kg. It is used as a fire retardant resin for fabrics, for boiler water treatment and in photographic processes.

Dosage Levels

oral - rat - 5.5g/kg/104 weeks; total dose 1.1g.

IARC Summary

Animal data

Thiourea produced liver, thyroid and Zymbal gland tumors in rats following oral administration. Intraperitoneal injection followed by oral administration also led to the formation of Zymbal gland tumors in rats.

Oral and s.c. administration to mice did not produce thyroid tumors; however, the experiment using the s.c. route was inadequate. An increased incidence of liver-cell tumors in trout was reported, but this result cannot be considered as conclusive until additional studies using properly controlled diets are reported.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 7:95

TRICHLOROETHYLENE

Usage

Trichloroethylene is used mainly in degreasing and drycleaning. It is also used in inks, paints, lacquers, varnishes, and adhesives.

It is estimated that 280,000 people are occupationally exposed. Annual production is estimated at 243 million kg.

Dosage Levels

stomach tube - male mice - 1200 mg/kg five times weekly; stomach tub - female mice - 900 mg/kg five times weekly.

Use Dose Rating

270

Special Comments

Preliminary evaluation of this substance by the National Cancer Institute indicates that this material is a potent liver carcinogen.

IARC Summary

This substance has not been reviewed by IARC.

Reference:

"Background Information on Trichloroethylene"

VINYL CHLORIDE MONOMER

Usage

In 1973 U.S. production was 2,428 million kg. Ninety-seven percent of vinyl monomer produced is polymerized. The remainder has been used as a refrigerant and aerosol propellant.

Dosage Levels

inhalation - mouse - 250ppm

Special Comments

In 1974 the EPA estimated that 90 million kg of vinyl chloride were discharged into the atmosphere.

IARC Summary

Animal data

Vinyl chloride monomer (VCM) is carcinogenic in mice and rats following exposure by inhalation. The tumors in mice were mainly lung tumors, mammary carcinomas and angiosarcomas (malignant haemangioendotheliomas) of the liver. Angiosarcomas of the liver and other organs, Zymbal gland carcinomas and nephroblastomas occurred in exposed rats. Preliminary studies have suggested that VCM also produces subcutaneous angiosarcomas in the offspring of rats that have been exposed during pregnancy.

Human data

In view of the extreme rarity of angiosarcoma of the liver in the general population, the observation of 16 cases in workers exposed to vinyl chloride monomer during the polymerization process is evidence of a causal relationship.

Reference:

IARC 7:291

GROUP II

Low Usage or No Information on Usage

ACETAMIDE
*2-ACETYLAMINOFLUORENE
ortho-AMINOAZOTOLUENE
*4-AMINOBIPHENYL
para-AMINOAZOBENZENE
2-(1-AZIRIDINYL)ETHANOL
AZOBENZENE
BIS(2-CHLOROETHYL)ETHER
*BIS(CHLOROMETHYL)ETHER
*CHLOROMETHYL METHYL ETHER
DIETHYLSTILBESTROL
DIETHYL SULPHATE
*para-DIMETHYLAMINOAZOBENZENE
DIMETHYLCARBAMOYL CHLORIDE
DIMETHYL SULPHATE
DIOXANE
ETHYLENE THIOUREA
GLYCIDALDEHYDE
HEXAMETHYLPHOSPHORIC TRIAMIDE
ISONICOTINIC ACID HYDRAZIDE
MAGENTA
2-METHYLAZIRIDINE
*4-NITROBIPHENYL
N-NITROSODIETHYLAMINE
*N-NITROSODIMETHYLAMINE
para-NITROSODIMETHYLANILINE (ACCELERENE)
NITROSOMETHYLUREA
OIL ORANGE SS
PONCEAU MX
PONCEAU 3R
1,3-PROPANE SULTONE
*beta-PROPIOLACTONE
[redacted]
SUDAN II
2,4-TOLUENEDIAMINE
TRYPAN BLUE
URETHANE (ETHYL CARBAMATE)

*Regulated by OSHA as a carcinogen

RSV 0013600

PESTICIDES

ARAMITE
BHC and LINDANE
CHLOROBENZILATE
DDT and associated substances
DIELDRIN
KEPONE (Chlordecone)
MIREX
QUINTOZENE (Pentachloronitrobenzene)
TERPENE POLYCHLORINATES (Strobane)

ACETAMIDE

Usage

Acetamide is used as a soldering flux ingredient, solvent, wetting agent and penetration accelerator for dyes, a component for urea molding compounds, a plasticizer in leathers and a chemical intermediate. No production data available.

Dosage Levels

oral-rat-360 mg/kg/152 weeks; total dose 0.072g.

IARC Summary

Animal data

Acetamide is carcinogenic in rats following oral administration (the only species and route tested) producing benign and malignant liver tumors.

Human data

No case reports or epidemiological studies were available to the Working Group.

References:

IARC 7:197
Suspected Carcinogens

2-ACETYLAMINOFLUORENE

Usage

This substance was initially used as a pesticide until it was discovered to be a potent carcinogen. No production data are available.

Dosage Levels

skin-rat-250 mg/kg17 weeks; total dose 0.052g.
oral-mouse-560 mg/kg14 days; total dose 0.014g.

Special Comments

2-Acetylaminofluorene is regulated by OSHA as a carcinogen. It has been shown to be carcinogenic in rats, mice, rabbits, dogs, hamsters and fowl.

IARC Summary

Not reviewed in IARC.

References:

Hazard Review of 2-Acetylaminofluorene
Suspected Carcinogens

ortho-AMINOAZOTOLUENE

Usage

This material is used as a color for fats and waxes. There are no production data available.

Dosage Levels

oral-mouse-700 mg/kg; total dose 0.0175 g.

Special Comments

ortho-Aminoazotoluene is a single dose carcinogen.

IARC Summary

Animal data

ortho-Aminozaotoluene is carcinogenic in mice, rats, hamsters and dogs following oral administration, producing mainly tumours of the liver, gall-bladder, lung and urinary bladder. It also produced a carcinogenic effect following its administration by other routes in mice and rats. There is some evidence that it produces papillomas of the bladder in rabbits following its administration by direct bladder instillation and in mice after bladder implantation. It is effective in single doses in newborn mice.

Human data

No case reports or epidemiological studies were available to the Working Group.

References:

IARC 8:61
Suspected Carcinogens

4-AMINOBIPHENYL

Usage

At one time the major use of this product was as a rubber antioxidant. It is not known if it is presently being produced.

Dosage Levels

oral-mouse-140 mg/kg; total dose 0.003 g.

Special Comments

This substance is regulated by OSHA as a carcinogen. It is clearly implicated in the induction of bladder tumors in humans.

IARC Summary

Animal data

4-aminobiphenyl is carcinogenic in the mouse, rat, rabbit and dog. Following its oral administration it has produced bladder and liver tumors in mice and bladder papillomas and carcinomas in rabbits and dogs.

Human data

In the epidemiological studies, confined to one series of workers occupationally exposed to commercial 4-aminobiphenyl, a high incidence of bladder carcinomas was reported. Thus bladder cancer appears strongly associated with occupational exposure to 4-aminobiphenyl.

References:

IARC 1:74
Hazard Review of 4-Aminobiphenyl

para-AMINOAZOBENZENE

Usage

Used as dye for lacquers, varnishes, wax products, oil stains and styrene resins, and as an intermediate in manufacture of other dyes.

Dosage Levels

oral-rat-2 g/kg of diet caused liver tumours.

IARC Summary

Animal data

para-Aminoazobenzene is carcinogenic in rats following its oral administration, producing liver tumours, and by application to the skin, producing epidermal tumours. Experiments involving its subcutaneous injection in mice and rats or its intraperitoneal injection in rats could not be evaluated because of the limited numbers of animals used or the inadequate duration of the studies.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 8:53

RSV 0013606

2-(1-AZIRIDINYL)ETHANOL

Usage

Production by one company in 1973 was about 45,000 kg. It is used in modification of latex polymers for coatings, textile resins and starches.

Dosage Levels

Weekly injections subcutaneously, of 0.3 mg in 0.05 ml tricaprylin for 75 weeks caused injection site tumors. Total dose 0.023 g.

Use Dose Rating

2 (Based on 45,000 kg.)

IARC Summary

Animal data

2-(1-Aziridinyl)ethanol is carcinogenic in mice, producing malignant tumors at the site of its subcutaneous injection in the only available study.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 9:47

AZOBENZENE

Usage

Azobenzene is commercially sold only in relatively small quantities for research purposes. However, it is produced and consumed as a dye intermediate in the production of benzidene.

Dosage Levels

oral-mouse-at 7 days of age 21.5 mg/kg. Same absolute amount then given daily until 28 days of age. Mice then transferred to a diet of 56 mg/kg of diet for up to 80 weeks. An excess of hepatomas were found in male mice only. The total dose was approximately 0.16 mg.

IARC Summary

Animal data

Azobenzene was tested by the oral route in mice and by the subcutaneous route in mice and rats. In the oral study in mice it produced an excess of liver-cell tumors over the controls in males (but not in females) in one of the two strains used. Subcutaneous studies in mice and rats were negative, but they cannot be evaluated because the adequacy of the dose could not be assessed.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 8:75

BIS(2-CHLOROETHYL)ETHER

Usage

One United States company sold 1.8 million kg. in 1973 but it is no longer produced for sale in the United States in commercial quantities. Two United States companies produce it for in-house use. One of these produces less than 0.5 million kg annually for use as a solvent and a chemical intermediate. It has been used as a soil fumigant and insecticide, paint remover and in dry cleaning.

Dosage Levels

oral-mouse-33 g/kg; total dose 0.83g.

IARC Summary

Animal data

Bis(2-chloroethyl) ether produced an increased incidence of liver-cell tumors in male mice of two strains following its oral administration. Its administration by the subcutaneous route in mice produced a low incidence of sarcomas at the injection site.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 9:117

BIS(CHLOROMETHYL)ETHER

Usage

There is no commercial production of this material for sale to others in the United States. It is produced by saturating a solution of paraformaldehyde in cold sulfuric acid with hydrogen chloride. It is used as a chemical intermediate in the production of various textile aids.

Dosage Levels

inhalation-mouse-5 mg/m^3/6 hours per day for 82 exposure days
inhalation-rat-100 ppb/6 hours per day for 20 weeks
subcutaneous-newborn mouse-12.5 ul/kg

Special Comments

This substance is regulated by OSHA as a carcinogen.

IARC Summary

Animal data

Bis(chloromethyl) ether (BCME) is carcinogenic to mice following inhalation, skin application and subcutaneous administration. In newborn mice it is carcinogenic after a single subcutaneous exposure. In the rat it is carcinogenic by inhalation and subcutaneous administration.

Human data

A high incidence of predominantly oat-cell carcinoma in a small population of laboratory workers exposed to BCME strongly suggests that exposure to this compound constitutes a serious human lung cancer hazard.

There is also epidemiological evidence to suggest that exposure to BCME may constitute a lung cancer risk amoungst workers exposed to it as a contaminant in the manufacture of the related chloromethyl methyl ethers.

References:

IARC 4:231
Suspected Carcinogens
Hazard Review of Bis(chloromethyl)ether

RSV 0013610

CHLOROMETHYL METHYL ETHER

Usage

In 1973 there was one United States producer. Production figures are not available. The material is used in the preparation of ion-exchange resins and as a chloromethylating agent in organic synthesis.

Dosage Levels

subcutaneous-mouse-313 mg/kg26 weeks; total dose 0.008g.

Special Comments

This substance is regulated by OSHA as a carcinogen. It was strongly implicated as a human carcinogen in one study in which bis(chloromethyl) ether may have also been present.

IARC Summary

Animal data

Chloromethyl methyl ether (CMME) is almost invariably contaminated by bis(chloromethyl)ether (BCME), and the latter may be responsible for at least part of the observed carcinogenic activity. Such contaminated CMME has been found to be carcinogenic on subcutaneous injection in the mouse and possibly to be an initiator for mouse skin tumors. Inhalation in mice and subcutaneous injection in the rat produced equivocal evidence of carcinogenic activity.

Human data

One study based on 4 cases of oat cell lung cancer observed among 111 workers exposed to CMME (and its associated BCME impurity), followed for 5 years, suggests an increased risk of lung cancer.

References:

IARC 4:239
Hazard Review of Chloromethyl Methyl Ether

RSV 0013611

DIETHYLSTIBESTROL (DES)

Usage

United States production data are not available. Total sales of DES and its diphosphate ester derivative for use in human medicine are estimated to be less than 500 kg annually. In 1972, a total of 3,053 kg was imported.

It is widely used in the U.S. as a feed additive and as an ear implant to fatten, promote growth and to increase feed efficiency of beef cattle and sheep.

Dosage Levels

stomach tumbe-mouse-170 mg/kg; total dose 4.25 mg.

IARC Summary

Animal data

Diethylstilbestrol (DES) was tested in mice by oral administration, local application and subcutaneous injection, in mice, rats, hamsters and squirrel monkeys by subcutaneous implantation and in hamsters by subcutaneous injection. Its administration to mice resulted in an increased incidence of mammary and lymphoid tumors in both males and females, and of interstitial-cell tumors of the testis in males and cervical and vaginal tumors in females, including those exposed only on the first day of life. In rats, increased incidences of pituitary, mammary and bladder tumors were observed. In hamsters, a high incidence of renal tumors was observed in castrated males and females and in intact males, but not in intact females. In squirrel monkeys, malignant mesotheliomas of the uterine serosa were observed.

DES treatment in most cases increased the incidence of mammary tumors in strains of mice having a spontaneous incidence of these tumors, which may be related to the presence of a virus; testicular tumors occurred in strains having a particular genetic susceptability to such tumors. No evidence of a possible role of a virus has been shown in rats. Bladder tumors occurred only in rats in which bladder calculi were present.

In most cases, an accurate assessment of the effective carcinogenic dose in implantation studies is not possible. However, in oral administration studies, the lowest statistically significant dose (p. 0.01) producing mammary carcinomas in mice was about 0.15 ug/day (6 ug/kg/day). This dose is similar to that used in humans in the control of menopausal symptoms by DES (10 ug/kg/day) and 30 times less than the dose given for the control of mammary or prostatic cancer (300 ug/kg/day).

Human data

The administration of diethylstilbestrol to women during pregnancy is associated with an increased risk or vaginal or cervical adenocarcinoma in their exposed female offspring. There may also be an increased risk of endometrial carcinoma in women with gonadal dysgenesis treated with this drug. It is possible that the administration of the drug therapeutically to men with carcinoma of the prostate increases the risk of breast cancer.

Reference:

IARC 6:55

DIETHYL SULFATE

Usage

This substance is used as an alkylating agent. There are no production data available.

Dosage Level

subcutaneous-rat-1.25 g/kg; total dose 0.25 g.

IARC Summary

Animal data

Diethyl sulphate is carcinogenic in the rat, the only species tested, following subcutaneous administration and pre-natal exposure. There is inconclusive evidence suggesting carcinogenicity in the rat following oral administration.

Human data

No epidemiological data are available to the Working Group.

Reference:

IARC 4:277

RSV 0013614

Usage

Separate production data is not available as this compound is included in a category with at least 20 other colors with total production of 265,000 kg in 1974. It is used for coloring polishes and other wax products.

Dosage Levels

oral-rat-800 mg/kg/40 days; total dose 0.16g.
skin-rat-155 mg/kg/4 weeks intermittently; total dose 0.031g.

Special Comments

This substance is regulated by OSHA as a carcinogen. It is associated with liver cancer in rats.

IARC Summary

Animal data

para-Dimethylaminoazobenzene (DAB) is carcinogenic in rats, producing liver tumors after its administration by several routes, and in dogs producing bladder tumors following its administration by the oral route. Results of oral administration studies were doubtful in mice and negative in hamsters and guinea-pigs; but these studies were of short duration, and the adequacy of the dose levels used was not known.

DAB has also been tested by subcutaneous injection in mice, and the results are suggestive of local and hepatic carcinogenicity. Treatment of newborn animals produced systemic carcinogenic effects in mice; the negative results obtained in rats are doubtful, since the period of observation was too short. Skin-painting with DAB produced epidermal tumors in rats but not in mice.

An extensive dose-response study was carried out in rats: the lowest effective dose was 1 mg/rat/day and the highest noneffective dose, 0.3 mg/rat/day.

Human data

No case reports or epidemiological studies were available to the Working Group.

References:

IARC 8:125
Suspected Carcinogens
Hazard Review of para-Dimethylaminoazobenzene

DIMETHYLCARBAMOYL CHLORIDE

Usage

This compound is used in the synthesis of drugs and possibly pesticides. Its last known commercial production was 1350 kg for drug use. It is estimated that fewer than 200 people are occupationally exposed.

Dosage Levels

inhalation-rat-1ppm-nasal tumors

Special Comments

Dimethylcarbamoyl chloride is an extremely potent carcinogen. There is no human data available at this time. The related compound diethylcarbamoyl chloride,has been found to be mutagenic.

IARC Summary

DMCC has not been reviewed by IARC.

Reference:

Current Intelligence Bulletin-Dimethylcarbamoyl chloride

DIMETHYL SULFATE

Usage

Dimethyl sulfate (DMS) is used as an alkylating agent. There are no accurate production data available.

Dosage Levels

single dose injection-rat-50 mg/kg; total dose 10 mg.

IARC Summary

Animal data

DMS has been shown to be carcinogenic in the rat, the only species tested, by inhalation, subcutaneous injection and following pre-natal exposure. It is carcinogenic to the rat in a single-dose exposure.

Human data

Certain case reports raise some suspicion as to the possible carcinogenicity of DMS in man, but good epidemiological evidence is not available to confirm this.

Reference:

IARC 4:271

DIOXANE

Usage

Dioxane is used as a solvent for lacquers, plastics, varnishes, paints, dyes, greases, fats, waxes and resins.

Dosage Levels

oral-rat-416 g/kg/57 weeks; total dose 93 g.

Special Comments

Dioxane induced liver and nasal tumours in rats when given in the drinking water over a period of 12 to 16 months.

IARC Summary

This substance is not reviewed in IARC.

References:

Dangerous Properties of Industrial Materials
Suspected Carcinogens

ETHYLENETHIOUREA

Usage

No production data available. Used as an accelerator for neoprene production.

Dosage Levels

oral-rat-7560 mg/kg; total dose 1.5g

IARC Summary

Animal data

Ethylenethiourea (ETU), tested only by the oral route in rats, produced thyroid carcinomas. The reported increased incidence of liver-cell tumors in 2 strains of mice following oral administration awaits confirmation.

Human data

No case reports or epidemiological studies were available to the Working Group.

References:

IARC 7:45
Suspected Carcinogens

RSV 0013619

GLYCIDALDEHYDE

Usage

Glycidaldeyde is used as an intermediate in manufacture of glycerol from acrolein.

Dosage Levels

skin-mouse-17 g/kg/48 weeks intermittently; total dose 0.42g.
subcutaneous-mouse-280 mg/kg/69 weeks intermittently; total dose 0.007g.

Special Comments

When painted on skins of mice, this substance led to local benign and malignant tumors in over 50% of the animals.

IARC Summary

Not reviewed in IARC.

References:

Suspected Carcinogens
Dangerous Properties of Industrial Materials

RSV 0013620

HEXAMETHYLPHOSPHORIC TRIAMIDE

Usage

Hexamethylphosphoric triamide is widely used as a laboratory chemical. No usage figures are available. Five thousand people are thought to be occupationally exposed, 90% of which are in research laboratories.

Dosage Levels

inhalation-rats-0.4 ppm - causes nasal tumors

Special Comments

This is an extremely potent carcinogen in rats. There are no human data available at this time.

IARC Summary

This substance has not been reviewed by IARC.

Reference:

Background Information on Hexamethylphosphoric Triamide

ISONICOTINIC ACID HYDRAZIDE

Usage

There is one United States producer whose production capacity is unknown. United States consumption in 1971 was estimated at 10,000 kg. According to IARC, the apparent United States supply in 1971 was 53,000 kg. This discrepancy cannot be accounted for at this time. It is used as an antitubercular agent.

Dosage Levels

Diets containing 0.25% caused 100% pulmonary tumors in mice. This corresponds to an approximate dose of 150 g/kg or a total dose of 3.75g.

IARC Summary

Animal data

Isonicotinic acid hydrazide (INH) is carcinogenic in mice after oral, subcutaneous and intraperitoneal administration. This observation of tumors in rats in only one of several oral studies is inconclusive. INH failed to produce tumors in hamsters when given orally.

Human data

Available evidence from the first 15 years of human exposure has not suggested that INH is carcinogenic in man in the doses applicable to treatment and prophylaxis of tuberculosis.

Reference:

IARC 4:159

RSV 0013622

MAGENTA Fuchsine

Usage

Magenta is used mainly as a dye or dye intermediate, and as a laboratory reagent in thin layer chromatography.

Dosage Level

subcutaneous - rat - 1850 mg/kg; total dose 370 mg.

IARC Summary

Animal data

The only evidence for the carcinogenicity of magenta is the induction of local sacomas in rats following subcutaneous administration of para-magenta, one of the components of commercial magenta. This positive result may have been due to the physical rather than to the chemical properties of this substance. Oral administration of commercial magenta to mice produced negative effects in a single study, but this finding was based upon an insufficient number of surviving animals.

Human data

One epidemiological study appears to establish a carcinogenic risk to workers involved in the manufacture of magenta. On present evidence, it is not possible to indicate whether the industrial bladder cancer found in magenta workers is attributable to exposure to magenta itself, or to one or more of its associated intermediates and impurities.

Reference:

IARC 4:57

2-METHYLAZIRIDINE

Usage

This product is manufactured in commercial quantities by one U.S. manufacturer. It is used exclusively as an intermediate, mainly to modify surface coating resins. Polymers modified with it have been used in the adhesive, textile and paper industries and as viscosity control agents in the oil industry.

Dosage Levels

oral-rat-1.12g/kg; total dose 0.22g.

IARC Summary

Animal data

2-Methylaziridine is carcinogenic in rats following its oral administration, the only species and route tested, producing a variety of malignant tumors.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 9:61

4-NITROBIPHENYL

Usage

There is no evidence that this compound is produced commercially in the U.S. at the present time. The only known commercial use is as a chemical intermediate in the preparation of 4-aminobiphenyl which was formerly used in large quantities, primarily as a rubber antioxidant.

Dosage Levels

oral-dog-5500 mg/kg caused bladder tumors.

Special Comments

This substance is regulated by OSHA as a carcinogen. There is evidence that 4-nitrobiphenyl is metabolized to 4-aminobiphenyl which is also regulated as a carcinogen by OSHA.

IARC Summary

Animal data

4-Nitrobiphenyl induced carcinomas of the bladder when given orally to dogs, the only species and route known to have been tested.

Human data

There are no data on the carcinogenicity of 4-nitrobiphenyl in man. However, it has been used in the production of 4-aminobiphenyl, which is a recognized human bladder carcinogen (IARC, 1972).

References:

IARC 4:113
Hazard Review of Nitrobiphenyl

N-NITROSODIETHYLAMINE

Usage

This product is used as a solvent for fibers, a softener for copolymers, a lubricant additive and in condensers to increase the dielectric constant.

Dosage Levels

oral-rat-64 mg/kg; total dose 0.0015 g.

Special Comments

In many cases 100% of the animals tested were affected.

IARC Summary

Animal data

N-Nitrosodiethylamine (DEN) is carcinogenic in all ten animal species tested, including sub-human primates. The main target organs are the nasal cavity, trachea, lung, esophagus and liver. It induces tumors following different routes of administration, including ingestion, inhalation and skin painting. It is carcinogenic in single-dose experiments and following prenatal exposure.

Human data

The possibility of a formation of DEN from precursors, diethylamine and nitrosating agents, in vivo, must receive further attention. No long-term studies of human subjects exposed to DEN are known.

Reference:

IARC 1:107

N-NITROSODIMETHYLAMINE

Usage

This substance is used as an industrial solvent and in the synthesis of rocket fuel, antioxidants, lubricant additives and in condensers to increase the dielectric constant.

Dosage Levels

oral-mouse-89 mg/kg; total dose 0.002g; kidney and lung tumors.

Special Comments

This substance is regulated by OSHA as a carcinogen. It produces cancer in a variety of sites and by several methods of administration in the rat, mouse, hamster, guinea pig, rabbit, trout, guppy and newt.

IARC Summary

Animal data

N-Nitrosodimethylamine (DMN) is carcinogenic in all seven animal species tested. The main target organs are the liver and the kidney. It induces tumors following different routes of administration, including ingestion and inhalation. It is carcinogenic following prenatal exposure and in single-dose experiments. Similarities in metabolism in human and rat liver tissues have been reported.

Human data

No long-term follow-up studies of human subjects exposed to DMN are known.

References:

IARC 1:95
Hazard Review of N-Nitrosodimethylamine

para-NITROSODIMETHYLANILINE (Accelerene)

Usage

Used in rubber manufacturer and as an intermediate in dye synthesis.

Dosage Levels

Not available

Special Comments

Low but definite incidence of tumors of the esophagus and stomach when given to rats in the drinking water for one year.

IARC Summary

Not reviewed in IARC.

Reference:

Dangerous Properties of Industrial Materials

NITROSOMETHYLUREA

Usage

Nitrosomethylurea is widely used in the synthesis of diazomethane. It has been proposed for use in chemotherapy.

Dosage Levels

Oral - rat - 90 mg/kg; total dose - 0.018g

IARC Summary

Animal data

Nitrosomethylurea (NMU) is carcinogenic in all six animal species tested. It has a local as well as a systemic carcinogenic effect, producing tumors at different sites, including the nervous tissue. It induces tumors following different routes of administration, including ingestion. It is carcinogenic in single-dose experiments and following prenatal exposure.

Human data

The compound may be formed *in vivo* from methylurea and nitrate or other nitrosating agents. No data on direct human exposure are available.

References:

Suspected Carcinogens
IARC 1:125

OIL ORANGE SS 1-(2-methylphenyl)azo -2-naphthalenol

Usage

No production data are available. Used for coloring varnishes, oils and waxes.

Dosage Levels

subcutaneous-mouse-6g/kg; total dose 0.15g.

IARC Summary

Animal data

Oil orange SS is carcinogenic in mice following its oral and subcutaneous administration, producing intestinal and local tumors; it also produced carcinomas of the bladder in mice following its administration by bladder implantation. Tests by the oral and subcutaneous routes in rats were either inadequately reported or of too short duration to be evaluated.

Human data

No case reports or epidemiological studies were available to the Working Group.

References:

Suspected Carcinogens
IARC 8:165

PONCEAU MX 4-[(2,4-dimethylphenyl) azo]-3-hydroxy-2,7 naphthalenedisulphonic acid, disodium salt

Usage

In 1972, three U.S. producers manufactured 17,000 kg. It is used mostly as a textile and leather dye and also to color inks, paper, pigments and wood stains.

Dosage Levels

oral-mouse-35.4 g/kg; total dose 0.88g.

IARC Summary

Animal data

Ponceau MX is carcinogenic, producing liver-cell tumors in mice and rats and possibly intestinal tumors in mice, following its administration by the oral route. A dose-response effect was noted in the mouse and rat studies.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 8:189

PONCEAU 3R 3-hydroxy-4 [(2,4,5-trimethylphenyl)azo]-2,7-naphthalenedisulphonic acid, disodium salt

Usage

This substance has been used to dye wool and in countries outside of the U.S., as a general food colorant.

Dosage Levels

oral-rat-33g/kg/65 weeks; total dose ~ 6.6g

IARC Summary

Animal data

Ponceau 3R is carcinogenic in rats following its oral administration, producing liver-cell tumors. It also produced bladder tumors in mice following its implantation in the urinary bladder. The oral study in mice was considered inadequate for evaluation.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 8:199

1,3-PROPANE SULTONE

Usage

Annual production of this substance in the U.S. is less than 500 kg. It is used as a chemical intermediate in synthesizing alcohols, phenols and soluble starches used in the textile industry.

Dosage Levels

oral-rat~1.3g/kg; total dose ~ 0.26g

IARC Summary

Animal data

1,3-Propane sultone has a carcinogenic effect in the rat when given orally, intravenously or by pre-natal exposure, and a local carcinogenic effect in the mouse and the rat when given subcutaneously. It is carcinogenic in the rat after single-dose exposures.

Human data

No epidemiological data were available to the Working Group.

Reference:

IARC 4:253

beta-PROPIOLACTONE

Usage

The substance was mainly used as an intermediate in the production of acrylic acid and its esters. As of 1973, two U.S. companies had a combined capacity of approximately 40 million kg per year of acrylic acid and esters based on beta-propiolactone. It is not known if beta-propiolactone is presently being produced in commercial quantities.

Dosage Levels

oral-rat-3.5g/kg; total dose 0.7g.

Special Comments

This substance is regulated by OSHA as a carcinogen.

IARC Summary

Animal data

B-Propiolactone is carcinogenic in the mouse by skin application, subcutaneous or intraperitoneal injection, and in the rat by subcutaneous injection. Oral administration in the rat gave some indication of carcinogenic activity. The results obtained in the hamster and guinea pig are equivocal. It is carcinogenic to mice after a single-dose exposure.

Human data

No epidemiological data were available to the Working Group.

References:

IARC 4:259
Hazard Review of beta-Propiolactone

RSV 0013634

PROPYLENEIMINE

Usage

Propyleneimine is used to produce intermediates in the synthesis of various resins, textiles and paper finishes.

Dosage Levels

Not available

Special Comments

When administered orally to mice, this substance induced breast, brain, and ear duct tumors and leukemia in a period of 60 weeks.

IARC Summary

Not reviewed in IARC.

VOID
Disregard this Page

Reference:

Dangerous Properties of Industrial Materials

SUDAN II 1-[(2,4-Dimethylphenyl)azo]-2-naphthalenol

Usage

Production in 1971 was 34,500 kg. It is used for coloring oils, waxes, hydrocarbon solvents for polishes, candles and polystyrene resins.

Dosage Levels

bladder implantation-mouse-2mg/40 weeks

IARC Summary

Animal data

Sudan II was tested in mice and rats by the oral and subcutaneous routes. Results of these studies cannot be evaluated because of the inadequacy either of the number of animals used, the duration of the experiment or the degree of reporting.

Sudan II was also tested in mice by bladder implantation, resulting in a high incidence of bladder carcinomas.

Human data

No case reports or epidemiological studies were available to the Working Group.

Reference:

IARC 8:233

2,4-TOLUENEDIAMINE (2,4-diaminotoluene)

Usage

This substance is used as an ingredient of dyes and as an intermediate for 2,4-toluenediisocyanate which is an important constitutent for making polyurethane foams.

Dosage Levels

When fed to rats at levels up to 1% in the diet, a high incidence of liver tumors occurred.

IARC Summary

Not reviewed by IARC

Reference:

Dangerous Properties of Industrial Materials

RSV 0013637

TRYPAN BLUE 3,3'-[(3,3'-Dimethyl (1,1'-biphenyl)-4,4'-diyl] bis (azo)]bis(5-amino-4-hydroxy)-2,7-naphthalenedisulphonic acid, tetrasodium salt

Usage

Trypan blue is used for dyeing textiles, leather and paper, in biological investigations, and in treatment of sleeping sickness.

Dosage Levels

Subcutaneous injections of a 1% solution of 0.5 to 1.0ml given at weekly or monthly intervals caused tumors.

oral-rat-440 mg/kg/21 days intermittently; total dose 0.088g.

IARC Summary

Animal data

Trypan blue is carcinogenic in rats following its subcutaneous or intraperitoneal injection, producing reticulum-cell sarcomas, mainly of the liver, as well as fibrosarcomas at the site of injection. Liver spindle-cell sarcomas were also induced in rats by single subcutaneous injections. Experiments by the oral route in rats and by subcutaneous injection in mice could not be evaluated because of the small number of animals used or because the adequacy of the dose used could not be assessed.

Human data

No case reports or epidemiological studies were available to the Working Group.

References:

IARC 8:267
Suspected Carcinogens

RSV 0013638

URETHANE (Ethyl Carbamate)

Usage

Urethane is used as a chemical intermediate in the preparation and modification of amino resins and as a solubilizer and co-solvent for pesticides, fumigants and cosmetics.

Dosage Levels

oral-mouse-160 mg/kg; total dose - 0.004g

IARC Summary

Animal data

Urethane has been shown to be carcinogenic in mice, rats and hamsters following administration by the oral, inhalation, subcutaneous or intraperitoneal routes, producing, among others, lung tumors, lymphomas, hepatomas, melanomas and vascular tumors. It is an initiator for skin carcinogenesis in mice both when given orally and topically. It was also shown to enhance the leukemogenic effect of X-irradiation. It is carcinogenic in single dose experiments and following prenatal exposure. Neonatal and infant mice are more susceptible to cancer induction by urethane than are adult mice.

Human data

No case reports or epidemiological studies were available to the Working Group.

References:

IARC 7:111
Suspected Carcinogens

ARAMITE 2-(4-tertiary-butylphenoxy)-1-methylethyl 2-chloroelthyl sulphate

Usage

The sole producing company has not reported production since 1970. Its usage is restricted to post harvest applications or non-bearing trees.

Dosage Levels

oral-mouse-130g/kg; total dose 3.2g

IARC Summary

Animal data

Aramite is carcinogenic in the rat and dog following its oral administration. It produced liver tumors in the rat and carcinomas of the gall bladder and biliary ducts in the dog.

Aramite was tested in 4 strains of mice by the oral route and produced a significant increase of hepatomas in males of one strain.

No tumors were reported in limited studies involving skin application and subcutaneous injection (single-dose) in mice.

Human data

No epidemiological studies were available to the Working Group.

References:

IARC 5:39
Suspected Carcinogens

BHC and LINDANE

Usage

Total U.S. production was less than 3 million kg in 1971. BHC is the common name for mixed isomers of 1,2,3,4,5,6 - hexachlorocyclohexane. Lindane is the common name for the gamma isomer of BHC. Isomers differ in spatial positions of chlorine atoms on boat and chain forms.

Dosage Levels

alpha isomer-oral mouse 8350 mg/kg; total dose 0.21g
beta isomer-oral mouse 31g/kg; total dose 0.70g;
LINDANE (gamma isomer) - oral mouse 62g/kg; total dose 1.6g
BHC (mixed isomers) oral mouse 22g/kg; total dose - 0.55g.

Special Comments

Lindane is a specially restricted pesticide in New Jersey when used in concentrations above 25%.

IARC Summary

Animal data

Technical BHC, its pure α, β and γ isomers and the mixture of $\delta + \varepsilon$ isomers are carcinogenic in mice, producing liver-cell tumors following oral administration. This effect was recorded in both sexes after exposure to the β and γ isomers; the technical compound, the α isomer and the $\delta + \varepsilon$ mixture were tested in male mice only.

Available feeding studies in rats were considered inadequate either because survival rates were low, the information reported was insufficient or because the doses given were too low.

No tumors were reported in limited skin application and subcutaneous implantation studies in mice.

Human data

No epidemiological studies were available to the Working Group. Patients dying with cancer did not show higher concentrations of BHC in fat tissues and liver than did control patients.

No firm conclusions as to a causal relationship with aplastic anaemia and/or leukemia can be drawn from available case reports.

References:

IARC 5:47
Suspected Carcinogens

CHLOROBENZILATE

Usage

In 1971, chlorobenzilate was manufactured by a single manufacturer with production of 1 million kg.

Dosage Levels

oral-mouse-71 g/kg; total dose - 1.8g

IARC Summary

Animal data

Chlorobenzilate was tested in a preliminary study by the oral route in two strains of mice and produced an increased incidence of hepatomas in males of both strains.

Reports of feeding studies in rats were considered inadequate for evaluation.

Human data

No epidemiological studies were available to the Working Group.

References:

IARC 5:75
Suspected Carcinogens

DDT and Associated Substances

Usage

Production in 1971 was approximately 20 million kg.

Dosage Levels

oral-mouse-73 mg/kg; total dose - 0.002g

Special Comments

Since December 13, 1972 this use of this substance has been banned by EPA for all but minor uses.

IARC Summary

Animal data

The hepatocarcinogenicity of DDT by the oral route has been demonstrated in several strains of mice. Liver-cell tumors have been produced in both sexes, and in CF1 mice some were found to have metastasized. Increased tumor incidences have been reported in some other organs; however, this finding was not confirmed in two recent multigeneration studies using a wide range of doses.

Oral administration studies in rats have provided no convincing evidence of carcinogenicity of DDT to this species.

In a single experiment, hamsters tolerated higher dietary intakes of DDT than did mice and rats and did not develop tumors in excess over the controls.

The negative results obtained in feeding studies with dogs and monkeys cannot be regarded as conclusive, due to limitations in group size and duration of treatment.

Liver-cell tumor induction in the trout cannot be considered as conclusive until additional studies using properly controlled diets are reported.

Skin application and subcutaneous injection studies in mice were too limited in duration and/or group size to allow any conclusions to be made.

Two dose-response studies on liver-cell tumor response to DDT following oral administration in two strains of mice are available. In both CF1 and BALB/c mice, dietary intakes of 250 ppm (corresponding to about 37.5 mg/kg/day) were highly effective in both sexes. The lowest dose used, 2 ppm in the diet (corresponding to an intake of 0.3 mg/kg/day), induced a significant increase in the incidence of hepatomas only in male CF1 mice.

The two DDT metabolites, p,p'-DDE and TDE (DDD), were tested by oral administration to mice. The latter produced a significant increase in lung tumors, while DDE was found to produce a high incidence of liver-cell tumors.

Human data

The cross-sectional epidemiological studies on workers exposed to DDT and the observational studies on volunteers were too limited and/or too short to allow any conclusions to be made regarding carcinogenesis.

Although fat concentrations of DDT residues were higher in terminal cancer patients than in control patients, this finding is inconclusive as to a causal relationship. A similar study with a different design did not show such a result.

References:

IARC 5:83
Suspected Carcinogens

DIELDRIN

Usage

Dieldrin is manufactured by only one U.S. company. Less than 450,000 kg were produced in 1971. Imports were 57,000 kgs. in 1972.

Dosage Levels

oral-mouse-870 mg/kg; total dose 0.022g

Use Dose Rating

20 (Based on 450,000 kg)

Special Comments

Dieldrin is a specially restricted pesticide in New Jersey.

IARC Summary

Animal data

Dieldrin was tested by the oral route only in mice and rats. The hepatocarcinogenicity of dieldrin in the mouse has been demonstrated and confirmed in several experiments, and some of the liver-cell tumors were found to metastasize. A dose-response effect has been demonstrated in both sexes with an increased tumor incidence in females at the lowest dose tested, 0.1 ppm in the diet (corresponding to about 0.015 mg/kg/bw/day). In mice, there is no evidence of carcinogenicity in organs other than the liver.

The available data in rats have not provided evidence of carcinogenicity at levels of up to 50 ppm in the diet (corresponding to an intake of about 2.5 mg/kg/bw/day).

The experiments in dogs and monkeys were too limited in duration and/or group sizes to allow any conclusions to be made.

Human data

The epidemiological study carried out on occupationally exposed workers does not allow any conclusions to be made concerning the existence of an excess risk of developing cancer.

Although fat concentrations of dieldrin residues were higher in terminal cancer patients than in control patients, this finding is inconclusive as to a causal relationship.

Reference:

IARC 5:125

KEPONE (Chlordecone)

Usage

Kepone was produced until July 1975. At that time, the U.S. EPA prohibited further manufacture. More than 99% of the production was reported to be for export and only about 0.8% for domestic use, mainly as bait for ant traps.

Dosage Levels

oral-mouse-2.24 g/kg in 80 weeks; total dose - 0.056; 20 ppm in feed

oral-female rat-0.739 g/kg in 80 weeks; total dose - 0.146 g; 20 ppm in feed

oral-male rat-0.8 g/kg in 80 weeks; total dose - 0.198 g; 20 ppm in feed

Special Comments

Hepatocellular carcinomas were found in mice and rats at the above levels.

IARC Summary

Not reviewed by IARC.

Reference:

Report on Carcinogenesis Bioassay of Technical Grade Chlordecone (Kepone)

RSV 0013646

MIREX (Dodecachloropentacyclodecane)

Usage

Mirex is manufactured in only one company in the world. Less than 454,000 kg were produced in 1971.

Dosage Levels

oral-mouse-2222 mg/kg; total dose 0.055g

Use Dose Rating

8

IARC Summary

Animal data

Mirex was tested in a preliminary study by the oral route in two strains of mice (the only route and species for which published results were available). It produced an increased incidence of hepatomas in both sexes of both strains used.

Human data

No epidemiological studies were available to the Working Group.

References:

IARC 5:203
Suspected Carcinogens

RSV 0013647

QUINTOZENE (Pentachloronitrobenzene)

Usage

Quintozene has been produced by a single company since 1966. 1.4 million kg. were produced in 1971. The only known use is as a fungicide.

Dosage Levels

oral-mouse-135g/kg; hepatomas; total dose - 3.3g

Use Dose Rating

0.4

IARC Summary

Animal data

Quintozene (pentachloronitrobenzene) was tested in a preliminary study by the oral route in two strains of mice and produced an increased incidence of hepatomas in males of one strain. A feeding study in rats was considered inadequate. Application of quintozene followed by croton oil to mouse skin gave positive results which could not be interpreted due to a lack of adequate controls.

Human data

No epidemiological studies were available to the Working Group.

Reference:

IARC 5:211

TERPENE POLYCHLORINATES (Strobane)

Usage

No production data have been reported since 1969. It is used mainly as an insecticide but has some usage as a moth-proofing agent for woolen clothes.

Dosage Levels

oral-mouse-1.27g/kg; total dose 0.032g

IARC Summary

Animal data

Terpene polychlorinates (Strobane) were tested in a preliminary study by the oral route in two strains of mice and produced an increased incidence of hepatomas in males of one strain. The suggestion that the product also increased the incidence of malignant lymphomas in this species awaits confirmation.

No adequate data were available for other species or other routes of administration.

Human data

No epidemiological studies were available to the Working Group.

Reference:

IARC 5:219
Suspected Carcinogens

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RSV 0013651

Appendix 8.

P U B L I C M E E T I N G

of the

SENATE COMMISSION ON THE INCIDENCE OF CANCER IN NEW JERSEY

Held:
June 11, 1976
Senate Chamber
State House
Trenton, New Jersey

MEMBERS OF COMMISSION PRESENT:

Senator John M. Skevin (Chairman)
Senator Joseph L. McGahn
Senator Wayne Dumont, Jr.

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SENATOR JOHN M. SKEVIN (Chairman): I would like to welcome everyone to the public meeting of the Senate Commission on Incidence of Cancer in New Jersey.

My name is John Skevin. I am a State Senator from District 38, and Chairman of this Commission. To my right is Senator McGahn of Atlantic County, the Vice Chairman of this Commission. Senator Dumont, also a member of this Commission, is on his way and will be with us shortly.

As Chairman of the Commission, I would like to make an opening statement.

In convening these hearings, let me emphasize one vital point: This will not be a forum for those who wish to pay lip service in the battle against cancer nor will it provide a platform for those simply seeking to make headlines. Rather, it will be a springboard for those who wish to help launch a meaningful, effective counter-attack against cancer: an opportunity for all those who are knowledgeable in the field to help us formulate a truly effective legislative program in New Jersey.

As we know, our State leads the nation in deaths from cancer. A dubious distinction at best. Every year, in fact, more than 14,000 of our citizens die from this disease and we can no longer tolerate such a staggering loss. It is too great a loss in both human and economic terms for us to accept. The time has come for us to do more than bury our dead - the time has come for us to fight back and here in New Jersey, where this killer is most prevalent. We should accept it as our responsibility to lead the way in this crusade. What other state has a more persuasive reason to take the initiative?

To dramatize my personal commitment to action, I have already introduced one bill in the State Senate bearing directly on the subject before us. That one would make cancer a reportable disease in the State of New Jersey. The fact is that while a doctor who diagnoses a case of measles must report that back to

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the State Department of Health, no such requirement exists with regard to cancer. What this means is that while we have a record of deaths from cancer, we have no idea as to how many cases are being diagnosed and treated or what types of cancers are currently prevalent. Such a lack of statistical knowledge is an obvious hinderance to any cancer control program. In the course of these hearings I shall seek the opinion of various witnesses on the merits of this bill, and fortified by their comments and suggestions I will then move for its passage in the Senate and then in the Assembly.

And today, as a further indication of my own commitment to action, I am introducing another bill in the State Senate, one that I am sure will be controversial, but one that, if adopted, will show that when it comes to the fight against cancer in New Jersey, it is literally ready to put its money where its mouth is. This bill would increase the cigarette tax by one cent per package. It would raise approximately $8 million a year. The bill provides that the amount so raised will be used exclusively to finance scientific and medical research into the causes, diagnoses, and treatment of cancer. One-half of such amount will go to the State Department of Health to provide matching funds for Federal grants. Institutions are to receive the remainder of such funds for cancer research, and the institutions must be in New Jersey, and certified by the Commissioner of the Department of Health to receive such funds - such as the College of Medicine and Dentistry of New Jersey, and to Rutgers, the State University. The bill requires such institutions to annually report to the Legislature how such funds are used.

New Jersey has qualified for relatively little of the $600 million the National Cancer Institute made available for cancer research last year. In 1975, New Jersey received $7,598,000 or $1.04 per capita. New York State received $107,251,000

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or $5.92 per capita. Massachusettes received $42,361,000 or $7.27 per capita. Pennsylvania received $34,788,000 or $2.92 per capita. And Maryland received $75,211,000 or $18.35 per capita. Figures show that if New Jersey were to attract its proportionate share of the National Cancer grants and contracts in relation to neighboring states, it would have received in 1975 $37,458,000 which reflects a loss of about $30 million in one year.

To correct this situation, and thereby receive its fair share of cancer research from Federal and other sources, New Jersey must provide stable funds to attract cancer research scientists and provide facilities and services where productive cancer research can be conducted.

Cigarette smoking is one of the major causes of lung cancer, and there is a measure of propriety in financing New Jersey cancer research capability by imposing a small tax on cigarettes. And, again, as these hearings proceed, I will be seeking the opinion and advice of the witnesses on this bill, and if their support warrants it, I will naturally press for passage in the Legislature, incorporating whatever amendments or modifications their testimony might warrant.

But most importantly, I hope these hearings will result in a full legislative package here in New Jersey, a series of bills intended to put teeth in our environmental control efforts where needed, legislation that will create the facilities required, and legislation that will provide the funds necessary to implement a crash program. The people who are dying are our friends and neighbors, our mothers and fathers, even our sons and daughters. It is time for us to fight back and so let us begin these hearings not on a note of despair, but with a sense of dedication and in the spirit of hope. Mankind has faced many killers down through the ages, from the plague to TB, pneumonia, and polio. We have

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conquered them all, each in its turn. We can win this battle too. Of course, it won't be won in the halls of the Legislature, but in the laboratories and our research centers. And it won't be won by politicians, but by doctors and scientists and dedicated laymen such as those who staff the American Cancer Soceity. However, we elected officials can play a part in the struggle. We can give those in the front line the tools they need. We can provide them with the weapons they need by adopting a forceful "let the chips fall where they may" legislative program. Drafting such a program is the purpose of these hearings.

I would like to also preface the remarks with a statement that this is a public meeting today, limited to certain officials in our State. But it by no means is limited to this public meeting today. We will have a number of public hearings with such groups as environmentalists, the industry and other interested groups. They will have an ample opportunity to express their views to this committee. We are not limiting anyone. We intend to have an extended public hearing. Today, as I indicated to the witnesses, we desire with some limitation to focus on the nature and extent of the problem.

Senator Mc Gahn, do you have a statement or a comment.

SENATOR MC GAHN: Not at this time.

SENATOR SKEVIN: Commissioner Bardin is our first witness and he has an engagement on the shore, so we are going to give him the opportunity to make the first statement before our Commission. Mr. Bardin.

D A V I D J. B A R D I N: Thank you, Chairman Skevin, Vice-Chairman Mc Gahn, we appreciate the invitation to the Department of Environmental Protection to participate in the important efforts of this Commission of the State Senate of New Jersey. You have requested that we address the extent of

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the cancer problem in New Jersey, as well as present the Department's views on the data, studies and statistical analyses which have been performed which attempt to define the scope of this problem in New Jersey. I will address my remarks today to those questions. I am accompanied by Dr. Peter W. Preuss of my Department. Dr. Preuss is the Deputy to the Assistant Commissioner of Science. I am also accompanied by Dr. Glenn Paulson, and Dr. Preuss will stay with you throughout the day, Mr. Chairman, and be available for any questions that might come up in my absence. Of course, Commissioner Finley and her able staff will be covering issues which interface with and complement the issues that we are analyzing.

A brief review of some of the cancer statistics for New Jersey and the nation will put the discussion into perspective. In 1974, cancer killed more than 350,000 Americans. The American Cancer Society estimates that this year about 370,000 people will die of the family of diseases that we call cancer. The Cancer Society also estimates that about 54 million Americans now living will eventually develop a cancer; that amounts to one of every four Americans.

New Jersey will unfortunately share in the toll of illness and death and share rather heavily. The Cancer Society estimates that there will be over 26,000 new cancer cases discovered in New Jersey in 1976, and that this year 14,600 people in our State will die from cancer. And I emphasize that the 14,000 includes some cases discovered in 1976 and some cases discovered previously. The anticipated death toll represents about 4% of those who will die from the disease in the United States as a whole; whereas the New Jersey population represents only 3.5% of the United States population. So the cancer death rate is expected to be higher in New Jersey than the national average. And that is a key fact any way you analyze the data.

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and we will in our statement go further into the question of statistics. But before we do, please note that medical science has only fragmentary knowledge as to how and why cancers develop; thus the exact cause or causes of all observed cancers cannot now be defined with much precision.

Several medical and scientific organizations - including the World Health Organization - suggest that in a broad sense the environmental factors may be the causal agents for a majority of the cancer cases in contrast to non-environmental factors, such as heredity on the one hand and viruses on the other. The environmental factors include a broad spectrum of concerns including,notably, cigarette smoking and exposure to cancer causing agents in industries, and exposure to cancer causing pollutants in the air, water,and in the food we eat, as well as dietary habits, the proportions of fatty and other foods in the diet of one community or one society and exposure to the natural solar radiation.

Last year the National Cancer Institute published a Cancer Atlas which galvinized public attention. We should be sure to try to understand correctly the statistics and maps in the Atlas. We have all seen the Atlas, which marks in red various categories of population and various categories of cancers, the significant high incidences in the highest decile. Please note that the Atlas uses the 3,050 counties throughout the United States as statistical units - geographical units. The areas marked in red in the Atlas are in each case a county or a group of counties. In order to be marked in red, the particular county must have been analyzed with regard to its mortality rate. A mortality rate is the number of deaths per hundred thousand of population. It is not the absolute number of deaths, but it is the number per hundred thousand - an attempt

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to achieve some equalization or comparablity between highly populated and less populated counties.

Nonetheless, in order to show up in red a county must meet two tests. First, it must rank amongst the top 10% of counties for that purpose. There are 305 counties in this country that could rank amongst the top in terms of male mortality rates or male mortality rates for a certain type of cancer or another. Second, and in addition, the statistical analysis by the National Cancer Institute must conclude that the county has a mortality rate which is higher than the national average mortality rate to a significantly higher extent. The difference must be significant. There are factors in the way statistics work that tend to give prominence to certain counties. I just want to caution you about that.

The statistical technique employed tends to show red for high population counties somewhat more often than for low population counties. Just to illustrate that, if you have a county with a population of 100 - and we have none such in New Jersey - you could be three times the national average with cancer mortality rates, or many more than three times, and it could not be very significant if it meant one mortality in a given time period. So the statistical technique discounts the low population county. It demands a much broader interval of significance - of statistical difference - for the relatively low population counties. Also, the map in the Atlas for all male cancers, for example, has about 120 counties colored red out of the total of 3,050 counties; and these 120 counties contain almost 45 million people - roughly a quarter of the population of New Jersey. So most New Jersey counties, since they are heavily populated, have a somewhat greater tendency to show up in red than counties in some other parts of the country. The same is true of other metropolitan counties outside

New Jersey. But having said all that, it should not minimize our concerns, because I remind you again, on the basic over-all statistic with which I started, the prediction that we are going to sustain 4% of the cancer deaths in the country even though we have only 3.5% of the population. Plainly, we have a problem which is somewhat greater in New Jersey than elsewhere. We have a national problem, Senator Skevin, and I think the remarks with which you started this meeting - very appropriate remarks - should really apply to many other states.

Elementary responsibility requires us to deal with the problem to protect our people, even if we were not the highest in any particular incidence, simply because we have a significant problem. For that reason, Governor Byrne issued Executive Order #40 last month,which you have. Basing it on the reports submitted to him by the Department of Environmental Protection and the Department of Health, the Governor established a cabinet committee on cancer control, and assigned to the individual departments and to the Committee responsibilities relating to the prevention, study and control of cancer. The committee will basically be a coordinating committee with the individual departments carrying out major pieces of the action - notably including the two departments represented here today.

My Department's program I would like to describe to you briefly as being a part of the overall effort. We now believe that my department should do six different kinds of things. One, determine the presence and concentrations of carcinogenic substances in the environment; two, test the aggregated samples of air, water and waste materials - which we may be collecting anyhow for other purposes - subject them to tests in appropriate laboratories for carcinogenic, mutagenic or other toxic potential; for example, are there any particular places in New Jersey where the conglomerate mass of particles in the air turn out to be more carcinogenic in

RSV 0013661

fact, for whatever reason, than elsewhere in New Jersey or the country.

Three, to inventory and monitor the production and transport of toxic substances and their waste products known or suspected of being carcinogenic.

Four, to monitor environmental conditions at areas determined to have either known or the potential for high cancer prevalence. What would trigger off that kind of monitoring would be studies and conclusions from the Department of Health working from the other end of the analytic spectrum.

Five, reduce the water-borne discharges of exotic substances and protect drinking water supplies. There is already general regulatory authority in the Department of Environmental Protection in these cases. And it is a question of using it to deal with these very special cancer health problems as they are identified by the national scientific community and our work with the Department of Health in academic institutions here in the State.

And, finally, reduce the air-borne discharges of such substances when the same points would apply, as I have just mentioned.

The Legislature has shown its appropriate concern by the Senate's establishing this Commission, and charging it with the tasks of studying the reasons for the high incidence of cancer in New Jersey, and to examine steps to control exposure to cancer causing agents; moreover, I raised the subject in March - three months ago - with the Joint Appropriations Commitee. At that time I requested and appropriation of $2,340,000 for the Department of Environmental Protection to fund a program dealing with cancer causing agents and other hazardous or toxic substances in the environment. The Joint Committee passed Resolution #68 which restores $500,000 for this program in the

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next fiscal year out of the amount which I requested. The $500,000 will permit a modest start to be made, but it will not allow implementation of the full program urged by the Department of Environmental Protection, not to mention the needs of the Department of Health and other needs that may be identified by the cabinet committee. So I ask this Commission's studied and reasoned support for additional monies to be provided at an appropriate early date in whatever supplemental appropriations bill may be possible for the forthcoming fiscal year.

I can't emphasize too strongly, Chairman Skevin, the point implicit in your opening remarks, that passing legislation or issuing declarations is not going to be meaningful by itself unless we are prepared as a State and a community and a society to raise the funds and make the effort to really carry through on our concern. My Department intends to utilize existing authority to implement programs that will be effective in reducing the exposure of people to carcinogens to the extent that our resources permit us to do so. During the period ahead, should there emerge a need for new substantive legislation, we will advise you promptly. We will also stand ready to report to this Commission on our progress as specific parts of Governor Byrne's program are selected and are implemented.

I want to stress and reemphasize that doing the job will take a sustained effort over a long time to identify and to control the environmental causes of cancer. Like other public health programs, any state program toward cancer control requires a continuing commitment on both the part of the Legislature and the Executive branches of government, as well as close coordination with the scientific and academic communities and the public, federal officials, all of which transcends elections and administrations. The mechanism should be established - such as those being discussed today - that will provide for the cooperation

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and mutual support necessary to begin to resolve the questions posed by the prevalence of cancer in New Jersey, and to take appropriate steps to reduce human exposure to the causative agents wherever feasible. A carefully planned and continuing program will give us a chance to reduce the incidence of cancer over a reasonable period of years. That program will take wisdom and foresight and simply the tough decision of putting our money where our mouth is. Thank you very much, Mr. Chairman. I will be happy to try to answer any questions that you may wish to address to me.

SENATOR SKEVIN: Thank you, Commissioner. In terms of environmental factors, we of course have heard the statistic that perhaps up to 90% of cancer can be attributable to environmental factors. Do we have some verification in that area?

COMMISSIONER BARDIN: As our report to Governor Byrne pointed out, those statements are based on a variety of experimental or analytic observations. For example, ethnic groups have been analyzed in their home countries for cancer rates - the mortality rates or sometimes the prevalence rate for 100,000 of population - and one finds that when an immigrant group leaves Country A and comes to Country B, where it is exposed to a different setting, in several cases there are dramatic changes in the cancer prevalence rate.

Number two, certain cancers that have been identified very specifically correlated statistically very specifically to particular industrial locations with a clear concentration of cases in terms of the work force immediately exposed or others which are exposed to emmissions or pollutions or cancer causing agents from those facilities. So there are these kinds of evidences and others. All of the evidence as to cigarette smoking deals with an environmental circumstance which many of

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our people impose upon ourselves by inhaling the smoke of the cigarette. Perhaps it has effects on others beyond the immediate user, and we find that medical statisticians found a correlation between cigarette smoking and the incidence of lung cancer, for example, but they also find a multiplier effect in some instances that where people are exposed, for example, to the asbestos particles and cigarettes, there are far, far higher cancer incidents, several times as many as just the excess cancer cases caused by cigarette smoking alone or the exposure to cancer.

Finally, we have very significant differences in the rates in different regions of the United States, and some of them seem to have a geographic incidence; for example, the amount of ultra-violet radiation that reaches the earth from the sun varies in belts around the globe, and that seems to be clearly a factor in certain skin cancer incidents.

SENATOR SKEVIN: Commissioner, in terms of our future public hearings, do you have any recommendations as far as research groups or experts in this area?

COMMISSIONER BARDIN: Well, there are certainly a number of groups with great expertise, and I think Commissioner Finley in some ways is particularly qualified to address that question over the next few months.

We have been touching base with some of the people in the medical field. And interestingly enough there is one of the great research institutions in this state and in the country that one doesn't think of primarily as medical, the Bell Telephone Labs at Murray Hill. Mr. William Baker, the President of Bell Labs, serves on the National Cancer Board, and he in that capacity is reviewing the statistics that we are discussing today. He is also the chief executive of one of the foremost research agencies in the country that has dealt with a number of environmental problems such as ozone and transport phenomenon concerning smog.

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He has been a leader in that, and that is just one of many institutions which could be helpful in suggesting to us ways to go, directions to go into, not to mention the medical research capabilities in New Jersey and in the area mutually surrounding New Jersey.

SENATOR SKEVIN: Commissioner, I know that tax is a sensitive subject these days --- Good morning, Senator, welcome. Senator Dumont is now with us.

In terms of taxes, of course, it is a sensitive subject. Do you have any reaction to the proposal of having a cigarette tax used for cancer research?

COMMISSIONER BARDIN: Well, today was the first I heard of the proposal, Senator Skevin. As I understand it, it is to raise $8 million for cancer research and cancer monitoring, and a program of the State's response to the issue and to raise it by adding 1¢ to the cigarette tax which would - as you put it - be a rather nominal addition to the price of a pack of cigarettes.

My first reaction is that it is an excellent proposal, Senator, and I congratulate you for it.

SENATOR SKEVIN: Senator Mc Gahn.

SENATOR MC GAHN: I have no questions to ask of the Commissioner.

SENATOR SKEVIN: Senator Dumont.

SENATOR DUMONT: No questions.

SENATOR SKEVIN: Commissioner, thank you. We are going to ask you to be available for future public hearings which we intend to have over the next year or so. We thank you for your appearance. Back to the beaches.

COMMISSIONER BARDIN: Thank you, Mr. Chairman.

SENATOR SKEVIN: The Commissioner of Health, Doctor Finley.

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D R. J O A N N E E. F I N L E Y: Senator Skevin and members of the Legislature's Commission:

It is my pleasure to address you on the topic of cancer in New Jersey, although no pleasure can be derived from this topic. But I do come here in a mood of optimism. The very fact that you are taking the trouble to investigate this whole problem to do something about it in New Jersey is one of the reasons for my optimism.

As I understand what you would like me to do, you have asked that I start by describing how the rates in the Cancer Atlas were derived, to give you some significant examples of the cancer problem in New Jersey, and then to suggest how the State Health Department, for now at least, can help derive more exact knowledge so we can control this problem.

Mason and McKay of the National Cancer Institute,who produced the Atlas to which Commissioner Bardin referred, certainly must earn our gratitude for a nationwide study describing cancer death rates by counties of residence of cancer victims. I am not going into as much detail because Dave Bardin has given both the pros and cons of some of these statistics. But I do think I need to say something about what is useful and what we need to go on and do from there.

Statisticians who work with mortality data, if they are doing their work properly, should see, first of all, that they report in race. I think that is sort of obvious as to why. For example, in a small county that had only a small number of cases - say, ten people - but a population of 1,000 - we would have a rate of one in one hundred. In a large county with, say,100 cases, but a large population, we might actually have a smaller rate. So you really don't have a fair understanding of the problem unless you reduce it to race. If it was just numerical, you would not fairly understand it.

Another thing to praise about the Mason and McKay statistics is that these are age-adjusted rates. What this means is that they

have corrected the rate for variations in age distributions in those populations. For example, if one county had 90 percent of its population under 30 and another county had 90 percent of its population over 60, it would not be fair to compare the rates of cancer in these two communities. Cancer risk, as we know, is most dependent on age. So the rates have been standardized to the distribution of age groups. Thus, the rates for New Jersey that we will be talking about are standardized and we may fairly compare any county to any other county.

How do these statistics come about? All death certificates in the United States are collected by the National Center for Health Statistics. The State Health Department and local registrars are responsible for the acquisition of these statistics and must report them on to the National Center for Health Statistics. Each death certificate is not only coded for the usual place of residence but also for the primary cause of death. Usually, if cancer is listed on the death certificate in any way, it will be implicated as a contributor to the cause of death and may be listed as the prime cause. Mason and McKay analyzed all death certificates for the years 1950 through 1969. For each county, they formulated rates of cancer by sex and by race. From these data, they were able to calculate which counties were at markedly high risk of a specific cite of cancer, and which were at markedly low risk.

They also used the maps which Commissioner Bardin has actually showed you. Those counties that had rates in the top 10 percent of all counties in the country were colored in red. New Jersey has had much publicity lately because many of our counties, unfortunately, are painted red.

One really cannot be critical of these studies. Mason and McKay have chosen the only method that one could have used to adequately compare the thousands of diverse counties in the United States. Perhaps all we can say is that the study did not go far enough, in that the only variable they looked at was usual place of

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residence rather than the many other variables that we now think are related to cancer risk; for example, usual occupation, place of birth, and several others. But again we might add these are things that are not necessarily on death certificates.

I would like to describe some of their results that are pertinent to us today that we really have to understand the meaning of for New Jersey. Yes, New Jersey has amongst the highest rates of cancer mortality of any state in the nation. As you will see from the first chart -- and I might say to people in the audience who are concerned, we have some extra copies of these charts -- you will see that New Jersey led the nation in cancer mortality for white males. The death rate for white males in this 20-year span was 205 per 100,000, compared to a national rate of 175. However, pay close attention - it was a close race. Although New Jersey leads, it is only fair to point out that we are not markedly different from many other states that follow on our heels through the whole bottom half of the pyramid. We are not alone in the problem.

For white females, the New Jersey mortality rate was 148. Now that was exceeded by only one other state, and compares to a national rate of 130. For nonwhite males, the New Jersey cancer mortality is fifth in the nation, with a rate of 230, compared to a national rate of 184. For nonwhite females, New Jersey ranks also fifth, with a death rate due to cancer of 163, compared to a national rate of 139.

Since I reviewed those numbers relatively quickly, why don't you look at the second page of the handout. This summarizes the New Jersey rates of cancer compared to the appropriate United States comparison. The black bars on this chart are New Jersey by race-sex groups and the white bars are the average rates in the nation. Again you can see that New Jersey always exceeds the national average. You can see from this graph that rates for non-whites exceed those for whites everywhere in the country and rates for males exceed those for females. To tie in what I said in

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beginning this presentation, these differences cannot be explained merely by saying that either New Jersey is a more populated state or that we have an older population. These factors have already been taken into account in standardizing the data and they do not play any role in explaining the differences in the rates that we see.

Let us discuss some of the specific cancers that seem especially to afflict the New Jersey population. For each cancer site - for instance, breast or bladder or colon - some county had to come out with the highest rate in the United States. For example, Flagler County, in the State of Florida, has the highest death rate for lung cancer among white males, not a New Jersey county; Corson County, South Dakota, has the highest death rate for breast cancer among white females. Salem County, New Jersey, has the highest death rate of bladder cancer among white males. This perhaps is the most telling information about New Jersey presented by the Mason and McKay study. The age-adjusted white male bladder cancer death rate for Salem County is 16.1 per 100,000 and this far exceeds the national rate of 6.8.

It has been suggested that bladder cancer is largely due to occupational exposure or, as Commissioner Bardin would say, an environmental exposure. The New Jersey State Health Department fortunately has been funded by the National Cancer Institute to look into the occupational associations of bladder cancer throughout New Jersey, and this research is now well on its way within our department.

You might ask how the bladder cancer rates in the rest of the counties in New Jersey compare to the United States in general. The next page of charts will show you something about that. The line down the middle has defined the average United States rate as "1". The relative rate for every other county in New Jersey is outlined. As you can see on the second line, Atlantic County has a relative rate of 1.25. Or, in other words, the rate there is 125 percent of the United States rate. On the other hand, Salem

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County, which is unmistakeable, the longest line on the chart, has a relative rate of almost 230 percent. Although Salem County is highlighted because of its outstanding risk of bladder cancer, you can see that every other county in New Jersey shares a similar elevated risk as compared to the United States.

In the next graphs, we have taken other leading causes of death by cancer by body site and, in all instances, while certainly we cannot say that New Jersey is first - and we would not want to say that we are first - you can see that most of our counties exceed the national rates.

It might be reasonable at this time to interject that one should not be complacent if our population comes close to the United States average, since with many of these cancers - and this has already been mentioned by Commissioner Bardin - there are marked differences in cancer rates between nations of the world. So, for example, if a county in New Jersey suffered from cancer of the breast at the same rate as the United States in general, we would still far exceed the much lower national averages that are seen, for instance, in Japan.

The last sheet of graphs shows you that lung cancer for white males is also far in excess in New Jersey. I want to stress that also because I am in total agreement about that statistically-proven relationships between lung cancer and smoking, but there is also the equally statistically-proven association between lung cancer, smoking and the introduction of other substances, such as asbestos or fumes that can be breathed. So lung cancer and bladder cancer both kind of lend themselves to my suspicion,which our research funded by NCI will set out to make more scientific, that it is mainly workers that we may have at risk in New Jersey.

What's to be done now? We can see from the Mason and McKay data that the population of New Jersey is at greater risk of death from cancer than the population of the United States on the average. This is a motivating force to find out more. I would love to be

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able to say thatwe knew all of the reasons for the excess risk in New Jersey and the solution was merely a matter of controlling exposure to these risks. But this would simply not be scientifically true. There are many feasible explanations for excess risk that we suffer, and we have to be honest in saying that to a large extent we are still trying to figure out what the causes of specific risks are in New Jersey. In order to do this job, we must have certain resources available. The prime resource necessary is something we call a cancer registry. Senator Skevin has already referred to his very good understanding of what an important tool this is.

Since a cancer registry means different things to different people, let me describe what the Health Department is suggesting. We believe that by approval of the Public Health Council, the Commissioner of Health can make cancer a mandatorily reportable disease in New Jersey. At present, more than 20 communicable diseases are reportable. Our department has had numerous years of experience in supervising the collection of this data, analysis and enforcement of disease reporting. Cancer could, we believe, be made a reportable disease. We also would have no difficulty in dealing with the problems of privacy or confidentiality. Our agency as a State agency is specifically covered in State health regulations. In fact, over the years dealing with such emotionally-charged health problems as venereal disease, our protection of individual rights and privacy has not been called into question. It really is important to raise the issue of confidentiality because it is a very delicate issue to handle in setting up a registry because there are other users of the data whom we would like to allow to use it, people who wish to research various treatment methods. But we are constrained from giving out the names of patients and we are really going to have to face how to deal with that. Cancer cases could be reported by either laboratories where most cancer

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is diagnosed through the viewing of pathology specimens, and could be required to be reported by hospitals where patients are cared for or by physicians. We have gotten very used in New Jersey thinking about the dollar value you get, thinking about cost-benefits. So we have estimated that a sufficient number of cases would be reported if we relied in the mandatory reporting on laboratories alone. It would double the cost to add the requirement that reporting be by hospitals and increase it even further to require that reporting be by physicians. We believe that well over 90 percent of living cancer cases could be picked up by, at this point, merely requiring laboratories,where diagnosis is made from tissue analysis or other analysis, to report.

Basically now I have described what a cancer registry is. Let me describe what it does and what it doesn't do. Any newly-diagnosed cancer seen by a laboratory would be reported to us. There is an advantage in reporting newly-diagnosed cases rather than at the time of death. I think that is pretty obvious. Patients would then be available voluntarily for interviewing rather than only having next of kin, as you have to do in studying the past histories of the dead. We would also be able to generate rates of cancer for different ages and sexes and races according to the communities in which patients live and according to such other needed information as occupation, ethnic origin, education, dietary and smoking histories. We would be able to offer explanations from this kind of data as to why rates might vary, not only by county but within a given group of people who may be exposed to the same problem. For instance, if we find that males who work in one industry have a higher rate of liver cancer than those who live in the same town but work in another industry, that is a scientifically important fact.

To be blunt, however, many states have cancer resgistries that only collect data. A registry must be sufficiently funded,

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not only to properly and adequately collect the data, but to have trained individuals around to investigate from these data. To have a registry alone gives us rooms full of paper only and that would be ludicrous.

Numerous experts in the field of cancer have estimated that greater than 80 percent of cancer is caused by environmental factors. No one knows - and you asked this question earlier - at this point for sure, except in a few cases like asbestos, vinyl chloride, etc., and smoking, exactly what the environmental factors are. And no one knows for sure whether these are environmental factors only in the working place or in the home or in the air breathed around the working place. We would hope that data generated by a cancer registry will help answer some of these problems. We are optimistic, however. If we should prove, for example, that 80 percent of cancer is caused by environmental factors, these exposures can be controlled and lives can be saved.

If we could get this running start in New Jersey to set up the registry, analyze these data and investigate the probable associations in clusters of cancer patients between work place or lifestyle, or general environment, we could use this knowledge to save lives.

For example, there are 14 chemical substances used or produced in the work place that have already been listed by the National Institute of Occupational Safety and Health as proven or suspected of being carcinogenic, at least in animal research. If these data generated by the registry I have spoken of leads us almost solely to workers dealing with these substances - and in a way that's a form of human research without having to subject human beings to dangers - there are both programs of protections of these workers, and for early detection through medical surveillance that can be set up.

Early detection and effective treatment in time have always been the key, in terms of our current knowledge, to preventing

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death from cancer. Many industries, in my belief, wish to cooperate, but the smaller ones often need the most assistance. The larger ones are already doing, in some instances, an effective job of worker protection; but they could, with proper organization, be combined to share their technical knowledge on effective protection and occupational health programs with those who do not have the ability to do this.

This points to another data set that we need. We must map the sources in our State of uncontrolled carcinogenic pollution. We need an inventory of work sites using, importing or producing the kinds of hazardous substances I have mentioned. We need to know what programs and protective systems these sites have established or what they could do they are not doing. We need to know what protections they offer to the general environment when they dispose of their hazardous wastes.

There is knowledge in the scientific community to apply to the early detection of many kinds of cancer when appropriate data point the way to go. There is even knowledge to apply in the laboratory to analyzing which members of an equally-exposed population are most likely to develop cancer because not all people exposed to the same thing come down with the same disease at all or at the same rate. So applying this kind of knowledge is the essence of prevention.

There is equally good scientific knowledge to apply to making the work place and the environment safer, either through protecting the exposed,or through some chemical alteration of a process, or a waste product. This is the essence of preventing economic decline. A productive economy is as important to New Jersey as a productive people. This also is a very delicate matter which we have to confront because we are not proposing to control cancer and drive industry out of New Jersey.

Knowledge is wealth and knowledge is health. If we can bring the epidemiologic knowledge from the use of a registry

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together with knowledge from an inventory of sites of carcinogenic substances, we can develop the proper programs jointly in a partnership with industry, occupational medicine, and union health and safety committees to keep our people alive and well and, at the same time, to keep their jobs intact, but safer. The Health Department can be the trigger mechanism to see that DEP, for example, goes to the right places at the right time to monitor the toxic substances.

Finally, New Jersey needs to improve its laboratory capabilities to monitor for the presence of these toxic substances. The equipment and techniques used in virologic or bacteriologic analyses may not lend themselves to hunting for the presence of, say, beta naphothylamine, a carcinogenic substance. There are very few laboratories in the United States, as a matter of fact, equipped to do these large-scale analyses.

If New Jersey is determined to do a job for its people and its industries to diminish our risk of cancer, where better to center the necessary capability than in the Health Department's laboratory? We already do the water analyses for DEP for the presence of bacteria. We would need to be equipped to do the same analyses for the presence of carcinogenic substances.

In a report prepared for your Commission and the Governor, the Health Department has given a budget. For $1.5 million, the full spectrum of what I have been talking about - cancer epidemiology, cooperative occupationally-based control programs, and laboratory back-up - could be mounted. The dollar value of productive lives saved, or the reduction in costs to Medicaid or health insurance programs for expensive hospitalization or exotic treatment for cancers which might have been prevented could easily be calculated in the billions.

I hope then that you will understand it as a constructive suggestion when I close by saying that the primary services a

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Legislature can perform to help us deal with this problem is to also tackle the State's eternal revenue-raising question. We must have enough revenue in New Jersey, raised in the fairest way, not only to properly educate our children, but also to save the lives and health of their breadwinner parents. Thank you.

SENATOR SKEVIN: Thank you, Commissioner.

Commissioner, in terms of the economic effect cancer has in New Jersey, I have heard the statistic that the total cost, including doctor bills, therapy, loss wages, insurance, indirect costs, etc., each year ranges between perhaps $750 million to $1 billion. Is that accurate? Do you feel that is within the area of the costs involved on those particular areas?

DR. FINLEY: I think that would at least be accurate. As I said, I would say closer to $1 billion. We will know more, again, when there is a registry and when we can study the living because you will know the age, you will know what their earnings were, you will know all the things actuaries take into consideration, and you will also know how long and in what kind of treatment they have been and what that kind of treatment has cost. But my guess would be with high rates like this, which means a fairly sizable number of probably productive people involved, that you would really have a huge economic loss.

SENATOR SKEVIN: As regards to suggested research witnesses and groups, Commissioner, Commissioner Bardin mentioned you might have some suggestions for our Commission.

DR. FINLEY: Well, I take a little different view, as a physician, of the kind of research that we need to be doing in the country now than some other scientist might take. I don't think that we should diminish the kind of research that the National Cancer Institute is doing in the basic laboratory, the clinical research. But there is very little priority given by any of the federal programs to research in applied control

techniques, the kinds of things that we know as we do in cervical cancer where we have a screening technique. And there are other cancers that we have in New Jersey where we have a knowledge we could apply. You could call that "research" if you want; I would rather call it a community-based program.

So I am one who said that I think there needs to be some shift in the priorities of the funding, a little bit less for pure research and a little bit more for community-based control programs, and also the kind of research that we do - substances research, etc.

You are also asking me, because Dave Bardin mentioned it, whom might I suggest in New Jersey. I do agree with his suggestion that some of our occupational health people that we have in our industries - he mentioned Bell Telephone - be included. Actually, in looking through a list of people, I note Exxon Research Lab in Linden has a former professor of Occupational Health of mine in medical school, a well-known person who has been all over the world studying the relationship between all kinds of diseases and the environment. I think it would be only fair to ask people from DuPont in Salem County. In other states, DuPont has - it may not have started it soon enough - today an outstanding program of worker protection, whereas a smaller company might not. This was brought up very clearly in the Keypone incident in Virginia where people got hysterical and wanted to close DuPont and DuPont didn't have any illnesses coming out of it. Keypone was a much, much smaller tag-end, almost in a garage. It was a distant cousin of Allied Chemical and had no protective program. So it would be interesting to hear from an industry everybody is saying probably did in the past cause the bladder cancer in Salem County. But we ought to hear what they are doing today. That is the kind of person you might call a researcher; I might call an applied scientist who is applying techniques to protection. I think you should call them.

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SENATOR MC GAHN: Commissioner, I think we can play games with statistics. Certainly, I think the increased cancer mortality that you have quoted that is existing in the State of New Jersey may simply indicate the rate of increase in cancer here. However, possibly also this may reflect the quality of medical diagnosis in this State, in that there is actually reporting on a factual basis. I think we are fully cognizant of the fact that in a number of states in cases where patients die of cancer, the death is not reported as cancer. It is reported as pneumonia. The primary cause is not stated as such. I think possibly that is one thing that has happened in the State. I think there is accurate reporting on the death certificates, particularly for those patients who die in hospitals, because there is a review of what has happened. I think this speaks well for the monitoring of health care in this particular State.

I would like to answer a question that the Chairman asked, "Should all this money be designated or delegated to research," by saying, no. John, I think community health programs, as the Commissioner has stated, are extremely important. Education, particularly as far as women are concerned, is vital, so that we can do screening for breast cancer and the Pap smear for cervical carcinoma, which they can get free of charge or for a nominal charge. They should be indoctrinated with the necessity for a yearly examination and, in certain specific instances, every six months. If they did this, I think, to a large degree, we could eliminate deaths - I did not say eliminate cancer - resulting from cancer of the breast and cervical carsinoma. They would require treatment, but that treatment at an early stage of the game can result in almost 95 percent cure.

That is about the only comment I have.

DR. FINLEY: I generally agree with you.

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SENATOR SKEVIN: Senator Dumont.

SENATOR DUMONT: Commissioner, your chart, particularly the colored one on the second page, indicates, of course, that the rate of death in male population is much greater than female. You also alluded to the business of smoking, and since it seems to me that there are more women smokers today than there are men, I would like you to explain why the death rate is higher among the men than the women?

COMMISSIONER FINLEY: The first thing that you are probably right about is that the men seem to have gotten scared sooner, and the rate of decreasing smoking is greater in men. Us women are not learning as fast.

I think that people who are following the trends in lung cancer deaths or in lung cancer morbidity year after year are finding a steady increase in the female population. This data which goes 20 years back, if it were the bars for just lung cancer, would show a higher mortality for men, because women --- Well, as the Virginia Slim ad improperly says, "You have come a long way, baby." Twenty years ago, you didn't think it was right to smoke and today you do. So men were smoking sooner, and therefore had a longer exposure. But, as I say, women seem to be catching up in the death rates.

SENATOR DUMONT: Yes, but there are also about ten million more women than men in the United States, and that is okay with me, but the fact remains, I am trying to find out what the correlation is between smoking and cancer deaths when the charts show - without exception - that regardless of the color of anybody involved here, the death rate is higher among the males than the females, considerably higher.

COMMISSIONER FINLEY: But I am saying, unless women learn to quit, you will probably not find that difference,

say, a decade from now.

SENATOR DUMONT: Do you think $1.5 million can cover the expense of what you have outlined in your statement, that is, the registering? In order to absorb and analyze the data that would be assembled in such a registry, is that sufficient to finance such a program?

COMMISSIONER FINLEY: Oh, registry is calculated in that budget which is at the end of the bound report. Registry is calculated to set it up and to use it properly, which is the key. It doesn't take any money to have the Public Health Council or the Legislature pass something, but it is the use of the data that is of concern.

We only calculate that to take approximately $200,000 so the $1.5 million is for a total program which extends our knowledge beyond the cervical cancer screening programs, that Dr. Mc Gahn has spoken about, to other kinds of control programs. We need to take the same knowledge we have for preventing the cervical cancer death into other areas, and extend it. I think the most neglected area in this State is the occupational health program area.

SENATOR DUMONT: But to do all you have listed on page 11 and previous statements, a budget of $1.5 million would be sufficient?

COMMISSIONER FINLEY: Yes, including beefing up the capability of the laboratory, yes.

SENATOR DUMONT: Thank you.

SENATOR SKEVIN: Senator Mc Gahn.

SENATOR MC GAHN: Senator Dumont, I think in your question to Commissioner Finley concerning the discrepancies apparent between the death rate of males and females, certainly there are other factors besides cigarette smoking alone. We must take into consideration the number of cigarettes that are

smoked; we must take into consideration other catalystic factors, for example, a man works in various types of environment, and generally speaking a woman does not. We do not know whether there is a genetic factor involved in this, and we do not know about individual immunilogical responses on the part of each of these individuals. These are the things that have to be looked into in order to explain the differences, as far as the rate is concerned. I don't think it is proper mainly to make a statement that smoking alone is involved in this.

Remember, also, that during flu epidemics there is an increase in the incidence of cancer deaths during any type of recurrent respiratory illness. Apparently the amount of respiratory illness is a factor that is responsible basically for flaring up this type of situation. All these factors, I think, have to be considered.

SENATOR DUMONT: I agree with you, but at the same time when so much emphasis is placed upon smoking it seems to me perhaps that is not quite right when there are these other factors involved.

SENATOR MC GAHN: I agree with you there, and I think if Senator Menza was here today he might say smoking cigarettes is dangerous to your health, but there is no relationship between smoking marijuana and lung cancer. And that is a plug for Senator Menza. I don't buy it, but it is a plug for Senator Menza.

COMMISSIONER FINLEY: Marijuana has not been shown to cause lung cancer.

SENATOR SKEVIN: Commissioner, on that note I want to thank you for your appearance, and again I want to extend our invitation to call you back for future hearings. Thank you very much.

COMMISSIONER FINLEY: Thank you.

SENATOR SKEVIN: Our next witness is John S. Quattrone of the American Cancer Society.

J O H N S. Q U A T T R O N E: Thank you, Senator Skevin and members of the Committee, the American Cancer Society has assumed a responsible role in helping to identify and hopefully reduce or eliminate the cancer causing substances in the environment. It also seeks to inform the public, the press and other media, as well as work with industry, labor and government to safeguard Americans from carcinogens.

Starting in 1951, the Society undertook the first major epidemiological study of the effects of cigarettes on the human body. This served to establish the connection between smoking and lung cancer, also heart disease, emphysema and chronic bronchitis. It has continued to actively campaign on the basis of scientific information to protect people from cigarette smoking, the single greatest known cause of fatal cancer in the environment. We believe lung cancer alone is reponsible for nearly 70,000 deaths in the United States annually.

Similarly, the American Cancer Society scientists have been in the forefront of those who established the relationship between inhaling asbestos particles in industries and the onset of certain tumors. This information has been put to work in helping change manufacturing processes to protect workers from the toxic effects of asbestos.

The Society has been the vanguard of those who determine the cancer causing potential of vinyl chloride, a substance which has very quickly been put under strict manufacturing controls.

The Society also continues to seek out, follow up, investigate and support research to determine and disseminate public information regarding other known and suspected environmental causes of cancer. Among those are over-exposure to ionizing

radiation, certain chemicals used in industry, certain food additives, medicines, asbestos and other pollutants both in the air and drinking water. It recently co-sponsored a scientific conference on the possible relationship of diet to cancer, and is sponsoring also research in this area.

I would now like to introduce Dr. Warren H. Knauer, a surgeon who specializes in cancer and its effects. Dr. Knauer is Chairman of the New Jersey Division's Executive Committee, which makes him our highest ranking volunteer associated with medical information. I would like Dr. Knauer to describe for this Committee what the New Jersey Division is doing medically in regard to the incidence of cancer in New Jersey. Dr. Knauer.

W A R R E N H. K N A U E R: Senator Skevin, members of the Committee. I would like to thank you for this opportunity of presenting the facts, as we in the Cancer Society know them, to you.

Here in New Jersey the American Cancer Society's New Jersey Division held on May 1st and 2nd of this year a seminar for phsicians of environmental cancer. It was entitled, "Cancer Risk Identification Within New Jersey, and Methods of Control."

Leading national authorities on major sites of cancer occurring within New Jersey - lung, breast, bladder, colon, rectum - as well as experts in screening and identifying carcinogens participated. More than 200 physicians from the greater New Jersey area attended. In addition to extensive media coverage, transcripts of the reports have been made available to the medical profession.

Also, here in New Jersey the American Cancer Society in conjunction with the New Jersey Dental Association recently conducted a statewide screening program for the detection of oral cancer, the cause of 8,000 deaths in the United States annually.

More than 9,000 New Jersey residents took advantage of this initial statewide effort that was provided by some 250 dentists, along with American Cancer Society volunteers and staff. Since early detection is the most positive known cure for cancer, we in the American Cancer Society are determined to help overcome fear, encourage through education regular and thorough medical check-ups, and provide screening programs for the detection of cancer. I am sure you are aware of our involvement in the breast screening program and also our human task force for cancer of the cervix.

The American Cancer Society believes in and will continue to pursue its long-established policy of applying science and medicine to identify carcinogens. It will cooperate with all groups, including labor, industry, the government, and other health organizations in eliminating the risks they pose. This is an essential part of the Society's announced purpose to control, and, if possible, to eliminate cancer as a disease of humanity.

The Society believes in concentrating the resources of science, medicine and cancer control on the problem of environmental carcinogens in order of priority, beginning with the most probably dangerous and widespread, and it belives that the solution to this very complex problem is necessarily pluralistic, combining public education, professional education, industrial responsibility, labor participation, government regulation, and individual action.

I would like to digress for a moment from my prepared remarks to answer Senator Dumont and his question of female lung cancer. I think if you look at the statistics just recently presented by the American Cancer Society over the past five to ten years, there is a sudden and tremendous increase in the rate of female lung cancer. This is paralleling that of the

male, however, it is twenty years later than the rise in the male cancer death rate. Inasmuch as we feel that cancer in some cases does take 15 to 20 years, I think in another 15 to 20 years, we will have a similar percentage in female as in male.

Secondly, you have been talking about tumor registries. I would like to comment on the fact that as Chairman of the Commission on Cancer, of the College of Surgeons, it is part of my responsibility to stimulate interest and give hospital tumor registry programs. We feel it is extremely important that we get relative and accurate data from each individual hospital before it is sent to any central agency, because inaccurate statistics will come out inaccurate from the central registry also.

I would like to suggest that any registry that might be formed accept the knowledge and the experience of the American College of Surgeons who have been doing this work since 1933. Finally, I would be happy to answer any of your questions before I turn the microphone over to Mr. Quattrone.

SENATOR SKEVIN: Thank you, Doctor. If I recall correctly, the American Cancer Society had a seminar in Cherry Hill in May. Would it be possible to obtain that transcript for our Commission?

DR. KNAUER: I will make it available.

SENATOR SKEVIN: I have been associated with the American Cancer Society, Bergen County unit for a number of years, and Mr. Quattrone, we don't have your title. I am very proud of the fact that I was counsel for them for many years. Are you the present President?

MR. QUATTRONE: The New Jersey Division.

SENATOR SKEVIN: I have no further questions. Senator Mc Gahn.

SENATOR MC GAHN: No questions.

SENATOR SKEVIN: Senator Dumont.

SENATOR DUMONT: No questions.

SENATOR SKEVIN: Thank you very much.

MR. QUATTRONE: Since we believe that the identification and elimination of environmentally caused cancer should be dealt with more effectively in the regulatory arena, we would like to make the following suggestions to the Legislature: One, to encourage by every means possible research aimed at pinpointing the causes of environmental cancer; two, encourage full utilization of the National Cancer Institute for screening potential environmental carcinogens; three, provide or support fair measures aimed at enforcing control over the introduction into water, air, or food of known or highly suspect cancer-causing agents.

Further, because cigarette smoking has been clearly established as the most positive cause of lung cancer, either singly or most usually in combination with other environmental carcinogens, we believe the Legislature should penalize through additional taxation the production, the dissemination of high tar, high nicotine cigarettes and at the same time help apply funds thus derived to the educational process of discouraging persons, especially youngsters, from acquiring the cigarette smoking habit. At a recent national meeting it was indicated that the number of young children, teenager, smoking has increased drastically over the past five years. This may be an area that you may be concerned with.

We also believe that the time has come to make cancer a reportable disease in the State of New Jersey. This in itself would strongly support cancer control efforts in numerous ways, specifically, making cancer a reportable disease would be a first

vital step toward the development of centralization cancer registry. This, by at least recording the incident by sight and outcome of the treatment,would help provide a specific picture of the status of cancer in New Jersey. Armed with that knowledge, our medical scientists could be greatly assisted in combatting cancer with the means at hand. They also would be able to direct future efforts with greater specifics and the effect to the end, that cancer may one day be eradicated as a human blight.

We, the members of the New Jersey Division of the American Cancer Society, agree with you, Senator Skevin, that this law against cancer can be won by the joint efforts of this Committee, other government agencies within the State,and the ACS. We thank you for allowing us the privilege of being here today to tell our story.

SENATOR SKEVIN: Thank you, Mr. Quattrone and Dr. Knauer. Are there any questions?

SENATOR MC GAHN: Mr. Quattrone, at what level do you think this disease should be reported? Should it be the responsibility of the physician or the hospital laboratory? As Dr. Knauer has already said, practically every hospital does have a tumor registry.

DR. KNAUER: At the present time, 25 hospitals in New Jersey have had a three-year approval by the College of Surgeons. There are 12 just recently who have requested evaluation. Over the past couple of years there has been a tremendous interest in developing a cancer program in the hospital which consists of not only a tumor registry but an educational program such as a tumor conference, whereby the phsicians go over the cancer cases in the hospital. But I think if we have programs like this in hospitals where the registry gets all the data not only from the pathologists, but the admission office and all the

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other areas that a patient has cancer, then each time the patient comes back to the hospital, his file is pulled, so they know that this patient has cancer.

SENATOR MC GAHN: Would that be the approach, then, that you would favor, tumor registry with tumor conference in practically all hospitals above a certain bed capacity, or if they have the ability to carry out actually that type of program?

DR. KNAUER: We are trying to initiate these programs in all hospitals over 100 beds, and there are about 110 or 112 in New Jersey.

SENATOR SKEVIN: Senator Dumont.

SENATOR DUMONT: What did you say, Mr. Quattrone, the Legislature ought to do in regard to smoking? What was that proposal again?

MR. QUATTRONE: We thought the increase in taxation would be warranted for a number of reasons, but we would like to go one step further. We feel that the funds should be used for educational programs throughout the entire state. We do this on a voluntary basis to clubs, organizations and industry, and our key to saving lives is through education. We are currently saving one out of three lives. We can save two out of three if we can get to the people and educate them about cancer's warning signals. We just can't get in to do the job educationally that we would like to, only because when we do ask, people are not agreeable to do it; however, I will say this: Right here in New Jersey and other areas where State agencies are, we have made available in the past several years educational programs to State employees that we have found to be extremely beneficial and has saved a lot right here in your own government. We only hope that we can get into every possible club and organization,

and industry in the State of New Jersey, because with this education, we can save two out of three lives - not one out of three. So maybe through this bill that is passed, possibly through the increase, part of this could be used to help us get the educational program going.

SENATOR DUMONT: You are referring specifically to an increase in the cigarette tax.

MR. QUATTRONE: Oh, yes, definitely.

SENATOR DUMONT: Well, do you realize that our cigarette tax already, which I think is 19¢ a pack, is higher than any of the states around us.

MR. QUATTRONE: If it was up to me, I would make it another 19c.

SENATOR DUMONT: Yes, I know, but the point is this: The higher we make it, the more we tend to put that industry in the underground, so that you get a lot of bootlegging and then you get the diminishing returns - not more money, but less money.

MR. QUATTRONE: I don't know the aspects of that. I only know that 70,000 deaths a year is more than the entire amount of deaths that we had in the Korean conflict, and we have 70,000 deaths from lung cancer alone.

SENATOR DUMONT: I am not denying that it may be a fine thing that you are proposing, but I am simply trying to point out to you that the tax will matter, because I think I know something about the tax situation in the State.

MR. QUATTRONE: A lot better than I do, sir.

SENATOR DUMONT: What I am trying to say is that you can very easily drive a particular business into bootlegging operations to the point where you don't get any increase at all by raising the tax because you lose on the whole tax, and you get into diminishing returns. Now, that is the thing

we have to be careful of. Our tax already is higher per pack than any state around. It is one of the highest in the nation at 19¢ a pack now. Thank you.

MR. QUATTRONE: Thank you.

SENATOR SKEVIN: Thank you. Dr. Lampan, Rutgers University.

J. O L I V E R L A M P A N: Good morning, Senators. It is a pleasure. I should perhaps explain my being here on behalf of Rutgers University. I am not directly - and I emphasize directly - concerned with cancer research; however, I am Director of the Institute of Microbiology at Watson Institute that has probably the largest cancer research program at the University, and since we are now attempting to take stock of what cancer research the University has, and organize a program there, I have had the responsibility, so at least as of this moment, I am the expert, at least, on cancer research, and not perhaps on cancer at the University. I can tell you what we are trying to do and where we stand.

Starting as I did at the last moment, what I had to do was to share with you some material. I have given you some information that I recently put together on the cancer related grants at Rutgers. I have also included three brief statements, one of which I made myself at the Institute. And finally, I have a bit of a summary included of the kinds of cancer research we are doing on the last page in terms of the type of research - the approaches to the cancer problem.

Let me just describe this to you briefly and then talk about their implications. If you look at the initial statement on cancer-related grants, you will see there are three or four major groups at the University concerned with cancer-related work. The Douglass College Biology Department is involved in problems of mutation and Dr. Axelrod and

Dr. Mc Cormick are now both becoming very active in certain problems of erythrocyte and hemoglobin changes in cancer and leukemia.

At the Institute we always try to have about five members of our staff or faculty working on problems related to cancer in both virology or immunology, really, as the means of approach. I should point out that a year ago we were considerably stronger in this field than we are. We lost two people who were most actively engaged in cancer research. One became chairman of the department at the university of a large cancer institute; another moved to a bigger cancer institute in the south; and I was very much taken aback, to find out that he said one of the reasons he decided to leave was because he didn't think much of the future prospects of Rutgers University. This has been the problem in building up, I think, the proper qualified people in this area with the expansion in cancer research that is going on in this country.

As you might notice, we have had two people on our staff who are involved - Simpson and Merimorish - from the standpoint of viruses, and who are interested in whether or not the many insect viruses, for instance, that are being injected into people all the time - although we don't realize it - are potentially carcinogenic, and the National Cancer Institute is funding this. Also there is a great deal of concern about the possible effects of things like the flu vaccines and some of the respiratory disease vaccines and the hidden potential there - that part of their information would get into the genetic apparatus of the cell and 30 years later cause cancer, this kind of thing. There is some evidence that some of them can be incorporated into the cell; on the other hand, I suppose one has to face the possibility of a potential danger. But on the other hand, their potential value is so tremendous that it

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is difficult to think in terms of halting their use, but what we are trying to do is intensively to investigate this whole problem and determine whether there is any significant problem.

Then we are involved in immunization against cancer in screening for materials that might have effectiveness in cancer. There are a couple of people in the Department of Chemistry who have rather large contracts for the National Cancer Institute synthesizing materials of eventual anti-tumor activity. I didn't give you any detail on these, but they are both related to some of the known anti-cancer agents. Then the viral biological research has always had several people working in this area such as Dr. Lethan who has been involved in ovarian cancer, and the history of the association of that group with cancer is that for a long time they ran a hospital for animals who carried cancer - largely dogs and cats - and used that for an experimental system and studied there the treatment of those cancers and the use of materials that were being considered as therapeutic agents for tumors.

As you will notice, there are also a lot of other small grants, largely funded by internal funds of the University from the research time of people or from such things as the Bush money, which is part of an estate left to the University for cancer-related work some years ago.

Finally, at the College of Agriculture, there are two men, Adams and Schwab, who are very much concerned with viruses of chickens. Of course, this,in itself, is of considerable economic importance. On the other hand, these viruses are very closely related to the leukemia viruses. So, at the same time, they provide a very good system for studying the nature and development of leukemia, and again its possible therapy.

On this last page, I have just sort of indicated that we have four or five people working on the relation of viruses to cancer - that is, the tumor virology; a couple of people on what you might call the straight molecular biology or cellular biology of cancer: and immunology, the tumor vaccines, anti-tumor enzymes, and cellular immunity to cancer; and then a certain amount of biochemistry in the synthesis of anti-tumor agents - things of this type.

What that means in essence is that we have a lot of faculty members who are spending small amounts of their time,as they have the research time or a student or a bit of funding, in this, but not very heavily personally committed. Then there are about seven or eight of the faculty who I think would probably identify themselves as primarily interested in the cancer problem, mostly working with outside funding: the National Science Foundation; the National Institutes of Health; and the American Cancer Society which has been quite active over the years too, particularly in our unit, supporting it.

I might point out and boast for our group that we have over the years trained a great many people who are now very actively concerned in the cancer program. In fact, Dr. Frank Raucher, the

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Director of the National Cancer Institute, is a graduate of our Institute. So we feel that we have been very closely concerned with this over the years.

We are at this point trying to hire two more people as replacements for the two who I mentioned have left, who will, in turn, be responsible for cancer work, one in immunology and one in the related virus program.

What this means is that there are a lot of small programs at Rutgers, largely funded from internal funds; a few special ones, largely on national funds. Of course, the University has seen fit, I think, to approach the matter in this way, in that we have not been organized for a direct assault on cancer any more than as the general University we have been involved in a direct program on heart disease or a direct program on diabetes, or things of that sort. It has been more of the broad background with, I think, perhaps the Medical Schools and groups of that sort being at the other extreme.

On the other hand, I personally also think this is an important role for the University. In the first place, we know that cancer is a variety of related diseases, a few of which we are now beginning to know how to cure, but most of which we have very little idea of how to deal with at this point. The statement has recently been made that it took 100 years to learn to control infectious diseases of bacteria and fungi, and things of this kind. Certainly, I think we are making a serious mistake if we believe that in ten years we are going to solve the cancer problem. And it will come out of, I think, the interrelated contributions of a number of groups.

So the University would continue this, I think, rather general approach, particularly since we know too, as has been mentioned, that there are multiple causes of cancer. It is clear that there are environmental causes. It is clear that in a number of instances, the deterioration of the immune system that occurs

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in elderly individuals or in animals that has been observed no longer permits the human or the animal to eliminate the foreign cancer cell as it appears. So the incidence goes up there. There is a fair amount of recent evidence, very difficult to interpret at this time, for nutritional factors in cancer. Of course, it is very clear that viruses can cause cancer in animals. There is much reason to think that they do cause it in humans, although we are not likely to have direct proof of that from an experiment. The point is that all of these may again be acting on each other; so we will have to understand probably the various ones before we can move very far.

Now, what can Rutgers then contribute under the circumstances to an expanded cancer effort? I would say, first, that within our current framework there are a number of people who, given somewhat better funding for supplies and assistants - encouragement of that sort - could make a number of contributions, this largely in the area of what one would term the cancer-related area rather than, I would say, directly on cancer. What we really need if we are going to ake the more direct approach are several groups of new and young, well-trained investigators, who could be in a couple of foci in the University where they could interact with one another and build up the mass needed to have a group that is working on cancer and emphasizing that and thinking about it and talking to each other about it, and, from this, coming up with additional ideas, etc.

I think only if such nuclei can be introduced can we really make a substantial amount of progress. And I must point out that it is really only that kind of a nucleus that traditionally has had any success in raising the needed research funds from the government. One of the things generally viewed in examining a research group is what the atmosphere is and how much help and interaction a person in a group will have. I think that research requires a continuing intensive effort of this sort; so this is really what we would have to do.

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As I think you know, Senator Skevin - I believe you are meeting with the Research Committee of the Cancer Institute of New Jersey in a couple of weeks - there are groups in the State that are trying to organize for major requests from the government for help. I think that two things are needed for this -- well, really three things. First, from what I have learned in talking to people from Washington, from the side of cancer treatment, a cancer registry of the kind you are talking about seems to be somthing, without which, they have no interest in giving any money for cancer control. They feel that without that they cannot properly assess the results of the programs and simply don't want to invest the money. I think that that is a critical beginning for this whole thing.

Secondly, they insist very strongly that while they are anxious to support large amounts of clinical research, there must be a solid base for this, so that the informations are in contact with people who are aware of the new developments in cancer. Again, without that, they are unlikely to do anything on a sizable scale in order to fund cancer research in New Jersey.

Finally, I think that the national government and the NIH are looking for direct evidence as well that the State and the people of New Jersey are serious about this business of wanting to be a part of the national cancer effort. Here is where action of the kind that you are talking about would be not only timely, but critical, at this point to get something moving, because while, as I have said, I think cancer is not going to be cured in ten years or twenty years - cancers will be cured, but not the over-all problem - the apparatus, the organization and the people who are going to do this are being assembled at this point. We are not training enough people. As you can see from our program, we really don't train people in cancer research. We train people who know things that can lead them into cancer research, but we are not directly in that. We must, I think, begin an effort on our own if we are to have any really substantial success in a couple

of years when it comes to attempting to obtain solid funding from the national government for a larger cancer program.

So I think perhaps I can certainly speak for the University in saying that we are working on this seriously and would certainly be prepared to cooperate and play whatever role we can in this entire program.

SENATOR SKEVIN: We welcome that cooperation and we certainly appreciate your presentation.

In terms of the research in New Jersey - and I am sure you are familiar with the entire picture - is there a center for the research program or are we going in many different directions in New Jersey?

DR. LAMPAN: Well, I can't tell you what information Dr. Finley's office would have on this. The American Cancer Society knows, I think, the clinical, the epidemiological side. But the Cancer Institute of New Jersey, the group that has formed itself for this purpose, is now attempting to pull together a reasonable resume of what is going on in clinical research; testing of drugs; new modes of therapy; a study of the cancer patient, himself; and basic research, laboratory and the related ones. It is for that that I prepared part of this material. Their Research Committee with whom you are going to meet has taken the lead in collecting the material, say, on the laboratory side. There is a comparable committee of physicians working, I think, with the Oncological Society of New Jersey, who are preparing a similar summary of the clinical research. That, hopefully, will give us the information we need as well in going to the government and it would be helpful to the State.

SENATOR SKEVIN: I am certain that you are aware of the fact that we have lost a considerable amount of grants and available moneys because we don't have the facilities and the matching funds and that type of thing.

DR. LAMPAN: Yes.

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SENATOR SKEVIN: Is that an accurate figure that we have lost about $30 million in 1975?

DR. LAMPAN: It seems reasonable. Certainly we have lost very substantial amounts. Again I might point out that our Institute was encouraged from some people from the National Cancer Institute several years ago to essentially convert itself into a Cancer Institute. This would have required rather large investments of money and new positions, and reluctantly we had to give it up. And we didn't want to give up our other activities as well. But there has continuously been this pressure from Washington that we have to be prepared to build up something substantial that will be a real contribution; otherwise, they are not interested in feeding in nickels.

SENATOR SKEVIN: Senator McGahn?

SENATOR MC GAHN: No questions.

SENATOR SKEVIN: Senator Dumont?

SENATOR DUMONT: How much would we have had to match, Doctor, in order to get this money that was lost?

DR. LAMPAN: A million plus maybe a million dollars a year to run it.

SENATOR DUMONT: One million to match? Is that what you said?

DR. LAMPAN: Yes, plus maybe a million dollars a year to run it.

It was the general feeling that the University was not in position to do very much. It was felt that we weren't in a position to do it. We had very limited resources ourselves. We still have a small amount of money from the endowment we have from the royalties on the streptomycin and neomycin patents, but not nearly enough to handle this.

SENATOR DUMONT: Thank you.

SENATOR SKEVIN: Doctor, I would like to extend an invitation to you to come back at our future hearings. Thank you very much

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for your fine presentation.

DR. LAMPAN: When you were mentioning other people who should be coming, I think you also may have hoped that I would be in position to speak for the College of Medicine and Dentistry of New Jersey. I am not, although I am generally aware of much of their cancer research. But they are actively organized for this purpose as well. I think a spokesman from there would be very worthwhile. Certainly, Dr. Coriell from the Institute for Cancer Research, which for a purely research cancer institute is probably the largest unit in New Jersey, and groups of that sort, I think could be very helpful.

SENATOR SKEVIN: Thank you very much, Doctor.

Congressman Minish who has been in the forefront of this cancer problem in New Jersey on a federal level was scheduled to appear this morning, but was unable at the last moment to make an appearance. However, he indicated he would send an aide or representative. Is his aide or representative here?

If not, this will conclude the public meeting of the Senate Commission on the Incidence of Cancer in New Jersey. We anticipate further public hearings. If you are interested in participating, please submit your name or the name of your organization to David Mattek, our Legislative Aide, or notify me of your interest in appearing at future public hearings by direct correspondence. Thank you very much.

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Appendix 9.

P U B L I C H E A R I N G

before

SENATE COMMISSION ON THE INCIDENCE OF CANCER IN NEW JERSEY

Held:
September 10, 1976
Senate Chamber
State House
Trenton, New Jersey

MEMBERS OF COMMISSION PRESENT:

Senator John M. Skevin (Chairman)
Senator Joseph L. McGahn

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SENATOR JOHN M. SKEVIN (Chairman): Good morning. We are going to convene the second hearing of the Senate Commission on the Incidence of Cancer in New Jersey.

My name is Senator Skevin from Bergen County. To my right is Senator and Doctor Joseph McGahn from Atlantic County. Senator Dumont, a member of this Commission, is unable to attend because of his recent operation. Senator Parker has a commitment that he cannot break, so he cannot be with us today. Senator Maressa, a recent addition to this Commission, is unable to attend, due to another commitment, but his aide, Eugene Feldman, is with us this morning.

Before going into the schedule of witnesses, as Chairman, I would like to take the prerogative of an opening statement.

When this Commission first met, I promised we would be an action group, not simply a platform for platitudes.

Indeed, the time for platitudes has passed.

The time for action is now. In fact, the hour is already late. Chemical names, such as Vinyl Chloride, Benzene, Carbon Tetrachloride, are becoming a part of our vocabulary here in New Jersey and our citizens have also learned another word - carcinogen. That is the scientific label for an ingredient that produces cancer. I say it is time to label these ingredients for what they are: criminals in our air and water and food - criminals that should be outlawed just as surely as we would outlaw any other activity that threatens our very lives, and even more importantly, the lives of our children and even the lives of those yet unborn.

As I have said, the time for action is now.

I believe the dimensions of a legislative program to mount a meaningful counter-attack against environmental cancer here in New Jersey are beginning to take shape.

Let me take a moment to discuss that program.

I see the need for a legislative package which would include the following:

(1) A bill listing by name those chemicals now established "beyond a reasonable doubt" to be cancer-causing agents and setting the limits beyond which their emission is illegal. Such a bill would, in my opinion, be "open-ended;" that is, it would be amended and additional names added to its list of illegal emissions whenever new evidence warranted their inclusion. We don't have to wait until that list is complete. We can start now.

(2) A bill banning the sale of any commercial or consumer product within the State of New Jersey, such as insecticides, herbicides, or building materials, which contains dangerous levels of cancer-producing agents.

(3) A bill providing for an increase in the State cigarette tax to provide the funds for the implementation of this counterattack, funds to purchase whatever testing and monitoring equipment is necessary and to supply the manpower for efficient enforcement.

(4) A bill making cancer a "reportable" disease so any doctor diagnosing a case of cancer in New Jersey would be required to report it to the Health Department as a means of improving our statistical knowledge of this disease.

We need the help of witnesses who are scheduled to appear before this Commission in drafting such legislation.

We need to determine what chemicals should be named and what levels should be deemed illegal.

We also need to determine what agency of the State government should be charged with the responsibility of enforcement. And I, for one, propose to put teeth in that enforcement policy by providing the agency with the power to close down any plant that violates our air.

I wanted to make my attitude - and I am sure that of this entire Commission - clear

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before witnesses begin their testimony today. If anyone is here to pay lip service to this problem, I warn you you are appearing before the wrong group.

We want to hear only from those who mean business, those who have had enough of scare headlines and who want to help provide our citizens with some positive signs of hope.

To those who say strong action will drive business from New Jersey and wreak economic havoc, I say I don't believe it.

I don't believe responsible management would refuse to join with us in such a crusade, rather than stand against us.

I am certain labor, who has been seeing its members dying premature deaths since the days of the Black Lung Disease in coal mines, will hail such action.

Let me ask this: If someone had evidence of a cannery that was canning food contaminated by botulism, would anyone suggest we should not close down that plant because we would put people out of work?

Would anyone defend a meat plant that was dealing in spoiled meat or a food processing plant that was operating under unsanitary conditions?

Why then this reluctance to ban the emission of death-dealing fumes into our air?

You have to subject your car to an emission test when you go for State inspection. Why shouldn't industry subject its plants to the same type of inspection?

To those who argue action here in New Jersey will be to little avail if other neighboring states continue to pollute the air, I say we have to start somewhere. So let's draw the line here in New Jersey and hope others will follow our lead.

The United States Environmental Protection Agency, which recently identified New Jersey as the site where the most deaths occur from environmental cancer, also says we are a long way from mounting any meaningful control measures.

I say they may not be ready to act in Washington, but we are ready to start moving in Trenton. We are ready to indict these chemicals, to say there is reasonable evidence to indict their guilt. And, most of all, we are ready to give the benefit of any doubt that may exist to our own people, not to the chemicals.

Now I would like to deviate from the order in which the witnesses are scheduled and call on Senator Maressa's aide, Eugene Feldman, to give the statement the Senator would have given this morning were he here.

E U G E N E F E L D M A N: Thank you, Senator.

The Senator sends his apology and he just wanted to make one or two brief statements, basically concerning the fact that numbers and statistics are fine to illustrate the needs for various activities, and we receive all this type of information from various sources as to what is going on. But until someone has lived through the heartache of a cancer victim and experienced the problems and situations that are going on the last months of his life, the effect it has on everyone involved with him, the pain and suffering, both physically and emotionally, of all concerned, one cannot realize the tragedy of it.

I am quoting the Senator. I have just been through this with my wife who died this summer at the age of 49. And I intend to do anything that I can to see to it that we in New Jersey, the Cancer Capital of the Nation, start to eliminate this undistinguished honor.

A lot of information has been gathered and more will be gathered today as to some of the reasons and causes concerning the high rate in New Jersey. Primarily, this information will concern a lot of facilities, many of which are found in my particular area of the State.

In Salem County, we have the large du Pont complex. In Winslow Township, we have

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the Owens Illinois complex. In Berlin, we have Johns Mansville. In Winslow, we also have the Matty Beck Chemical Company - making fairly complex and sensitive chemicals.

I realize that we have the tiger by the tail where these industries employ thousands of people and that the unemployment situation in New Jersey is critical. But the health and safety of all the people who live in the State should be protected. I don't doubt that through the years, the EPA in Washington and the DEP in New Jersey certainly have acquired enough information that will allow these industries to continue to operate, but with the needed safeguards to clean up the cancer-causing agents. The costs can no longer be the primary reason for not enforcing many of these principles.

I hope that when additional hearings are scheduled, that we are able to schedule one in the South Jersey area. Thank you.

SENATOR SKEVIN: Thank you, Mr. Feldman.

Senator McGahn, do you have any questions?

SENATOR MC GAHN: No.

SENATOR SKEVIN: I will call our first scheduled witness, Dr. Martin Zanna, Director of the Chronic Disease Services, New Jersey Department of Health.

D R. M A R T I N Z A N N A: Senator Skevin and Dr. McGahn, I am Dr. Martin Zanna, Acting Director, Chronic Disease Services, New Jersey State Department of Health, and I appear here today in behalf of Dr. Joanne Finley, New Jersey State Commissioner of Health.

The New Jersey State Department of Health fully recognizes the need to enact new legislation to support and reinforce our capabilities of controlling cancer in New Jersey, a State known to have one of the highest over-all mortality rates for cancer in the nation, and is, therefore, pleased to be called upon to provide you with information to help you in your legislative efforts.

Let me begin by addressing the question of what we know about carcinogens in the human environment. A carcinogen by definition is a cancer-causing agent. Chemical carcinogens are really a subgroup of cancer-causing agents. There are many suspected chemical carcinogens for which we have insufficient evidence to be certain that they indeed cause cancer; NIOSH, the National Institute of Occupational Safety and Health, has published a list of over 12,000 toxic substances, many of which are felt to be potentially capable of inducing cancer. There are, in addition, a rather limited number of substances for which enough evidence has been collected and analyzed for OSHA to set standards for as known carcinogens. There are three such packages of OSHA standards: (1) the fourteen known (or probable) carcinogens. Rather than bore you by reading all fourteen. I will let you refer to your own copy where they are listed. (2) Asbestos. (3) Vinyl chloride. A listing of the fourteen known carcinogens is available through OSHA and is also listed in the document, "Controlling Cancer in New Jersey," published by the New Jersey State Department of Health in May of 1976.

Before a standard is set by OSHA, NIOSH submits criteria documents providing supportive evidence that a substance is indeed carcinogenic. These criteria documents take from 12 to 18 months to complete and cost several hundred thousand dollars. In order to establish causal relationship between a chemical substance and cancer, there has to be strong epidemiological evidence - this implies a statistical association between exposure to a substance and cancer - as well as confirmatory results from animal studies.

Recently, relatively quick screening tests have been developed using bacterial systems (mutagenesis testing) which assay a substance's carcinogenic potential. Because of the large number of toxic substances which we have little knowledge about and the fact that there are over 500 new chemicals introduced into industry yearly, it is

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envisioned that the screening tests will assume increasing importance in setting priorities for further investigating these potentially carcinogenic substances.

The question of knowing what technology is avilable to control possible carcinogenic emissions from industries, the efficacy of such pollution controls and their costs is really a question which should be directed to the Department of Environmental Protection. However, there are key roles which a properly supported Health Department can play to help the Department of Environmental Protection.

1. The laboratory of the State Department of Health is already equipped to do some toxicologic analyses. For example, it was able, in the unfortunate event of the recent Legionnaire's disease, to assay tissues for the presence of nickel. Nickel carbonyl is, incidentally, an industrial substance believed to be carcinogenic. The laboratory also does do testing in a contract arrangement for EPA. With an enhanced capability (toxicologic analysts and more equipment) the Health Department could serve the DEP and the public by telling them whether emissions are indeed hazardous enough to warrant "forceful" regulation.

But there also must be authority given to test internal environments. We still believe in the Health Department that the first magnitude of risk is to those exposed in the workplace. If something toxic is coming out the smokestacks, or is being discharged into the waste disposal systems, think what it must be like inside where people are exposed to these substances before any dilution, and for eight hours a day, year in and year out.

2. The epidemiologic studies the Department carries on,which, incidentally, require the establishment of a Cancer Registry to get us to those still alive for studies of causation, will best show DEP where to go. It does not appear to us to be cost effective to sample the general air or water. But where epidemiology points the way to a town, or a section of a city, or a population cluster with a high incidence of certain kinds of cancer, then it becomes socially responsible to pinpoint the source with monitoring techniques, and to control it with effective technology that will not stall the economy or deprive people of jobs.

We believe the interiors of plants, and the workers in these plants, must also be afforded the necessary protections, whether it be by proper ventilation and disposal systems, or by protective clothing or devices. These technologies do exist. They are reflected, where promulgated, in OSHA standards, and they are generally less costly than air pollution emission control devices.

I think a state could help speed up the process of developing the criteria documents and thereby contribute to the development of standards, be they State or Federal standards, by enacting legislation that would allow the State Health Department the right of entry into industries where a health hazard is suspected, to document exposures, conduct epidemiological studies to establish a causal relationship between a suspect carcinogenic substance and cancer, and to cooperate with NIOSH and OSHA in enforcing protective standards.

The worker is really a sentinel for the community at large in that he is the one that is exposed to the highest concentrations of known or suspected carcinogens since he works longest and in closest proximity to these agents. It is incumbent upon us to study the health effects of such exposures on the worker so that the results can be assessed and later applied to the general population. The same applies to improving on medical surveillance techniques with regard to cancer. They should be studied and initiated in high risk groups. Workers are captive groups and are high risk by nature of their exposure. But to really carry out such activities in any meaningful way, we will

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need new legislation allowing the State right of entry into industry.

Thus, the message I will close with is, let's establish ways for the State to get into the workplace to ensure both the health of the worker, as well as the community at large.

(The fourteen known, or probable, carcinogens not read by Dr. Zanna can be found on page 24.)

SENATOR SKEVIN: Dr. Zanna, thank you. Has your department discussed or thought about, short of shutting down plants completely, controlling the emission standards through the installation of an efficient control device? Can this be done?

DR. ZANNA: We really haven't discussed that. But it seems to me, since you are talking about what is going to get out into the ambient atmosphere, this would be a concern for DEP. I am not really sure that that would be a Health Department role.

SENATOR SKEVIN: Dr. McGahn.

SENATOR MC GAHN: With regard to the Chairman's statement about controlling emissions from industrial plants in New Jersey, is it your contention or the contention of the Department that air emissions in New Jersey alone are basically responsible for what is happening in New Jersey? Putting it another way, can standards set in New Jersey apply to contiguous states and is air flow from New York, Pennsylvania, Ohio and Michigan carrying with it various emissions into the State?

DR. ZANNA: I don't think we have to be so concerned about contamination of New Jersey air from such far sites as Michigan. But I really think we should concern ourselves with the air that is in close proximity to the plants where known carcinogens exist and we should sample and monitor the air that is immediately surrounding those plants.

SENATOR MC GAHN: Would you have any figure of the cost to effectively monitor the air in the neighborhood of the plants that you are referring to?

DR. ZANNA: I am sorry but I don't have that information. That is a good question.

SENATOR MC GAHN: Doctor, how does New Jersey rank in deaths as far as chronic respiratory ailments are concerned?

DR. ZANNA: I can only speak for respiratory cancer, in which New Jersey is among the top five.

SENATOR MC GAHN: You cannot speak as to emphysema or any other respiratory ailment.

DR. ZANNA: I don't have data for that.

SENATOR MC GAHN: Okay.

SENATOR SKEVIN: Dr. Zanna, on page 4, you indicate or suggest that the State Health Department be permitted by legislation the right of entry into industries where a health hazard is suspected. Do you have any authority under the present statute for entry into these industries for any other reason?

DR. ZANNA: Only in instances of imminent threat to health can we go in.

SENATOR SKEVIN: Could you speak into the microphone.

DR. ZANNA: Only under the broad powers of the Commissioner where there exists imminent danger to health. But, other than that, we really don't have right of entry.

SENATOR SKEVIN: Only if there is imminent danger to health?

DR. ZANNA: Yes.

SENATOR SKEVIN: Thank you, Doctor.

Dr. Glenn Paulson, Assistant Commissioner, New Jersey Department of Environmental protection.

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D R. G L E N N P A U L S O N: Chairman Skevin and Senator McGahn, my name is Glenn Paulson. I am the Assistant Commissioner for Science in the New Jersey Department of Environmental Protection. With me, to my right, is Dr. Peter Preuss, Special Assistant to Commissioner David Bardin, who works with me on our cancer program, within DEP.

Dr. Preuss has distributed to each of you a copy of some prepared remarks. I will read a slight variation of that statement, with some slightly different emphases. I have provided the recorder with a copy to ease her job as well.

Thank you for inviting DEP's continued participation in the efforts of your Commission. You have asked that DEP provide certain information on the identification of cancer-causing potential - carcinogenicity is the technical word for that - the process by which certain individual substances are designated as cancer-causing agents or carcinogens, and programs to control the human exposure to such agents.

Before I cover those points, let me say at the outset that we heartily agree that this nation, as a whole, which has poured extraordinary amounts of money into a search for cures for cancer, is long overdue in making a commensurate commitment to prevent cancer from occurring in the first place, even if the efforts ultimately point to the need for uncomfortable changes in life styles and business practices. That is not to say, however, that the efforts so far in prevention of cancer have been wasted. A brief review of some of the federal systems for testing substances suspected of causing cancer, the progress made with these tests, and what remains to be done, will provide a useful perspective for your deliberations.

First, on activities at the federal level, the National Cancer Institute, a branch of the National Institute of Health, is the major federal agency which tests substances for their ability to cause cancer. This work is based on biological assay techniques, usually abbreviated as "bioassays." To give you some idea of the magnitude of their effort, for the fiscal year ending a year ago June, the NCI bioassay program had about 540 chemicals under test. These assays required approximately 225,000 animals during the year, mainly such creatures as mice and the like. The chemicals being tested were of a wide variety, including drugs and pharmaceutical agents; chemicals used in industrial operation; pesticides, herbicides and other agricultural chemicals; food additives; chemical intermediates; and the like.

Such bioassays, as Dr. Zanna has pointed out, are expensive and time-consuming. Three years or longer may be needed to complete the bioassay on just one chemical. The cost typically is $100,000 or more. The results of such tests on a single substance are usually reported to the professionally-interested public in special NCI reports or in scientific journals.

We have an example here of one of the publications from the National Cancer Institute, their Carcinogenesis Program, Special Monograph, and we have attached as Appendix I to our statement some charts and other factual information that describe this bioassay program in some more detail.

Another federal agency has compiled this bioassay and other information into more readily usable lists. This agency, the National Institute of Occupational Safety and Health, annually publishes a list of toxic substances that Dr. Zanna referred to earlier. The 1975 list, which is in this volume here, contains over 15,000 substances. It is now out of print. The 1976 edition is due out later this fall and we will provide the Commission with a copy when it becomes available. A separate subfile or extract of this large list of toxic substances specifically covers known or suspected cancer-causing agents. We have a copy of that volume, the Suspected Carcinogen List, as well.

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Just for perspective, if there are 15,000 toxic substances, of which 1,500 are suspected of known cancer-causing agents, it is important to recall that at least one to two million chemical substances have been synthesized by man since the dawn of modern chemistry in the 1700's.

Finally, all of these compilations by federal agencies rely very extensively on the work and publications of the World Health Organization, which has put out a series of nine volumes which we have with us here, under the authority of the International Agency for Research on Cancer. This array of material that we are surrounded with here is only a small sampling of the technical information that DEP has gathered and compiled and which we use as our basic working tools literally every day.

Let me now turn to the testing for cancer-causing potential. At present, there are only a relatively few substances of classes of substances which are definitely known to cause cancer in humans: these are the so-called "known" - firmly "known" - human carcinogens. One such class is cigarette smoke. But even here, the exact chemical substance or, more likely, combination of substances that carry the cancer-causing potential in cigarette smoke have not yet been fully isolated and characterized. This lack of certainty, if you will, is typical for many other carcinogens.

There is, of course, one main reason for the fact that only such a relatively few substances are positively known to be human carcinogens. Such information does not come from tests planned and designed to be performed on human beings: such tests are morally and ethically unacceptable. Instead, information on these known carcinogens comes from epidemiological studies and statistical analyses of large human population groups.

For many of the known human carcinogens, our information comes from disease and death rates in small populations that have been exposed to relatively high levels of a certain substance for a relatively long period of time - generally in a work or occupational environment. Vinyl chloride, asbestos and beta-naphthylamine are three relatively well known examples of this. In other words, the process resulting in the identification of a firmly known human carcinogen is simultaneously both tragic and after the fact. Unfortunately, I expect other human carcinogens to be so identified in the future, as additional epidemiological studies are performed. To sum it up, in short, information on known human carcinogens is basically and unfortunately derived from human body counts.

The more extensive information available concerning the much larger group of substances which have been identified as potential or suspected human carcinogens is primarily derived from controlled experiments in other organisms - in laboratory animals, if you will. The amount of information available on any such suspected carcinogen varies greatly from one substance to another, due to variations in the number of different species used in the tests, the strains of animals used, and other factors. As a result, it is generally difficult to apply the published laboratory data on suspected cancer-causing agents to situations that we actually encounter in, for example, the air in certain locations in New Jersey.

Many of the scientific questions we are discussing today should really have been asked and answered a long time ago. Once a substance has found its way into the environment, it may be too late to wait for the definitive biological and environmental studies before taking necessary regulatory actions. This general phenomenon is true not only for cancer-causing agents, but for other toxic and hazardous pollutants in the environment: heavy metal contaminants, certain pesticides, and the like. It is a characteristic of environmental contamination and possible human health threats and threats to other organisms from traced contaminants, small amounts of contaminants, in one or another part of the environment.

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One need certainly is to continue the evaluation in animals and other organisms - bacteria and so on - of the thousands of chemicals in common use in our society which have not yet been evaluated for their cancer-causing potential. A second need is to see if the already known or suspected carcinogens have biological effects at the very low concentrations found in the environment, these concentrations being typically hundreds or even thousands of times lower than those used in laboratory experiments or found in industrial work places. In the absence of this kind of badly needed data, however, it seems to me that prudence dictates that our policy for the present must be based on two fundamental elements:

First, substances which have been clearly shown to be cancer-causing in animals must be assumedto pose a risk to any humans exposed to them; and, second, substances which have been shown to be cancer-causing in animals at high concentrations must be assumed to pose a risk to both animals and humans at even lower concentrations.

Let me now turn to activities to measure and minimize exposure to these kinds of agents. In spite of the uncertainties I have mentioned, both federal and state agencies already have a number of regulatory programs underway to reduce human exposure to these materials. In some cases, standards have already been set which limit the exposure of people to known and suspected cancer-causing agents. For example, the federal Food and Drug Administration is prohibited by statute, the so-called Delaney clause, from permitting any substance that has been shown to cause cancer in animals to be used as a food additive. Similarly, the Occupational Safety and Health Administration, as Dr. Zanna mentioned earlier, has promulgated extensive regulations for eliminating occupational exposures to a number of carcinogens.

These regulatory standards for human exposure, if you will, are based on work by the National Institute for Occupational Safety and Health. Each such carcinogen is the subject of a special monograph put out by the National Institute for Occupational Safety and Health, on a substance-by-substance basis. We brought along just for an example the summary documents for several of these known carcinogens.

The U. S. Environmental Protection Agency (EPA) has also proposed or promulgated a few standards limiting emissions of carcinogens into the air and water. Attached to my statement is a recent EPA publication that tabulates in summary form the EPA programs on a large number of chemical agents. That is the document appended to the statement you got. (See page 25 for the appendix to which Dr. Paulson refers.)

This listing of chemicals in this document in summary of EPA programs is not confined to cancer-causing agents, but other hazardous and toxic pollutants as well. Even perhaps more useful than this tabulation is an ordering of the NIOSH carcinogen list that EPA has developed that is based on a variety of special factors, particularly related to environmental exposure roots for these agents. This volume is titled, "An Ordering of the NIOSH Suspected Carcinogens List," and provides very important information, not only for federal use, but by the use of any state program, such as the one we are developing in New Jersey that is trying to reduce human exposure to these materials.

One very recent development in Congress may further reduce human exposure to such agents through the testing of chemicals before they are actually marketed, thus before humans come in extensive contact with them. This is the Toxic Substances Control Act, which has now passed both Houses of Congress. It requires, among other things, the premarket testing of certain substances, a concept which DEP fully supports. In addition, one part of this Act is of particular interest to us. An amendment introduced at Governor Byrne's suggestion by Senators Case and Williams in the Senate, and by Congressman Maguire in the House, authorizes a state demonstration program for toxic and hazardous materials,

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including carcinogens. Hopefully, this program will be funded in the federal fiscal year 1978, not the current year, but the next one, and will enable one or more states to obtain federal funding to develop and implement a program to complement EPA efforts in this same area. Needless to say, we think New Jersey is a highly-appropriate state to receive funds for developing this kind of a program and are already planning to apply for these funds, if President Ford signs this bill. At the House-Senate conference just yesterday afternoon, Congressman Rinaldo fortunately succeeded in reversing an earlier decision in the conference, and increased the authorization for this program from $1 million to $1.5 million.

Another area in which federal agencies have been active is the monitoring of trace contaminants in the environment. Information important to state regulatory programs has often been obtained through the aid of such federal monitoring activities. This is true not only for what we call the exotic pollutants, the trace contaminants, carcinogens and the like, but for standard well-recognized pollutants of the air, for example, sulphur dioxide, and the water as well. We are actively exploring with EPA ways in which monitoring and certain other efforts planned by both them and by us can be dovetailed to make the greatest and most effective use of the limited resources available.

Now I would like to turn to the third topic that you requested us to comment on, Senator Skevin: the philosophy and technology of pollution control. Our basic approach is straightforward - we must do our best to protect human health. It is aptly summarized in a recent publication dealing with the risk of cancer to humans, and I quote: "We are talking about people and not patients; if we are trying to preserve life, we should be looking at people before they are patients." This philosophy is being implemented already in DEP programs.

National standards already limit the emissions of three hazardous pollutants into the air: asbestos, beryllium and mercury. A fourth standard for vinyl chloride has been proposed,but not yet finally adopted. Currently, DEP enforces these standards in New Jersey and really most other states as well. Under the authority of the Clean Air Act, DEP has petitioned EPA for the right for us in New Jersey to enforce the standards for New Jersey.

During the past year, DEP adopted rules to control the emissions of volatile organic substances (hydrocarbons, petroleum chemical derivatives, if you will) from storage facilities - tank farms and the like. Some of these substances are known or suspected carcinogens; examples include benzene and certain of its derivatives, carbon tetrachloride, and others. DEP is continuing its efforts this year, using part of the appropriation already provided by the Legislature and approved by Governor Byrne for our department, to define necessary and appropriate additional regulatory programs to further reduce emissions of such substances into New Jersey's air.

On the 28th of this month, representatives from states in the entire northeastern United States will gather together with EPA to continue discussions on additional steps over this region to further control the emissions of volatile organic substances in a coordinated and coherent manner. This effort is necessary, since such hydrocarbon substances can travel long distances in the air. For the last several years now, DEP's Bureau of Air Pollution Control has been collaborating with Bell Labs on very extensive studies that show, for example, that the hydrocarbons emitted into the air in New Jersey can be transported in sometimes chemically-changed forms into more toxic forms, if you will, for long distances, not only over New York City but into Connecticut, and, in fact, even as far as Boston. We anticipate that work done upwind of us, which is underway by Bell Labs, may show the same kind of transport over scores or even hundreds of miles into New Jersey for some of these agents that we are concerned about.

Emissions to water are also controlled through a combination of federal and state

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programs and standards. In addition, and unlike the situation for air, standards also exist to regulate the quality of water as it is directly used by people, that is, for drinking.

Radiation is also a potential cancer-causing agent. DEP activities are aimed at reducing the exposure of New Jersey residents to man-made sources of radiation, including that from x-rays, from nuclear reactors, and from other sources.

Finally, many toxic and potentially cancer-causing substances find their way into the solid waste stream. DEP has worked to improve treatment and disposal methods in order to reduce the deleterious effects associated with environmentally unsound disposal methods for solid wastes as well as hazardous chemical wastes. These efforts will be continued and new regulatory programs developed in this area.

In addition to these and other current programs, DEP has expanded its activities to focus more specifically on the relation between cancer and the environment. These programs, which were discussed by Commissioner Bardin when he testified before you on June 11th, will supply us with additional pertinent and useful information, specifically related to the problem of cancer in New Jersey, and thus will be one of the main bases for future regulatory programs.

I would like to say a word about the broader activities of State government, if I may. DEP and the Health Department are not the only State agencies working on the cancer issue. In addition, the other State agencies that compose the Governor's Cabinet Committee on Cancer Control (DEP, the Health Department, the Departments of Labor and Industry, Agriculture and Higher Education) have already begun to plan, develop and implement portions of a coordinated and comprehensive program. As a result, for example, efforts to establish a cancer incidence registry, to begin both monitoring and the biological testing of samples from the environment, and to survey the use patterns of certain chemicals by New Jersey industries have already begun.

These cooperative and coordinated efforts characterize the multi-faceted approach that must be taken in order to grapple with this complex problem. The basic goal of all of these elements underway and being designed is directed toward the same end I mentioned earlier, namely, to reduce human exposure to cancer-causing agents. This goal is the common threat for all of these activities.

Nonetheless, and in all honesty, we currently lack much of the data necessary to adequately understand and evaluate the conditions in our State related to cancer and its incidence. Whether it be information on the incidence, not only death, but occurrence short of death, or the actual exposure of New Jersey residents to potential cancer-causing agents at their places of work, residence and play, these and other gaps in our knowledge must be filled as quickly as possible. One problem is that the resources currently available are simply not adequate for the job at hand. While the State has received some federal help and is looking aggressively for more, that will not add enough for even a fully-adequate beginning during this fiscal year. We look to this Commission to provide the legislative leadership in undertaking significant State programs for the sake of all our citizens and will be glad to help you further in any way that we can.

Thank you for your attention. Dr. Preuss and I will be pleased to try to answer any questions that you may have.

SENATOR SKEVIN: Thank you, Dr. Paulson.

In terms of the legislation that I proposed in my opening statement, Doctor, the bill listing by name those chemicals now established beyond a reasonable doubt to be carcinogens, setting the limits, etc., would you say that the list provided by OSHA of the 14 substances would be a start in that direction in terms of those chemicals that

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are dangerous to the people of our State?

DR. PAULSON: Let me ask Dr. Preuss to answer that question.

DR. PREUSS: Senator Skevin, the list provided by OSHA is insufficient in terms of potential exposures of residents in New Jersey, in that many of the substances that are on the OSHA list are either used very sparingly or almost not at all. They have either been phased out or are research chemicals. So one of the things that we have been attempting to do in our department, both for our carcinogen-use survey and for the monitoring effort that we are developing, is developing a list, such as you have described in essence, of those suspected or known carcinogens that we feel should be of concern to us in New Jersey.

SENATOR SKEVIN: Could you tell us the substances on that list, Dr. Preuss?

DR. PREUSS: I am sorry. It is a rather extensive list. I, unfortunately, don't have a copy with me. But I would be glad to provide you with a draft of that.

SENATOR SKEVIN: Would you provide us with that?

DR. PREUSS: Yes. I must say that this list is not yet completed: it is in the draft stage. But, of course, we will be glad to provide you with the draft.

SENATOR SKEVIN: Dr. Paulson or Dr. Preuss, if we are to control this pollution in our air, this dangerous condition, is there any method short of shutting down completely the culprits involved, the industry involved, to control the emission? And is the cost within reason?

DR. PAULSON: Yes, there are methods that are far short of shutting down a facility or even a whole industry that is using these materials. Even though we are at a primitive stage in controlling these agents as a society as a whole, there is already ample experience from, for example, the asbestos situation, that shows that simple changes in process, just the way in which materials are mixed or packaged or handled, can drastically reduce the exposure of humans to these agents. Process changes are one possible area; and while you can't predict for every carcinogen that you could go to a different process or even a different raw materials, that is one area particularly prone to provide success in this region.

It is really impossible to develop cost estimates across the board for these agents; reducing, for example, exposure due to x-rays involves different measurements, different machines, different standards, different calibrations, and thus different costs than reducing exposure of die makers to beta naphythylamine. So it is not possible to get an aggregate figure. It almost has to be a case-by-case basis. But the trend is already fairly clear that, when the need is clearly defined and when standards are properly thought through and soundly and fairly established, the ingenuity of the private sector can often find ways to skin a particular cat of this sort that could not be predicted in advance.

Dr. Preuss reminds me of one very simple one. One part of our regulation on hydrocarbon emissions that I mentioned earlier is the very simple step of requiring the tanks to be painted white - this was an idea originally of Commissioner Bardin, personally - the reason being that white reflects sunlight, thus keeping the tank relatively cooler, keeping the pressure of the gasses inside relatively lower and reducing the emissions substantially, just through the choice of the color of the tank.

SENATOR SKEVIN: You mentioned asbestos before. Is asbestos used in food processing, Dr. Paulson?

DR. PAULSON: Is asbestos used in food processing?

SENATOR SKEVIN: Yes.

DR. PAULSON: I can't think of any examples right offhand. It is used in processing of pharmaceutical agents.

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SENATOR SKEVIN: How about soda?

DR. PAULSON: It possibly may be. Asbestos filters have been used in the pharmaceutical industry. It is conceivable to me that they may be used in producing soda and beer, for example. I do not have any specific information on that point.

SENATOR SKEVIN: Dr. McGahn.

SENATOR MC GAHN: Dr. Paulson, in view of what has been happening in Philadelphia and the bad P.R. because of the so-called Legionnaires' Disease, I am concerned about two statements: One is that New Jersey is the cancer capital of America; and, number two, that 90 percent of all cancers are caused environmentally. I would like you to address yourself to both those statements, if you will.

DR. PAULSON: I assume, Dr. McGahn, that you have in mind the recent articles in the Star Ledger on New Jersey's rank relative to other states for a variety of causes of death.

SENATOR MC GAHN: No, not really. As a matter of fact, I only saw that for the first time this morning, but I do have others. If you will address yourself to this, maybe I can be a little more specific.

DR. PAULSON: We did go over this in some detail in the report that DEP published in May on this issue as to the Health Department. The basic fact remains that for one important segment of the human population, namely, white males, the cancer death rate from 1950 to 1969 was higher in New Jersey than for any other state in the country. If you look at other populations, females, for example, then you can get different relative conclusions. But, in general, New Jersey does have a substantially higher rate pretty much across the board for cancer as a whole. For certain individual cancers, there is no question that New Jersey or even single counties in New Jersey are highest in the country for that 20-year period. For other cancers, we are substantially below the national average. But overall we have to consider - and we consider - the incidence of cancer in New Jersey to be a serious problem.

Now, on the second point, I think we have to keep in mind that the use of 90 percent or 75 percent - it depends on what authority you go to - of all cancers being environmentally related, is based on the use of environment in the way that we use it in DEP, to include everything. We consider that environmental carcinogens are derived from exposure to cigarette smoke, to food agents, to standard pollution exposures in air, water, solid waste, and the like, to personal habits, life style, dietary matters, and the like. So it is a very, very broad concept of environment that lies behind that designation. The potential agents in distinction to environment really boil down to being genetic factors, factors beyond the control of the human individual or society, for that matter, and possibly viruses.

SENATOR MC GAHN: Thank you. I think you answered my question with the genetic and the viral background that we were talking about because I think that what I was really trying to say is that there are basically factors other than just air and water emissions that we are talking about that have a tremendous amount to do with this. We have not as yet, of course, - and this is the genetic background of it - talked about the differing immunological responses on the part of individuals; because,otherwise, if you had 25 men exposed for 20 years to exactly the same environmental carcinogen, then theoretically you should have 100 percent having the same effect at the end of those 20 years if you are going to get a causal relationship between the carcinogen and the individual. But this is basically not true because of genetic differences and varying immunological responses on the part of the individual. I think that is a point that very frankly should be made - the interdependence, if you will, and not only that of an

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environmental factor plus, as you say here, a virus, acting upon the nucleus, actually to throw the programming of that cell completely out of whack. And, instead of having normal reproductive growth, you have abnormal wild growth, which is basically cancer.

DR. PAULSON: Dr. McGahn, we could not agree with you more. We are very much aware of the multi-factorial approach, if you will, that probably results in cancer-causing agents. Some of the factors that let some asbestos workers be saved from cancer while others get it and die from it are known: many more are probably not yet known. We have to look, I think, at cancer - and Senator Maressa's aide made this point earlier -- cancer has a tragic personal dimension for the individual who has it, and for the family members and loved ones of that individual. It also has, if you will, a social dimension because of the multitude of sources of potential cancer-causing agents, not only through the air and water, but through radiation, through foodstuffs, etc., etc., etc., personal habits and so on. The treatment of the individual disease - therapy, x-ray treatment, surgery, and the like - is an important dimension and the dimension which has been emphasized by this society so far. The treatment of the causes, which is not a matter of individual action so much as it is a collective, social and thus political action, to reduce emissions, etc., is one that we are only now beginning to address.

SENATOR SKEVIN: Dr. Paulson, in a report submitted by Dr. Corielle of cancer and cancer research of New Jersey, Dr. Corielle indicates that the economic consequences in terms of medical expense and lost wages to the people involved in the State of New Jersey is in the area of somewhere between $750 million to a billion dollars a year. Would you say that is an accurate statistic?

DR. PAULSON: You might wish to direct this to the Health Department as well. Our estimates are that the direct cost for medical treatment, hospitalization, and the like, would be less than the figure that you cited.

SENATOR SKEVIN: $75 million to $100 million - that's the figure.

DR. PAULSON: That is right in accord with ours. Then when you add in the indirect dollar costs - there is no way, of course, to put a dollar on human suffering - the lost wages, the lost productivity, and the like, then the larger figure seems to us reasonable, perhaps even an underestimate.

SENATOR SKEVIN: So that the overall cost factor would be in the area of a billion dollars: is that what you are saying?

DR. PAULSON: Yes.

SENATOR SKEVIN: -- for the people of New Jersey and the economy?

DR. PAULSON: Yes.

SENATOR SKEVIN: Senator McGahn, do you have any further questions?

SENATOR MC GAHN: I think that is an extremely interesting statistic. And I certainly hope that the Senate takes cognizance of that when a particular bill attempting to set rates as far as hospitals are concerned comes up.

SENATOR SKEVIN: In our schedule of witnesses, we have Kenneth Gibson, Mayor of the City of Newark: and also Congressman Maguire. I understand, because of other commitments, they will have representatives submitting statements. So I would like to go to the next witness, Dr. Alfred Ellison, Deputy Director for Science of the United States Environmental Protection Agency. Then we will come back to the statements to be submitted by Mayor Gibson's and Congressman Maguire's representatives.

Dr. Ellison.

D R. A L F R E D E L L I S O N: Thank you very much, Senator Skevin.

I am Dr. Alfred Ellison. I am Deputy Director of the Environmental Sciences Research Laboratory of EPA, which is located in Research Triangle Park, North Carolina.

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Sitting with me is Dr. Robert Mason from EPA's Regional Office in New York City. We have been working closely with them in our work in New Jersey.

I am sorry I do not have prepared comments. I would like to make a few from notes that I have just made, and then I will be happy to answer any questions that you may have.

Our Environmental Sciences Research Laboratory conducts research related to air pollution in the fields of chemistry, physics and meteorology. We are interested in ways of measuring air pollution and we also study the phenomenon that was discussed earlier: namely, the transport of air pollutants from the source, through the atmosphere, to whatever its final fate is to be.

In our research on developing measurement methods for air pollution,up until a few years ago, we were mainly concerned with the air pollutants for which National Ambiant Air Quality Standards have been promulgated. These are, as many of you may know, total suspended particulates - sulfur dioxide, nitrogen dioxide, hydrocarbons, oxidants, and carbon monoxide. However, when we h ad reached the point when our research in that area was essentially complete, we turned our attention to some of the specific materials that get into the air, such as the materials we are talking about today. But our work, I would like to emphasize, is mainly to provide the tools whereby problems of this sort can be studied. However, in the process of developing these tools, we do have to go into the field and make measurements and see, indeed, if these measurement techniques will actually work in the real atmosphere, as opposed to the laboratory atmosphere where they were developed.

It was in the course of this type of a program that we did come to New Jersey and measure or collect samples which were subsequently analyzed to see what materials were present and, at the same time, to evaluate our measurement methodology. I should also say that New Jersey is not the only place we have been. We went first to Baltimore, Maryland. We have been to Houston, Texas. We have been to Los Angeles. All of these places have a situation very similar to the one, on the work we have done so far, that exists in New Jersey.

What we have done in New Jersey -- and we came here, as we went to the other places, because there is heavy industry here. And,in the vicinity of heavy industry, you do find higher levels of air pollutants of this type than you will out in the country. This is certainly no surprise. Of course, in evaluating measurement methods, we want to evaluate them where there are things to be measured, not where things to be measured do not exist. So this is why we came to New Jersey. We didn't come because of the Cancer Institute's report on the incidence of cancer in various places. It really had nothing to do with that. However, to the extent that the results of our work can help this Commission or help the New Jersey Department of Environmental Protection to do its job, we are happy to participate.

In New Jersey, we made measurements on a couple of days in March. We made a few more measurements on a few days in late June and early July. We have not made very many measurements. The information we have is largely qualitative. In the measurement technique that is used, one collects the sample and does first a qualitative analysis to find out what is present. After we see what is present, we then have to apply calibration procedures to determine how much is present of any particular material. So the qualitative information comes first and after some further work - again selecting the more important materials for this quantitative work - we do get quantitative information on these materials. As of now, we have very little quantitative information. However, a final report on this work is due out in a few weeks. This will then quantify the information that we have. In other words, it will say what was the concentration of these different

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materials in the air at the time that the samples were collected. But, again, this is very preliminary. The data applied to only a few days and, before one could really make a judgment as to the significance of these results with respect to the exposure of people in these areas, one would have to do quite a bit more work.

I should also mention ---

SENATOR SKEVIN: How much more work, Dr. Ellison, before we know the exact danger involved or the extent of the danger?

DR. ELLISON: Well, of course, the danger relates to the health information. I really can't comment on how long it will take to establish what is called a dose-response relationship: in other words, before a health research program could say that if a person is exposed for so many years at a certain level or at an average level of given material, their chances of getting cancer are so much.

SENATOR SKEVIN: But we know there are dangerous substances in the air in New Jersey at this present time: isn't that correct, sir?

DR. ELLISON: Yes, I think that is correct. But, again I would say, this same situation is found in other places as well.

SENATOR SKEVIN: They have the same dangerous substances in other places also?

DR. ELLISON: Yes. But again, it is hard to say how dangerous.

SENATOR SKEVIN: It is dangerous though for the people here in our State with these substances in the air: isn't that correct, sir? Your only argument here is as to how dangerous: is that so?

DR. ELLISON: Yes, I think that is true. Also I should say that I am a physical scientist, not a health scientist, and it really isn't appropriate for me to comment on the health consequences of these materials. But the ones that have been identified as carcinogens and are here -certainly this is cause for great concern and something should be done about it.

SENATOR SKEVIN: And what is the federal government doing about it, Doctor?

DR. ELLISON: Well, I can give you an example.

SENATOR SKEVIN: Are there any controls for these substances by the federal government in our area?

DR. ELLISON: There is a provision in the Clean Air Act for controlling any hazardous air pollutant. If this is a material which clearly involves a health hazard, there is a provision in the Clean Air Act of 1970 to control those emissions. This is the authority under which asbestos, beryllium and mercury are under control and under which the proposed control of vinyl chloride will be carried out.

SENATOR SKEVIN: But we are a long way from mounting any meaningful control measures in this area, aren't we, Doctor. Isn't that so?

DR. ELLISON: Well, it is hard to say. We are a long way, but the long way is not because we don't have the authority: it is because we don't have the information that the legislation requires that we have before we can take action. But once the information is there, there is no doubt but that the authority is there.

SENATOR SKEVIN: But we know it is dangerous at this moment?

DR. ELLISON: There is great cause for concern.

SENATOR SKEVIN: There is great cause for concern, isn't there, Doctor?

DR. ELLISON: Yes, sir.

I would just like to say another word about where do we go from here. We have had meetings with Dr. Paulson and Dr. Preuss, and Dr. Mason from our regional office is involved. We do plan to continue to coordinate our efforts with your efforts to add to the information base we have, so that the necessary action can be taken because you have the information you need.

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I could cite an example. You mentioned earlier about control and how does one go about controlling. Actually, the first activity we were involved in similar to this one was a case in Baltimore, Maryland, where some independent researchers said that some N-nitrosamines were present in the air in the vicinity of a plant there. We, using the same kind of technology and with the same people, went to that site and made measurements and indeed confirmed the fact that this material was present in the air. This was a chemical complex which did manufacture a chemical using N-nitrosamine as an intermediate in this manufacture. The company didn't know that this material was escaping from their operation. But, once they did know, they took independent action to monitor their own activities and they did find places throughout the plant where leaks were occurring and they were able to eliminate many of those leaks so that the problem was abated.

So, when you have a case where you can identify the source, it doesn't always follow that you have to shut the plant down or you have a very expensive control program needed. It may be a relatively simple one.

SENATOR SKEVIN: Doctor, in terms of the areas that are examined by the federal government, we have heard mentioned Paterson; Clifton; Fords, New Jersey; and in the area of Hudson County, perhaps Hoboken. Are there other areas that are being tested by the federal authorities in New Jersey?

DR. ELLISON: Our work has been limited to the vicinity around the Kim-Buc Solid Waste Disposal landfill. We have concentrated there mostly because we felt that would be a good place to evaluate our measurement procedures. But I am sure that the local EPA regional office and the Department of Environmental Protection are planning measurement work in other parts of New Jersey.

SENATOR SKEVIN: Do you have any further comments?

DR. ELLISON: No, sir.

SENATOR SKEVIN: Dr. McGahn.

SENATOR MC GAHN: I have no questions.

SENATOR SKEVIN: Thank you, Dr. Ellison.

Miss Vivien Li, a representative from Mayor Kenneth Gibson's Office.

V I V I E N L I: Senator Skevin and Senator McGahn, I am Vivien Li, Project Director for the Air Quality Office in the City of Newark.

Unfortunately, Mayor Gibson could not make it this morning and I have been asked to read his statement and to answer any questions.

(Reading Mayor Gibson's statement)

Thank you for permitting me an opportunity to testify before you today. As Mayor of the largest and one of the most industrialized cities in the State, I am concerned about the high incidence of cancer experienced by our people.

If the latest health reports are accurate, then we in New Jersey have much reason for concern. Our state has been shown to have the highest levels in the country for three specific categories of cancer. Moreover, Essex has been ranked as one of the top three New Jersey counties with the highest cancer rate, although Bergen has the highest number of actual deaths resulting from incidence of cancer.

In reviewing the death rates in Newark, we have found that between the years 1972 to '75, cancer-related deaths have accounted for one-sixth of all health deaths of Newark residents. These figures do not even reflect the statistics of those workers who are employed in Newark industries, but who do not reside within the City.

Many of these deaths are directly attributable to environmental factors. Nationally, environmental factors account for 75-90 percent of all cancer incidence.

In a federal Environmental Protection Agency (EPA) report released last month, recent measurements of ambient air quality in urban areas clearly demonstrate that air quality has not improved for many of our urban residents. Within Newark, for instance, the report cited readings of up to 300 micrograms per cubic meter of benzene and 37 micrograms per cubic meter of chloroform in the industrial portion of the City.

Although press accounts have indicated that one chemical plant had been responsible for these levels, the federal report was less explicit. Rather, the usage of various plants within the report was designed to serve more as a locational tool than as an actual enforcement designation. The readings, therefore, reflect the air quality levels of the area, and not necessarily those levels created by a single industry.

It has become imperative that we put those contributors of this health menace on notice. Accordingly, I am requesting today that the state Department of Environmental Protection and the federal EPA undertake a survey of pollutors in those areas with high pollution readings and that all such industries be placed on a compliance schedule immediately. If, at the end of one and a half years, levels are not reduced to acceptable health levels, then DEP should move to cease operations at non-conforming plants until proper adjustments are made. For, without a clearly defined timetable, the health and welfare of our residents and workers will only be further jeopardized.

But, even if these efforts are to be meaningful, preventive, as opposed to remedial approaches, must be taken. On the national level, both the proposed Toxic Substance Bill and the EPA interim guidelines for suspected carcinogens (Federal Register, Vol. 41, No. 102, May 25, 1976, pp. 21402-5), when implemented, will provide a sound regulatory framework for control of questionable chemicals.

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As presently adopted by both houses, the proposed Toxic Substance Control Act would require pre-market testing of all new chemicals suspected of contributing an "unreasonable risk to health or the environment." The EPA guidelines,moreover, outline specific standards for evaluating the health and economic impacts of restricted distribution of chemicals.

With joint house action scheduled for the Toxic Substances Control Act, it now appears that final action will occur prior to recess of this Congressional session. With the additional likelihood that the Maguire amendment will be adopted during joint conference deliberations, states like New Jersey can expect grants of up to 75% funding for the establishment and operation of programs to prevent or eliminate undesired chemical substances.

As one of its first priorities, therefore, the state should begin to identify programs which will complement the federal thrust and which will be eligible for federal support. It would appear that many of these programs are already identified within Executive Order #40, issued by the Governor in May of this year. It represents an initial, comprehensive attempt at monitoring and evaluation of carcinogenic sources by all state agencies.

For its part, the State Legislature must assume several responsibilities if a serious effort at abatement is to occur. First, appropriations must be made available both for implementation of Executive Order No. 40 as well as for matching federal funds from the Toxic Substances Act. Earlier this year, although the Department of

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Environmental Protection had requested $1.8 million for control of carcinogenic substances, the Legislature approved a minimal $500,000. If our efforts are to be effective, and if we are to be able to match federal funds, then substantial increases in appropriations will be necessary.

Over the next five years, $5 million dollars should be made available for control of carcinogenic substances. These funds should be used for the following: establishment of a comprehensive monitoring and surveillance alert system in all industrialized areas of the state; establishment of a source inventory; coordination and development of cancer treatment and rehabilitation facilities; and establishment of training and education programs for the prevention of cancer.

On another matter, this Committee should initiate legislation to monitor carcenogenic substances emitted during industrial processes or created due to synergistic reactions. Specifically, legislation requiring industries to submit chemical discharge permits should be developed. Such legislation should be similar to the federal water discharge permit system, requiring information on the amount of pollutants-both air and water-discharged into the environment; possible health problems from such pollutants; a delineation of potential synergistic effects; the time schedule for compliance with environmental standards; and the development of a continual reporting system. Such legislation should be developed in a manner consistent with existing reporting and permit systems, and not result in duplication of information.

Moreover, in reviewing such permits, state agencies must evaluate each based upon regional considerations. Permits should not be viewed in isolation, but rather, as part of a network of discharges for a given area. Thus, a permit for discharge in Newark should be evaluated and scrutinized in a different fashion than one submitted for Burlington County. Attention should be given to preventing new sources from locating in pristine areas such as the latter area, while sources in urbanized areas should be given support to upgrade existing equipment.

Even in the quest for goals as desirable as clean air, however, consideration for the economic viability of our urban areas must be given. Undoubtedly, urbanized areas with older, more obsolete plants will initially be most affected by any regulations adopted. In order to provide these plants with incentives to remain in the urban area as well as to continue operations, low interest bonds available under the Industrial Pollution Financing Act administered by the Economic Development Authority should be made available to these plants first. Otherwise, pollution control, without due economic consideration, will most adversely affect those it was initially meant to save.

It is my hope that with the implementation of the above measures, significant improvements to the quality of life in our state will occur. I look forward to coming back to this Committee personally next year and reporting on that change. Thank you.

(End of Mayor Gibson's statement)

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SENATOR SKEVIN: Thank you.

Senator McGahn.

SENATOR MC GAHN: No questions.

SENATOR SKEVIN: There are no questions. Thank you.

Congressman Maguire's representative.

C H R I S B U R D I C K: Chairman Skevin and Senator McGahn, my name is Chris Burdick. I am a Legislative Assistant to Congressman Maguire. I know the Congressman had wanted to be here. He is in Washington because of the possibility of the Clean Air Act being raised today, in which Mr. Maguire has been very active. So I will read the statement of Congressman Maguire.

(Reading Congressman Maguire's statement)

I want to thank the Special Commission on the Incidence of Cancer, and you, Chairman Skevin, for inviting me here today to testify.

Last May, I chaired a hearing of the House Commerce Investigations Subcommittee in Newark. Because the National Institute of Health had identified New Jersey as having one of the nation's highest cancer rates, there was a certain urgency to our hearing on the environmental causes of cancer.

It is now generally accepted that perhaps as much as 90 per cent of the incidence of cancer is environmentally caused. Therefore, we must look around us for the cause.

At our May hearing, the Subcommittee heard testimony that 144 workers had died of cancer at a du Pont plant in Belle, West Virginia. Belle and Baltimore, Maryland, had been identified earlier by the U. S. Environmental Protection Agency as two areas of the country where there is a high level of nitrosamines in the air. And EPA has acknowledge that "as a family of carcinogens, nitrosamines have no equals."

Dr. David Fine, who has been testing for nitrosamines for EPA and other government agencies, said he believed New Jersey's air is also suspect. Nitrosamines are a byproduct of the combination of nitrous oxides (NOx) from automobile exhaust and amines, which are present in the air and in the human body. New Jersey, as a major corridor state, has the heaviest concentration of cars in the country, so it is logical to expect nitrosamine pollution here too.

At our hearing, Joseph Padgett, director of strategies and air standards for EPA, gave us a commitment to conduct further testing for nitrosamines here in New Jersey. Some of those tests have already been done and they indicate that these dangerous hazardous compounds are in our soil and water. The tests of air, however, were done under adverse conditions during a short period following rain, not during a temperature inversion, when automobile pollutants could be expected to build up and combine with airborne amines from chemical plants and sewage treatment plants.

Because these tests were both limited and inconclusive, it is essential that EPA expand its testing. EPA must make New Jersey as the number one cancer state its first priority for testing for carcinogens, such as nitrosamines, and others, such as were found near industrial plants in Clifton, Hoboken, Newark, Passaic, and Paterson.

Expanding its cancer study, the Investigations Subcommittee obtained voluntarily the 20-year cancer registry maintained by the E. I. dupont de Nemours Company, which owns the Belle Plant, as well as eight plants in New Jersey. These plants are the Chambers Works in Deepwater, a chromate plant in Newark, the Repauno Works at Gibbstown, two plants at Parlin, a plant in Pompton Lakes, the Graselli Works in Linden, and the Carney's Point Works in Salem County.

On a negotiated schedule, duPont has turned over to the subcommittee hundreds of pages of documents, including internal company reports and a computer printout of each

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incident of cancer among its employees. The Subcommittee has sent this information to several government cancer experts and outside specialists for evaluation. We expect to issue a report in the next couple of weeks.

This may tell us more about the cancer situation at plants engaged in different types of manufacture and it may tell us something about the types of records that chemical companies should maintain and should furnish to the EPA on a regular basis.

Industrial emissions are but one piece of the New Jersey cancer problem. Auto emissions are the other. Together with my colleague, California's Henry Waxman, I've been fighting for the Clean Air Act to prevent the Ford Administration and its allies in Congress from postponing indefinitely any controls on nitrous oxides.

Because of the chemistry of nitrosamines, it is easier to limit their formation by cutting NOx emissions than by eliminating the amines from the atmosphere.

Beyond your trying to control auto and industrial emissions, we must also begin looking at another potential cause of a future increased cancer rate in this country - herbicides.

Last week, at a Washington Press conference, I called upon EPA Administrator Ruasell Train to ban immediately four pesticides, one of which - Eli Lilly's Treflan - is in widespread use.

Tests revealed a week ago at the American Cancer Society meeting in San Francisco indicate that these herbicides have up to 640,000 times the level of nitrosamines found over the air in Baltimore. Because there is no known safe level of nitrosamines, this represents an imminent hazard to the health of those who work with these herbicides. In addition, the nitrosamines in the herbicides may work their way through the food chain into the bodies of those who eat products grown in fields using the spray.

The EPA Administrator should use his power under the Federal Environmental Pesticide Act to immediately withdraw these chemicals from the market. EPA should also begin at once testing the entire spectrum of pesticides to see if it obtains similar results.

I will ask the EPA what it intends to do about the herbicides at a subcommittee hearing planned in Washington September 20th. I will not tolerate any bureaucratic buck-passing or wheelspinning. Lives are at stake.

I was pleased when the House passed the Toxic Substances Control Act on August 23rd, giving the federal government new authority to control hazardous chemicals. I successfully amended the bill to include a provision granting EPA the authority to fund up to 75 percent of the effort of states such as New Jersey to run their own environmental carcinogen control programs.

I will lead the fight in Congress next session to give EPA the funds necessary to help states which develop effective programs to limit the spread of carcinogens.

Finally, let me say that I am glad you, Mr. Chairman, are holding this hearing today. If the public is made aware that cancer is environmentally caused, this will create the critical political support for the tough kinds of action we in Washington and you in Trenton will have to take to prevent a potential epidemic 20 or more years from now.

(End of Congressman Maguire's statement)

Thank you, Mr. Chairman.

SENATOR SKEVIN: Thank you, Chris. It was a fine statement from Congressman Maguire. There are no questions. I just want to mention that we recognize the fine work of Congressman Maguire and also Congressman Minish on a federal level and their interest in New Jersey's problems. So express our appreciation to the Congressman for us.

Thank you very much.

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This will conclude our hearing today on the high incidence of cancer in New Jersey, and we will have further hearings to be announced in the near future. Thank you.

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The fourteen carcinogens contained in the written statement of Dr. Martin Zanna, but not read, are as follows:

(1) the fourteen known (or probable) carcinogens including 4-Nitrobiphenyl; Alpha Naphthylamine; 4,4 Methylene bis (2-Chloroaniline); Methyl chloromethyl ether; 3,3-Dichlorobenzidine; Bis-chloromethyl ether; Beta Naphythylamine; Benzidine; 4-Amino dinhenyl; Ethyleneimine; Beta Propiolactone; 2-Acetylaminofluorene; 4-Dimethylaminoazobenzene; N-Nitrosodimethylamine

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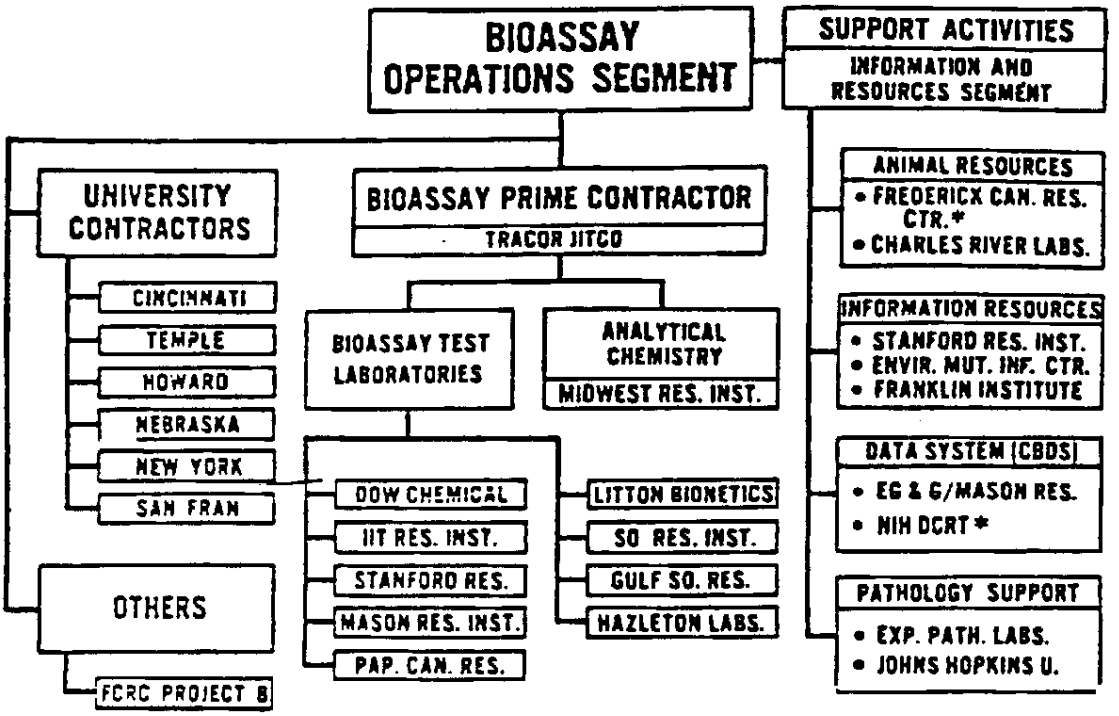


Figure 1. Organization of Bioassay Operations Segment

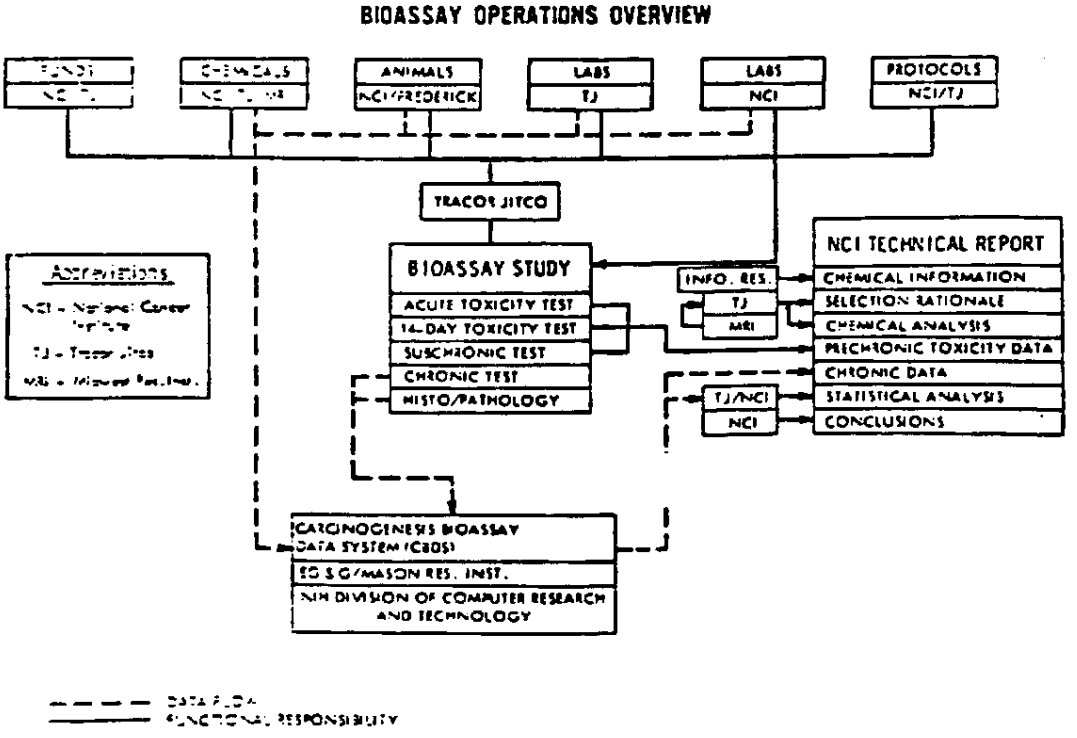


Figure 2. Review of Bioassay Operations Indicating Responsibilities and Flow of Research Data to CBDS and Technical Report Preparation

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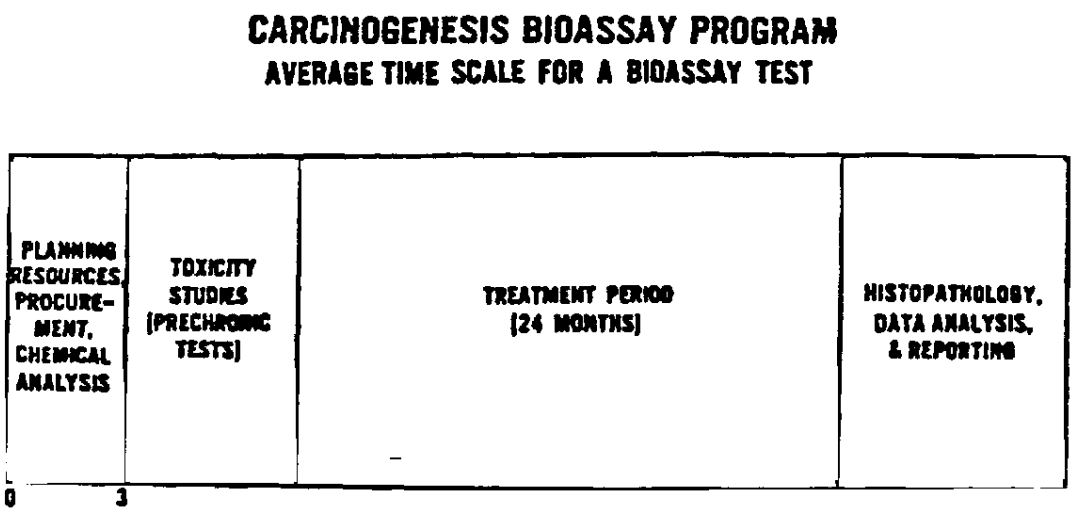


Figure 3. Average Time Requirements to Complete Each Phase of a Carcinogenesis Bioassay Test

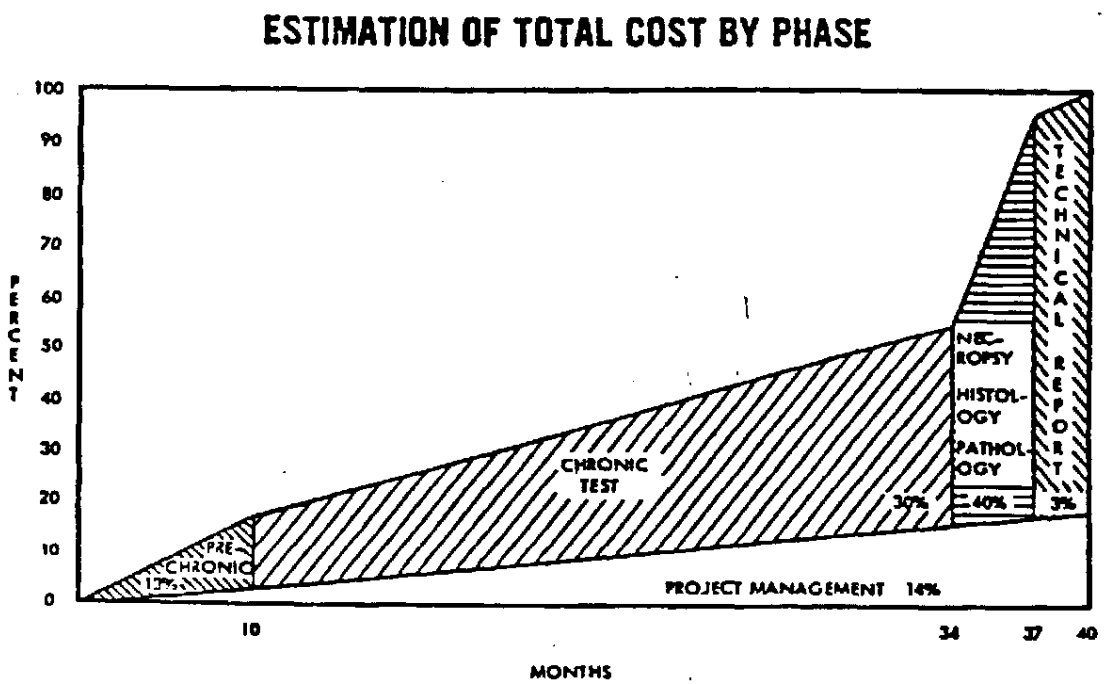


Figure 4. Average Percentage of Total Cost for Various Phases Involved in a Carcinogenesis Bioassay Test

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Appendix 10.

P U B L I C H E A R I N G

before

SENATE COMMISSION ON THE INCIDENCE OF CANCER IN NEW JERSEY

Held:
October 1, 1976
Senate Chamber
State House
Trenton, New Jersey

MEMBERS OF COMMISSION PRESENT:

Senator John M. Skevin (Chairman)
Senator Joseph A. Maressa

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Joseph Katz Tobacco Tax Council	14, 30, 31 & 32
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Diane T. Graves Conservation Chairman New Jersey Chapter Sierra Club	24, 33 & 35

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SENATOR JOHN M. SKEVIN (Chairman): Good morning. This is the third session of the Senate Commission on the Incidence of Cancer in New Jersey.

My name is John Skevin, State Senator from the 38th District and Chairman of this Commission. On my right is Senator Joseph Maressa from Camden County. Senator Joseph McGahn from Atlantic County is on his way. Senator Dumont has a medical problem this morning. And Senator Parker has other commitments.

We have scheduled for today a number of witnesses from industry - the chemical industry and the tobacco industry - and also representatives from the environmental groups.

I would like to make a few opening remarks.

Today we will be hearing from representatives of industry. Before we begin, I want to make it perfectly clear that I in no way consider these representatives to be "hostile witnesses." I have said all along, and I shall continue to believe, all reasonable people, whether speaking for industry, government, science or some other special interest group, share a common commitment to the eradication of environmental cancer. Despite this agreement pertaining to our goal, we must expect there will be differences of opinion as to how that goal is to be achieved. Let me indicate for the record what may seem to be some prejudices of mine.

First, let me emphasize that I am prejudiced in favor of people over statistics. By that I mean, the impact of a single death from what may well be a man-made killer far outweighs, in my mind, any dollars and cents value or any other economic yardstick we can apply to the cost of conquering cancer. And here in New Jersey, we are not dealing with the impact of a single death, but with more than 14,000 deaths each year.

Let me also state another prejudice, and that is simply, in my mind, chemicals are not people: and, therefore, are not entitled to human rights. In our society, we have based our system of justice, and rightly so, on the premise that a man is to be considered innocent until proven guilty. However, the logic of claiming that this same benefit of the doubt should be extended to such things as chemicals escapes me. It seems to me, in this instance, the process should be reversed - that before anyone is allowed to add anything to our food, our air, our water or our soil, they should be compelled to first prove beyond any doubt that the additive is safe.

A final point on which I am admittedly opinionated is anyone questioning the government's right, including that of the State government, to regulate such matters. Let me quote briefly, not from the United States Constitution, but from our State Constitution, Article I, headed "Rights and Privileges":

"All persons are by nature free and independent, and have certain unalienable rights, among which are those of enjoying and defending life and liberty . . . of acquiring, possessing and protecting property, and of pursuing and obtaining safety and happiness."

I submit that what we are attempting to accomplish at these hearings comes under the defense of our lives and the pursuit of safety and happiness, and that the real question involved is not by what right do the people propose to regulate materials which are emitted into the air, but by what right does anyone contaminate that air in the first place, thus threatening our safety and robbing many grief-stricken families of their inherent right to happiness?

While it may be argued that the constitutional right to possess and protect property could be cited by industry to support its position in the matters before us, I would submit that the real property in question, the property which we are seeking to protect, is the very air we breathe - and that belongs to the people and not to any special group.

As I conceded when I began these remarks, the opinions just expressed may have a bearing on my reaction to some of the points I expect to be raised here today. However,

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I believe those who are about to testify must also admit to a certain bias and degree of opinionation. But if we all strive to keep our minds as open as possible, if we listen to each other with mutual respect, I am confident we can today take a step forward toward the achievement of our mutual goal, the protection of the welfare and lives of the people of the State of New Jersey.

Our first witness will be Mr. Chris Hansen, Vice Chairman of the Chemical Industry Council.

C H R I S A. H A N S E N, J R.: Senator Skevin and members of the Commission, I am Chris Hansen ---

SENATOR SKEVIN: Excuse me, Mr. Hansen. Do you have prepared remarks?

MR. HANSEN: Yes, I do.

SENATOR SKEVIN: Could we have copies of it so we can follow it?

MR. HANSEN: Would you like it following my remarks, or now?

SENATOR SKEVIN: I would like it now.

MR. HANSEN: Senator Skevin and members of the Commission, I am Chris Hansen and I am here today to represent the Chemical-Industry Council of New Jersey. I am presently First Vice Chairman of the CIC and Chairman of its Legislation Committee. I am a chemical engineer by training and I have worked in the chemical industry for over 20 years, and I am most pleased to be able to appear before this group on behalf of that industry. I am here to present to you the New Jersey Chemical Industry Council's views regarding the allegations that the chemical industry, and/or chemicals are a major factor in New Jersey's high rate of cancer, supposedly the highest in the nation.

The chemical industry would like to reiterate its interest and concern with all aspects of job safety and the safety of surrounding communities. We certainly agree with Senator Skevin: we are not here as the opposition. We are here because we want to be good citizens and we want to help. The chemical industry pledges its support to this Commission and other responsible groups or agencies. We want to determine, probably more than anyone, whether we have a problem. At this point, we are not aware of any problems for which remedies have not already been set in motion. As new problems are found, rest assured that the chemical industry will take prompt action.

We believe that working conditions are adequately controlled and do not present any problems with respect to promoting the incidence of cancer among chemical workers. There are now about 15 known chemical carcinogens. Prompt, effective action was taken by industry in cooperation with government in each case and these situations are now under control. However, we strongly support the idea of additional study to insure that there are no factors unknown to us. You may be interested to know that the Manufacturing Chemists' Association is conducting studies costing over $5 million on various chemicals to insure that adequate safeguards are being employed.

The chemical industry employs about 120,000 people in New Jersey. The industry's most important asset is its people and we want to protect them just as you do. The chemical industry has a fine safety record, among the best in American industry. Chemical workers are healthier than the general populace. They have longer life expectancies, and lower rates of cancer than the general population. This fact indicates that a good job is being done to protect chemical workers.

We believe that emissions from chemical industry operations are adequately controlled. Each vent and all effluents from each plant must have permits from either the State or the federal government, or both. We are puzzled by some of the test results obtained recently by the EPA which showed the presence of many different chemicals in the air. Based on what we know, the results don't seem to make a lot of sense. There are

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probably some errors and the concentrations may be extremely minute and of no concern. So, the first order of business should be to get quantitative, accurate data so we will all know what we are dealing with. We don't think the chemical industry has any uncontrolled or unknown emissions which might be causing the problem. However, there are many different sources of emissions: from cars, from planes, from power plants, vaporization, painting, and on and on. These could be causing a problem since New Jersey is a highly-urbanized state. Further, the material could be migrating into New Jersey. In any event, this is a good example of a problem where we need to have prompt, cooperative effort among government, industry, and the scientific community.

Senator, we live here too: our families are here: we have the same concern as you and the rest of the people in New Jersey about the environment in which we live. Again, we want to help and cooperate.

It is highly questionable whether New Jersey does have the highest cancer rate in the United States. Please look at Table I. These are the most recent, unadjusted data available to us and come from the U.S. Department of Health, Education, and Welfare. As you can see, New Jersey is not the highest in the U.S. and even ranks low with respect to neighboring states. If these data were age and population density adjusted, New Jersey's relative position would probably be improved. Data presented earlier showing New Jersey to have the highest cancer rate was only for white males. Furthermore, by simply drawing a line across the chart, which is attached, for the United States average, it is readily apparent that, with the exception of Louisiana, all of the 15 states above the line are highly urbanized, industrialized states. There is no state in the northeast below the line except Vermont, which is extremely close to the average for the United States. Using a standard urbanization correction of about 10 to 12 percent, New Jersey's rate is reduced to 181 for white males, only slightly above the national average of 174. However, there is no question that New Jersey's cancer rate tends to be high. Some specific cancers are the highest without question. We need to understand why.

We think that comprehensive, rational studies should be made based on extensive and current data, using the most sophisticated procedures and talented people available. We think the time has come to end scare tactics. We think the "Cancer Alley" label is a "bum rap," so to speak, against our State. First, we don't think it is true. There is just as much data, maybe more, that say, overall, New Jersey is among the healthiest places to live. Secondly, we think it is bad politics and bad business to demean a state which has so much to offer. Let's emphasize the positive. New Jersey is a good place to live. I, for one, like it and am proud to be here and a part of the State.

Most importantly, we think the time has come for intelligent action. Industry is only a small part of the problem. Of the 27,000 cancer cases in New Jersey this year, reliable authorities estimate that only 500 will be industry related. These same authorities estimate that industrially-caused cancer in New Jersey can be counted on two hands. Obviously, industry wants to stop every one. However, I think we need to work on the big problem, that is, the 26,500 cases, with the highest priority.

I understand, Senator Skevin, you are establishing a scientific advisory committee to the Commission. I commend you for this. These people will certainly place priorities properly and will advise you as to what programs should be undertaken to deal with cancer in New Jersey. The New Jersey Cancer Institute appears to be a very good vehicle to carry out the many facets needed to deal comprehensively with cancer in New Jersey. They are off to a good start. They have some talented, experienced, and dedicated people already working. The CIC recommends that you consider helping fund what appears to be an organization that can deal with New Jersey's problem effectively.

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We are sure that you are aware that a Federal Toxic Substances Act has been accepted by Congress and is awaiting the President's signature. The chemical industry has supported this Act. This legislation will provide additional testing and control of the manufacture and distribution of suspected and known dangerous substances. Specifically in the area of carcinogens, Section 4 F of the bill will require further testing of any substances that have the potential to induce cancer in humans.

In conclusion, the CIC would like to emphasize that the number of cancers caused by chemical manufacturing is very small. Although even one case is regrettable, the contribution of the chemical industry to the overall cancer rate in New Jersey is minute. On the other side of the coin, I would like to point out that chemicals have added in great measure to the quality of life and,even more important, chemicals have been extremely valuable in helping reduce, cure, and eliminate many crippling and fatal diseases, including cancer. On balance, I think it fair to say that chemicals and the chemical industry have saved far more suffering, and many, many more lives than it has harmed.

In summary, Senator, the CIC will cooperate, will help gather facts, will help analyze the facts to define problem areas, will act aggressively and vigorously to eliminate any problems, and supports funding of sound programs to accomplish the foregoing. Thank you, sir.

(See pages 28 and 29 for charts attached to Mr. Hansen's written statement.)

SENATOR SKEVIN: Mr. Hansen, you presented your point of view very well. As I understand it, in the overall picture, there really is no problem as far as chemicals and cancer are concerned, in New Jersey. Is that correct, sir?

MR. HANSEN: I think my viewpoint and the facts are stated in the paper to the point, yes, sir, that chemicals contribute very little to the cancer rate in New Jersey.

SENATOR SKEVIN: And it is very healthy here in New Jersey: is that what your overall conclusion is?

MR. HANSEN: I have seen many facts that say that New Jersey is among the healthiest states in the nation.

SENATOR SKEVIN: Do you have any quarrel with the statistics that show that cancer is the number two cause of death in the entire country?

MR. HANSEN: I have no reason to dispute that.

SENATOR SKEVIN: You have no quarrel with that?

MR. HANSEN: No, sir.

SENATOR SKEVIN: You have no quarrel with the statistics which show that cancer has caused 355,000 deaths in the United States last year? You have no quarrel with that?

MR. HANSEN: I presume that is correct, yes, sir.

SENATOR SKEVIN: You have no quarrel with the fact there were 655,000 new cancer cases in the entire country last year?

MR. HANSEN: That sounds reasonable based on data I have seen.

SENATOR SKEVIN: You have no quarrel with the fact that in New Jersey last year we had 14,000 deaths resulting from cancer?

MR. HANSEN: That sounds ---

SENATOR SKEVIN: --- fairly reasonable?

MR. HANSEN: Yes.

SENATOR SKEVIN: You have no quarrel that there were 26,000 new cases of cancer in New Jersey last year?

MR. HANSEN: My number is a little bit higher than that.

SENATOR SKEVIN: Approximately. You have no quarrel with that?

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MR. HANSEN: No.

SENATOR SKEVIN: And you have no quarrel with the fact that since 1933 when we have taken statistics that cancer has increased 1 percent each year. Do you have any quarrel with that?

MR. HANSEN: I am not familiar with it, but I take the Senator's word for it.

SENATOR SKEVIN: You will accept my statement?

MR. HANSEN: Certainly.

SENATOR SKEVIN: You have no quarrel with the fact that cancer increased from 3 to 4 percent last year, according to the statistics that we have available?

MR. HANSEN: I am not familiar with that, but again I accept your word for it.

SENATOR SKEVIN: You have no quarrel with the fact that from an economic standpoint, in terms of medical expense and lost earnings in wages, there is a $15 billion loss to the entire economy in the United States. Do you have any quarrel with that?

MR. HANSEN: I would want to examine that more before I accepted it.

SENATOR SKEVIN: But if I told you we had had that type of testimony and that type of information, would you accept that as a statistic?

MR. HANSEN: Well, Senator, one thing I have learned in examining a lot of the testimony that has been made before this Commission and a lot of other articles that have been published is that you have to be very careful ---

SENATOR SKEVIN: --- with statistics.

MR. HANSEN: (Continuing) --- with statistics and with accepting facts on face value. You need to examine them very carefully.

SENATOR SKEVIN: --- just as you have testified about authorities. You didn't mention any authorities in your statement. You have testified about authorities saying this and authorities saying that about the chemical industry, but you ---

MR. HANSEN: We can substantiate what we have said.

SENATOR SKEVIN: So in terms of substantiation, you haven't given it at this point; you have just indicated you have had authorities.

MR. HANSEN: Yes.

SENATOR SKEVIN: Would you accept the fact that in terms of economic loss in 1975, in New Jersey approximately $100 million were expended for medical expense for cancer patients? Would you accept that? Do you have any quarrel with that?

MR. HANSEN: Senator, I accept your word for that.

SENATOR SKEVIN: Do you have any quarrel with the fact that overall people in New Jersey through medical expenses and lost earnings and wages have lost approximately $1 billion? Would you have any quarrel with that?

MR. HANSEN: I accept your word, Senator.

SENATOR SKEVIN: Okay. If we say "authorities," and you said "authorities," you will accept that, won't you?

MR. HANSEN: If I may ---

SENATOR SKEVIN: If we accept your statement that you have authorities, you will accept that statement as authorative?

MR. HANSEN: Yes.

SENATOR SKEVIN: Okay. Would you accept the statement that most cancers involve environmental factors, that up to 90 percent of cancers involve environmental factors? Do you have any quarrel with that statement?

MR. HANSEN: That sounds high. The number I have heard, Senator, and I think it is a fairly accurate number, is about 80 percent. And are you going to discuss what

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environmental factors are?

SENATOR SKEVIN: Well, we are talking about just the percentage - say 80 to 90 percent.

MR. HANSEN: I think we need to define what environmental factors are. It is extremely important to recognize that that includes all factors involved in a person's life, such as whether he smokes or whether he drinks, the kind of atmosphere he lives in.

SENATOR SKEVIN: Or the substances in the atmosphere; isn't that so?

MR. HANSEN: Yes. --- all of his own personal activities, as well as his surrounding environment.

SENATOR SKEVIN: Including chemicals.

MR. HANSEN: Including chemicals.

SENATOR SKEVIN: And chemicals are involved in the air and in his food - in the ground.

MR. HANSEN: Chemicals are involved in every phase of the environment. Everything in this room is composed of a chemical.

SENATOR SKEVIN: --- in every phase of the environment. Isn't that correct, sir?

MR. HANSEN: Involve chemicals, certainly. Our whole world is made up of chemicals.

SENATOR SKEVIN: And you feel at this point there really isn't any problem involving chemicals and cancer in New Jersey. Is that what your conclusion is?

MR. HANSEN: I did not say that, Senator. What I said is that the problem involving chemicals in New Jersey, in our opinion, is extremely small, extremely minute - that it is a very small part of the overall problem. Yes, there is a problem. Anytime you have ten people affected by cancer, which is our best estimate, by the chemical industry, that is a problem and we are trying to deal with that problem.

The other thing, Senator, that we should bring out is the fact that, as you know, I am sure, it takes many years for cancer to appear. And the ten cases that are estimated that were caused by the chemical industry - these exposures occurred 20 years ago, 30 years ago. Under current conditions, today's conditions, which is the important thing, we don't think that we have problems that are promoting cancer among chemical workers or among the surrounding communities in New Jersey.

Admittedly, years ago, we did have some problems. But they have been recognized and they have been corrected to the best of our knowledge.

SENATOR SKEVIN: In terms of your opening statement, Mr. Hansen, where you say in your second paragraph, "We want to determine, probably more than anyone, whether we have a problem," that seems to be in conflict with what you just told me. Do you say that we have a problem or that we don't have a problem?

MR. HANSEN: Well, I guess it's my engineering background, my scientific training, coming out, Senator. You try not to keep a closed mind in any area; you try to keep an open mind and always be amenable to new facts, new pieces of data and new findings. So I think all of us need to be alert. I think it would not be sound to take a position that there is absolutely nothing wrong, that we can't do any better, and that we should stop doing anything. We think we should continue to try to improve.

SENATOR SKEVIN: Do you agree with my statement that the same approach should not be used for chemicals as human beings, that we should consider chemicals guilty until proven free of any dangerous substance or cancer-causing substance?

MR. HANSEN: Well, Senator, I think that is kind of the way the chemical industry approaches the way it deals with the materials that it handles and manufactures. Each time we develop a new substance, a new material, and each time we get involved in a new process, we spend a great amount of time and money to explore the substance, the process

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and all the facets of it, to make sure that problems are not being created.

SENATOR SKEVIN: That there are no cancer-causing substances in the process? Is that what you are saying?

MR. HANSEN: No, sir, I did not say that.

SENATOR SKEVIN: Oh, I see. So you are not looking for cancer-causing substances?

MR. HANSEN: Yes, we are. But I should make myself clear that we try to understand and know what we are dealing with and to make sure that we handle whatever the material is in the proper fashion.

SENATOR SKEVIN: Approximately how many new chemical substances are introduced each year?

MR. HANSEN: I don't have that fact.

SENATOR SKEVIN: Twenty-five hundred? Would that be about it?

MR. HANSEN: I think it is more than that.

If I may, Senator, let me introduce George Polzer. He is Chairman of the Chemical Industry Council of New Jersey. He is here with me today. Possibly George can help you on this question.

MR. GEORGE F. POLZER, JR.: I am not absolutely certain of those figures. But if you check with the Federal EPA, I think with respect to the Toxic Substances Act which was recently passed, they used the statistic of about 100,000 new chemicals every year.

SENATOR SKEVIN: And the chemical industry makes sure that there are no problems with cancer-causing substances with 100,000 substances each year?

MR. POLZER: Yes, sir.

SENATOR SKEVIN: What standards do you meet under those tests?

MR. POLZER: The standards which are defined as far as OSHA requirements are concerned - EPA, FDA, NIOSH, and you name them, Senator.

SENATOR SKEVIN: How long have those standards been in effect?

MR. HANSEN: Many, many years.

SENATOR SKEVIN: The same standards have been in effect ---

MR. HANSEN: Not the same standards. The standards are modified as we learn ---

SENATOR SKEVIN: As we learn more about cancer and the factors involved, the standards have been changed. Isn't that correct?

MR. HANSEN: Yes.

MR. POLZER: Senator, I feel that if you want to pinpoint how long they have been in existence - I don't know the date either - but the FDA was probably the first body in that respect.

SENATOR SKEVIN: And the Toxic Substances Act that you referred to, that has been before Congress for five years; isn't that correct?

MR. POLZER: It was a long time.

SENATOR SKEVIN: Five years -- over five years. Isn't that correct?

MR. POLZER: I am not certain.

MR. HANSEN: I don't know.

SENATOR SKEVIN: Less than five years?

MR. HANSEN: I said I don't know.

SENATOR SKEVIN: Will you take my word for it that it has been before Congress at least five years?

MR. HANSEN: Yes.

SENATOR SKEVIN: And we have never really had any action on that until industry and government came to agreement. At first, the initial Toxic Substances Act met great

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resistance: isn't that correct?

MR. POLZER: No. There was a bill introduced in Congress: there were several bills introduced. Some of those met great resistance, yes.

SENATOR SKEVIN: By the chemical industry.

MR. POLZER: We gave our opinion with respect to the various segments of the bill.

SENATOR SKEVIN: They were too stringent: isn't that correct?

MR. POLZER: Yes.

SENATOR SKEVIN: They were too stringent for the chemical industry.

MR. POLZER: I don't like the word "stringent." I think it is better to say that it gave a man a dictorial power as an enforcement agency.

SENATOR SKEVIN: --- in order to protect the people in terms of the dangers involved in toxic substances: isn't that correct, sir?

MR. POLZER: I think that is the basic reason for the Act.

SENATOR SKEVIN: And it took five years or at least five years. We are not arguing ---

MR. POLZER: I don't know what point you are making. I think the bills were finally introduced about a year and a half ago.

SENATOR SKEVIN: But they have been before Congress ---

MR. POLZER: As far as the legislative process is concerned, you are more familiar with that than I am. And it could have been kicking around in the Senate and the Congress for seven years. I don't know how long it has been kicking around.

SENATOR SKEVIN: But it is not something that has been enacted recently. We are in agreement on that.

MR. POLZER: It hasn't been enacted yet. It hasn't been signed by the President, Senator.

SENATOR SKEVIN: On the second page of your statement, Mr. Hansen, in the second paragraph, you talk about your safety record, being "among the best in American industry." Again we will accept your authorities, but are you talking about New Jersey in that regard?

MR. HANSEN: Yes, as well as the chemical industry as a whole.

SENATOR SKEVIN: And are you talking about safety in terms of employees being injured on the job: is that what you are talking about?

MR. HANSEN: All aspects of safety, including that, yes.

MR. POLZER: Including the environmental hygiene atmosphere of our plants, etc.

SENATOR SKEVIN: In the next paragraph, you talk about being puzzled by some of the test results. You mentioned you heard some of our testimony at the last hearing. We had a Federal EPA expert who expressed concern about the danger as a result of those tests and concern about the danger in four or five areas in New Jersey. And we were told by the expert that the federal authorities were not able to do anything about this problem and they are conducting further tests - they are not ready to move on this problem. Could you reconcile what you are puzzled about and the expert from the EPA as far as his concern about the dangerous substances and the dangerous conditions?

MR. HANSEN: Yes, sir. I read his testimony pretty carefully and the facts presented there did not make a lot of sense to us with respect to the kind of operations being conducted in the area. The first supposition you make in looking at that sort of thing is - well, there's a chemical plant and that's the reason that you picked up these particular materials. But there is no consistency. There doesn't seem to be good correlation at all with respect to the chemicals picked up in the analyses and what is going on in the area. And that is the basis for our, as I put it, puzzlement. The

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facts just don't seem logical.

The other thing that the witness, I think, did a very good job of doing is pointing out to the Commission the uncertainty of the data, the fact that they did not know what the data meant, and that it should be handled very, very carefully because it was preliminary and more work needed to be done to understand the results that they were getting. They were not at all sure of what they had and what the meaning of the data is.

SENATOR SKEVIN: But there was no uncertainty when he said there was a danger and a concern there.

MR. HANSEN: A concern, but I don't recall the reference to danger. But certainly there is concern: and, if he did say danger, I would have to defer judgment on that until we had data that would give us concentrations and that sort of thing.

SENATOR SKEVIN: What you want is more tests and more studies involved.

MR. HANSEN: We want the facts, yes, sir.

SENATOR SKEVIN: You want the facts. People are dying from cancer and contracting cancer in New Jersey, but you want us to wait until we get all these facts and study this problem more. Is that what you want?

MR. HANSEN: No, sir. I think we need to look at the facts, Senator. From the work I have done, the study I have done and the people I have talked with, there is no clear correlation with the cancer rate in the State of New Jersey and the chemical operations that are in New Jersey or the chemicals that are in the air in New Jersey.

SENATOR SKEVIN: There is no clear conclusion to the contrary, is there, Mr. Hansen, that chemicals are not involved?

MR. HANSEN: Well, there is an awful lot of data and an awful lot of facts that say that Jersey is not different from other urbanized areas. I would be glad to share that with the Commission.

SENATOR SKEVIN: But you will agree that New Jersey's cancer rate is at least 14 percent more than the national rate?

MR. HANSEN: More than the national rate, but not more than comparable areas, whether there are chemical industries or not in those areas. New Jersey's rate is equal to or less than comparable areas.

SENATOR SKEVIN: But you are not quarreling with me about the fact ---

MR. HANSEN: Senator, ---

SENATOR SKEVIN: Let me finish now. You are not quarreling with the fact that cancer in New Jersey is 14 percent more in terms of the incidence of cancer and deaths as far as the national level is concerned? You are not quarreling with me about that, are you, sir?

MR. HANSEN: Let me think about that a second.

SENATOR SKEVIN: Will you accept it if I tell you that authoritative people have submitted that type of statistic?

MR. HANSEN: I have presented before the Commission data for 1974 that say just the opposite, that show New Jersey is lower than the national average.

SENATOR SKEVIN: In cancer?

MR. HANSEN: Yes, sir, if you look at Table I, that for a comparable area ---

SENATOR SKEVIN: I am not talking about comparable areas; I am talking in relation to the national average of cancer.

MR. HANSEN: With respect to the total U. S.?

SENATOR SKEVIN: Where do you think we get the designation of the number one - the capital of cancer? Where do you think that comes from -"New Jersey, cancer capital; Bergen in top 10 percent"? Do you think somebody made that up or somebody was trying to make headlines out of that?

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MR. HANSEN: That particular analysis that you referred to, if I am familiar with it, comes from the so-called Cancer Atlas. And those statistics as presented are very, very misleading.

SENATOR SKEVIN: Fourteen thousand deaths - is that misleading?

MR. HANSEN: No, sir.

SENATOR SKEVIN: Twenty-six thousand new cases of cancer in New Jersey - is that misleading?

MR. HANSEN: Those are factual - no question about that.

SENATOR SKEVIN: You will accept that.

MR. HANSEN: The point, Senator, that I am trying to get before the Commission is the fact that,when you compare New Jersey with comparable areas, whether there be chemicals or not in those comparable areas, as long as the population density is comparable, New Jersey's cancer rate is not out of line: that, if anything, it may be a little lower.

SENATOR SKEVIN: Is that acceptable to you, Mr. Hansen, that it is not out of line?

MR. HANSEN: Cancer is not acceptable to me or to the chemical industry or, I think, to any of us. It is something we would all like to wipe out.

SENATOR SKEVIN: You are not trying to lull us into a situation where we will study this problem more and not do anything about it?

MR. HANSEN: No, sir. I think I have said just the opposite. I think we need to take aggressive, objective and rational action.

SENATOR SKEVIN: Your approach is to study it more.

MR. HANSEN: No, sir. Part of my approach is to study it. A good part of my approach is to take a lot of action: action in the area of communications, of telling people what the facts really are, of improving the medical care, the medical services, of trying to prevent cancer, of finding cures. There are many, many rational things that can be done and should be done; and we should improve the quality of what we are doing. That is one reason I mentioned this National Cancer Institute that was started about a year and a half ago. It looks as though it can be a very effective organization: and, if we don't do that, something similar to that should be done.

SENATOR SKEVIN: Let me get to another area here, Mr. Hansen. With regard to your studies toward which you contributed $5 million, could you tell us over what period of time that was?

MR. HANSEN: I think that was in about the last year and a half or so. Let me just check here for a second. It looks like that is over a period of about two years, Senator, and there is another $2 million that is currently being considered for additional areas that work will be done.

SENATOR SKEVIN: In terms of your total gross product, Mr. Hansen, could you tell us what percentage that $5 million represents?

MR. HANSEN: Not for the United States offhand. In the State of New Jersey, the chemical industry is one of your largest and represents over $6 billion a year in sales in New Jersey alone. In the United States, the chemical industry probably is --- What would you say, George?

MR. POLZER: I would have to refresh my memory on it. I think it is safe to state, Senator, that New Jersey is still the number one chemical state in the United States.

SENATOR SKEVIN: I am just trying to find the relationship between your concern about the cancer problem and your studies to your total product. Could you give me some idea? You talk about $6 billion in New Jersey.

MR. HANSEN: That is in terms of sales, yes.

SENATOR SKEVIN: And the $5 million, was that national?

MR. HANSEN: The Manufacturing Chemists' Association is a national organization,

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composed of most of the people that work in the chemical industry.

SENATOR SKEVIN: Your concern here is $5 million in terms of --- what is your total gross total product sales? You must have some idea of that, Mr. Hansen. You say $6 billion in New Jersey. Could we safely multiply that by 50 states?

MR. HANSEN: No, that is too much. That is way too much.

SENATOR SKEVIN: You'd like to have it that way.

MR. POLZER: We don't have that statistic.

SENATOR SKEVIN: You don't have that statistic.

MR. POLZER: But it can be made available to you. There are Bureau of Census figures.

SENATOR SKEVIN: All right. We are talking about a $6 billion industry in New Jersey and the chemical industry expends about $5 on a national level for studies.

MR. HANSEN: That is through this national organization. Now within each chemical company, there are many millions of dollars being spent in addition to that. And that total, I think, would be highly impressive to the Senator if we had it. We don't have it.

SENATOR SKEVIN: But $5 million is a comparatively small percentage of the total gross sales; wouldn't you agree to that, Mr. Hansen?

MR. HANSEN: Certainly.

SENATOR SKEVIN: You mentioned cancer and cancer research in New Jersey. I have a compilation of statistics provided to me by Dr. Corielle. I would just like to quote from the opening paragraph about his statistical analysis, which he says, "as shown in Table I indicates that New Jersey has 3.5 percent of the United States population, but 4 percent of new cancer cases, which makes the incidence of cancer 14 percent higher in New Jersey than in the United States." Would you accept that as authoritative? Dr. Corielle, incidentally, is involved in cancer ---

MR. POLZER: I accept it as an authoritative conclusion made by interpretation of data.

SENATOR SKEVIN: Sure. It is a matter of statistics. And Dr. Corielle is involved with the Cancer Institute that you mentioned before.

MR. HANSEN: Is he?

SENATOR SKEVIN: Yes, he is.

Senator Maressa, do you have any questions?

SENATOR MARESSA: Yes.

There is one thing, Mr. Hansen, that I don't follow in regard to establishing New Jersey as having the highest incidence of cancer. I am not particularly proud of that and I don't know that we should really want to establish that. If it is, it is; and I am sure everybody here, as your statement indicates, wants to cooperate in reducing the incidence of cancer in New Jersey.

MR. HANSEN: Yes, sir, we want to help.

SENATOR MARESSA: But if we have the largest chemical industry in the United States - and we do, as I interpret the statistics - and if we are the most urbanized state in the Union, having the largest number of people per square mile, and if we put the two together, how can we escape the fact that the incidence of cancer in particular areas - and I think the reference was to the most congested or urbanized areas - is not in New Jersey? -- if my question makes any sense.

MR. HANSEN: I think I understand your question, Senator. Some very careful studies, and even these have to be looked at ---

SENATOR MARESSA: Excuse me. Maybe you can just preface that with -- I think you said in comparable areas.

MR. HANSEN: Yes, sir.

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SENATOR MARESSA: Can you identify or describe what you mean by "comparable"?

MR. HANSEN: Yes, sir. If we take Newark, for example, and compare it to New York, compare it to Philadelphia, compare it to Boston, to Buffalo, to Washington, D. C., Baltimore and Pittsburgh, they are all in the same geographical area with many different things going on in these areas. Some are highly industrialized; others are not. They all have high population density. They are all here in the Northeast. Newark has a cancer incidence of 215; New York City, 215.6; Philadelphia, 221; Boston, 223; Buffalo ---

SENATOR MARESSA: Excuse me. I think there is no need to go through each one of them. But really you can't fragment the state and say, "Well, look, New Jersey is not number one because the incidence of cancer in Newark is not as high as in New York or Buffalo." But if you take the state as a whole, without this fragmentation, are we number one?

MR. HANSEN: Of cancer in the United States?

SENATOR MARESSA: Yes.

MR. HANSEN: No, sir.

SENATOR MARESSA: Upon what statistic do you base that?

MR. HANSEN: That is Table I, if you will just look at Table I for 1974. Even if we go back to the Cancer Atlas, the data that was presented to the Commission before which showed New Jersey to be number one was based on all white males and that is not the total population.

SENATOR MARESSA: Well, are we number one for all white males?

MR. HANSEN: Based on the Cancer Atlas statistics, which are about six years old, yes, sir.

SENATOR MARESSA: I am afraid with statistics, you can take a look at them from different points of view and arrive at any conclusions that you wish.

May I ask a couple more brief questions.

Who are the members of the group that you represent?

MR. HANSEN: The Chemical Industry Council of New Jersey?

SENATOR MARESSA: Yes.

MR. HANSEN: It is composed basically of - what? - about 60 or 70 percent of the large chemical manufacturing and small ---

MR. POLZER: -- of all chemical manufacturing.

MR. HANSEN: -- of all chemical manufacturing in the State of New Jersey.

SENATOR MARESSA: Does that include the Owens-Illinois Plant?

MR. HANSEN: No, I don't think they are one of our members.

MR. POLZER: They are not in the chemical business; they are in the glass business, I think.

SENATOR MARESSA: They are not.

MR. HANSEN: No, sir.

SENATOR MARESSA: How about Johns-Manville?

MR. HANSEN: No, sir.

SENATOR MARESSA: How about Matty Bishop?

MR. HANSEN: No, sir.

MR. POLZER: Matty Bishop is cosmetics, if I remember correctly. I am not familiar with them. They are not part of our industry.

SENATOR MARESSA: They are not?

MR. HANSEN: No, sir.

SENATOR MARESSA: Give me some idea of who are members?

MR. HANSEN: Well, we have my company, Linden Chlorine Products; we have Witco;

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we have Du Pont; we have Hercules; American Cyanamid; Allied; Carbide. Help me, George.

MR. POLZER: There are some of the smaller companies, such as Mallinckrodt. There are roughly 62 companies all told.

I might add, Senator, if I may, that the question that you just posed is an indication, in many cases, of the chemical industry being blamed for something when it may be the metal plating industry who are using chemicals and sort of allied type of businesses. This is not the main function of the chemical business.

SENATOR MARESSA: I understand. You say the Manufacturing Chemists' Association conducted studies costing over $5 million. What is the Association of the Manufacturing Chemists to the chemical industry?

MR. POLZER: None whatsoever.

SENATOR MARESSA: They spent $5 million; have you spent anything collectively?

MR. HANSEN: As an organization, no. The member companies have spent many millions.

SENATOR MARESSA: But as an organization you have no funds?

MR. HANSEN: We have a relatively small budget.

SENATOR MARESSA: You have indicated your willingness to help. Of course, I don't expect this as a commitment; but if Senator Skevin's ideas take wing - and we hope they will - do you suppose your Council would be willing to contribute any sums of money towards cancer research and help this Commission?

MR. HANSEN: Senator, certainly we would be willing. Whether we would be able to is something else again. But certainly we are going to work to help in this area as much as we possibly can. I can't make a commitment as you indicated, but the commitment we can make is that we want to help, we want to work, and we will try to get whatever help is required.

SENATOR MARESSA: Fine. There has been some discussion about the fact that your Council lobbied against the Federal Toxic Substances Act. Can you explain why that was done?

MR. HANSEN: Well, there were provisions of the Act which were very restrictive and very harmful that went far beyond what is required for adequate control of the substances involved, and particularly with respect to small companies. Some of the requirements and some of the restrictions were just out of reason. So we worked with Congress to try to get a bill put together that would do the job and do it in a manner that would make sense to the companies involved, as well as to the taxpayers. We think that is what has happened. There is a good, sound bill that will be a real asset to the American people.

SENATOR MARESSA: Mr. Hansen, just finally, you say, "We are here because we want to be good citizens and we want to help." I am satisfied that that is true. Speaking for myself, what I would like to see happen is for you to follow through on this and we be able to call upon you for financial help, for expertise, and for whatever other help we are going to need.

MR. HANSEN: That's right.

SENATOR MARESSA: There is just too much cancer in the State. It is increasing if our diagnosticians and if the people in the Health Department and DEP have any knowledge at all. We are all going to have to get together and do something about it.

MR. HANSEN: Yes, sir.

SENATOR MARESSA: Thank you very much.

SENATOR SKEVIN: Mr. Hansen, if I could just add a footnote to the remarks well expressed by Senator Maressa, we are certainly interested in your statistical data that you mentioned and referred to in your statement. Will that be available to our Commission?

MR. HANSEN: Yes, sir. We will cooperate in all areas.

RSV 0013743

SENATOR SKEVIN: When can we have that statistical data?

MR. HANSEN: Senator, I think it will take us about a month to get it to you.

SENATOR SKEVIN: If you could expedite that, I would appreciate it.

MR. HANSEN: We will try.

SENATOR SKEVIN: In terms of follow up and help in the area of research, of course, you know we are on the bottom of the barrel in New Jersey. We have very little research funds and very few research facilities that are involved in this area. We appreciate your concern and your help and we would look forward to your involvement in this area.

MR. HANSE: Thank you, sir.

SENATOR SKEVIN: Thank you, Mr. Hansen.

Joseph Katz, Joseph Katz Company, Tobacco Tax Council.

J O S E P H K A T Z: Thank you, Mr. Chairman and Senator Maressa. I am Joseph Katz and I am New Jersey Public Affairs Counsel for the Tobacco Tax Council, which is a national organization whose sole purpose is to work against the imposition of unfair or too high cigarettes taxes and, to a degree, taxes on other tobacco products.

New Jersey has been a focus of interest of the Tobacco Tax Council because we have the third highest cigarette tax in the nation. Our concern is not with the health aspect of cigarette smoking.

SENATOR MARESSA: Excuse me. Mr. Katz, who is one and two?

MR. KATZ: Connecticut and Massachusetts. I think they are 22 cents and we are 19.

SENATOR SKEVIN: Connecticut is 21 cents. Massachusetts is 21 cents.

MR. KATZ: Twenty-one. I'm sorry.

As I was saying, we are not expert on the health impact of cigareete smoking. We are all aware of the Surgeon General's report. That, of course, has been dealt with in many ways on the national level, which I think we have to expect is the only way to deal with this health problem of smoking. It would be pretty hard to do it on a state-by-state level.

We are concerned about the economic, the social and the law-enforcement aspects of cigarette taxation. And individually, speaking for myself, of course I am concerned as a citizen about these statistics that indicate that New Jersey is a center for cancer and we have a particular problem here. It is frightening. The question is: What is the relationship of cigarette smoking to that problem? I have some statistics here and I will leave them with the reporter: (See pages 30, 31 and 32 for statistics to which Mr. Katz refers.)

New Jersey is somewhat below the national average in tax-paid cigarette sales. The figures for 1975, which are the latest figures I have, show that our tax-paid sales per capita were 122.3 packs per capita, while the national average of all taxing states per capita was 130.9. Now we realize that because of New Jersey's high cigarette tax there have been a lot of untax-paid sales in New Jersey, as in New York. Nevertheless, that might move us up to the national average or slightly above. But we don't stand out with regard to cigarette smoking like we do in the incidence of cancer. So, if those figures are accurate - and I have no reason to dispute them - about the incidence of cancer, I don't think by the process of arithmetical deduction that they can be attributed to cigarette smoking. Cigarette smoking may be an underlying national factor in cancer and other ailments, but no particular factor in New Jersey, at least according to available statistics.

I want to talk a little about the other impact of New Jersey's cigarette taxation, and there are some other people here from the wholesale and retail industry who can give you more direct testimony on that.

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First of all, cigarette taxes have been found by the most authoritative studies of tax policy that we have had - and we have had a number - to be the most regressive taxes in our arsenal of taxation. I was interested in Senator Skevin's remarks that he was interested in people more than statistics. The Tax Policy Committee had two direct comments in its 1972 report about cigarette taxes. They did a study of regressivity of taxes and they listed some nine different taxes. They found that tobacco taxes in New Jersey were four times as regressive as our State sales tax, for instance, which is supposed to adversely affect the little man. Tobacco taxes were the most regressive taxes of all, below corporation business taxes, which were the least regressive: the sales tax: public utility taxes, tobacco taxes being twice as regressive as public utility taxes: motor fuels taxes: motor vehicle fees: insurance taxes: alcoholic beverage taxes: spectator admission taxes: and inheritance taxes. Tobacco taxes hit the least able to pay the hardest.

In Part I of the report, the New Jersey Tax Policy Committee concluded: "Besides the property tax, public utility and tobacco taxes impose higher effective rates on the under $5,000 income groups than any other class."

This is directly related to a bill by Senator Skevin which has a laudable intention of raising some additional funds for cancer research in the State. There have been many, many millions expended at the national level; and with the Cancer Institute and other efforts, the State certainly has a good opportunity to have some pinpointed studies of cancer made. But I would like to respectfully contend that this bill places a new burden in the wrong place, on the people least able to pay, to finance a condition that I think by the statistics I cited earlier cannot be traced to smoking. I grant it is for a laudable purpose. But I think the condition you are trying to cure can be tracked elsewhere.

I want to give you a few other figures on the economic impact of cigarette taxation in New Jersey. Our tax is higher than any of the surrounding states. We are all familiar with the problem of "butt-legging", smuggling or whatever it has been called. It is a tremendous problem in New York. I have seen statistics that indicate that almost half of the sales in New York are illegal, are untaxed. In New Jersey, they are not that high, but they are approaching that. Our tax, as we noted, is 19 cents a pack here. In New York State, not New York City, which has a special tax, the levy is 15 cents a pack. They do impose the sales tax on cigarettes in New York, so that brings the tax in the surrounding New York counties, near Bergen County, Senator Skevin, to about 17 cents a pack, which is a two-cent differential. In Pennsylvania, there is an 18-cents tax - 10 cents a carton difference. In Delaware, it is down to 14 cents: and in Maryland, which isn't very far away, it is 10 cents a pack.

What this does is encourage people to travel across to Rockland County, for instance, or over the river here to Morrisville and Bucks County, and buy their cigarettes in the stores and take business away from many of our smaller merchants who depend on this kind of trade. A person who comes in and buys a pack of cigarettes, a newspaper, some razor blades and a few other things - say he works in Trenton and lives over in Bucks County -- such a person when he has to buy any of these items is not going to stop on this side of the river - he's going to stop on the other side. The same goes for Bergen County and Gloucester County. New Jersey is a state with many close neighbors. That is a big problem, with which I think you are all familiar from past debates on the tax burden. And it is a real tax burden because when you lose the sale of one pack of cigarettes, you are losing 19 cents in tax. So if you raise the tax a penny, for each pack that is diverted, you have to sell 19 more packs to make up the loss. That's a big

RSV 0013745

problem. Unfortunately, when you raise cigarette taxes a penny, often that is reflected in a nickel increase in a machine. You have to be concerned about that.

Therefore, from an economic standpoint and from the social standpoint, in line with the statistics I have presented, I would like to urge you not to advance S 1540 as a solution to the problem with which you are dealing. As to the problem in general, I think every person of goodwill stands behind you.

I would be glad to try and answer any questions you might have, gentlemen.

SENATOR SKEVIN: Thank you, Mr. Katz, for your fine presentation. You always make a fine presentation for your many, many clients. It is typical of the good work you have done in your many years in political and public service.

MR. KATZ: Thank you.

SENATOR SKEVIN: In your statement, did you say there is no relationship between cancer and cigarette smoking?

MR. KATZ: No, sir. I would never make that statement. But there is no special relationship between New Jersey cancer and New Jersey cigarette smoking. Indeed, the figures we have show that cigarette smoking in New Jersey is about average or perhaps, if I want to be a stickler, it is somewhat below average because we are dealing with tax-paid sales and we are below the national average. Assuming there are bootleg sales, that would bring us up to around the average. We don't stand out in the amount of our smoking.

SENATOR SKEVIN: Our cigarettes in New Jersey don't cause as much cancer: is that what you are saying?

MR. KATZ: I say, if they cause cancer, they cause as much cancer as they do in Wyoming.

SENATOR SKEVIN: They cause the same amount of cancer.

MR. KATZ: Arithmetically.

SENATOR SKEVIN: It depends on the cigarette, is that correct?

MR. KATZ: I am no expert on findings. I read in the newspapers if you smoke a lower-tar cigarette, you have a better chance.

SENATOR SKEVIN: That has been a recent development which has been publicized well recently. I think the cigarette industry, to its credit, has now admitted that the lower the tar and nicotine, the less cancer. Isn't that correct?

MR. KATZ: Yes, sir.

SENATOR SKEVIN: And they are talking about 20 percent less cancer if you smoke cigarettes with less nicotine and less tar.

MR. KATZ: So I understand.

SENATOR SKEVIN: You don't have any quarrel with that?

MR. KATZ: No, sir.

SENATOR SKEVIN: Would you advocate or suggest to this Commission that we prohibit the sale of cigarettes that cause more cancer because they contain more nicotine and tar?

MR. KATZ: Well, I am not an expert on it. As I say, all I know about is the impact of the cigarette tax. As to the impact of a special type of ban on high-tar, high-nicotine cigarettes, my opinion is no better than anyone else's.

SENATOR SKEVIN: But it is pretty well accepted that low nicotine and low tar cause less cancer. I see someone shaking his head that they don't cause less cancer. Mr. Katz, you are familiar with the advertising. Isn't it the thrust of the cigarette industry that we have now less cancer with low tar and low nicotine?

MR. KATZ: That is my impression.

SENATOR SKEVIN: Isn't that an admission that cigarette smoking does cause cancer? We just had the chemical industry here deny any relationship. If I recall correctly,

RSV 0013746

that was the argument of the cigarette industry at one time.

MR. KATZ: I am not representing the cigarette industry, per se. I am representing one aspect of the cigarette industry that is concerned with cigarette taxation. As to the health aspects, I just don't know anything about it. I have no information on it.

As an individual - you asked me for my impression - my impression is, yes, there is a relationship and the advertising seems to reflect it.

SENATOR SKEVIN: The next logical step would be to prohibit the cigarette sales that involve a large content of nicotine and tar.

MR. KATZ: Let me go one step further. I think the logical step, if you want to deal in that frame of logic, would be to prohibit cigarette sales. But, apparently, socially, psychologically and for whatever other reasons there are, Congress has found it impossible to do so. They have had one great experience with Prohibition back in the '20's. So they have taken other steps: controlling advertising, requiring the Surgeon General's warning to be printed on the cigarette packs, and there have been various efforts taken to discourage smoking. That is a political-social decision about which I know no more than any other layman.

SENATOR SKEVIN: In terms of your presentation, what essentially you are stating is the economic effect of this increase.

MR. KATZ: Right. My arguments would be worth nothing if we could show that taxation reduced smoking.

SENATOR SKEVIN: Your argument about the economic effect is that it would mean perhaps less sales of cigarettes?

MR. KATZ: Less tax-paid sales.

SENATOR SKEVIN: Less tax-paid sales.

MR. KATZ: I think statistics show that New Jersey is pretty much around the national average in its smoking.

SENATOR SKEVIN: If we had less tax-paid sales perhaps we would have less cancer.

MR. KATZ: No, sir, because you can get cancer from a North Carolina cigarette as easily as you can from one purchased in New Jersey.

SENATOR SKEVIN: What you are saying is that it is an economic effect in terms of ---

MR. KATZ: Yes, sir - economic and social to the extent that it affects the livelihood of some people.

SENATOR SKEVIN: We might have some problems in terms of employment?

MR. KATZ: Yes, very much. I think Mr. Goldfarb and his associates will comment in greater detail on that.

SENATOR SKEVIN: That is similar to the argument we heard from the chemical industry, isn't it ---

MR. KATZ: I followed it, but I don't think so.

SENATOR SKEVIN: (Continuing) --- of the economic effect in New Jersey since we have the largest chemical industry, and, with stringent controls, we might affect the employment situation and we might have an economic loss here in New Jersey? Isn't that correct? The argument is sort of similar, isn't it?

MR. KATZ: We are not manufacturing cigarettes in New Jersey. It is not similar, no. It is a different thing.

SENATOR SKEVIN: You have an economic argument here; isn't that so?

MR. KATZ: From that standpoint, yes.

SENATOR SKEVIN: In terms of the area involved, we are really competitive, aren't we, with our neighboring states in the amount of the cigarette tax? Connecticut is 21 cents; isn't that correct?

MR. KATZ: Yes, sir.

SENATOR SKEVIN: Massachusetts is 21 cents: isn't that correct?

MR. KATZ: That is not a neighboring state. I am talking about New York, Pennsylvania and Delaware.

SENATOR SKEVIN: Okay. New York is 15 cents plus 8 cents in New York City, a total of 23 cents in the metropolitan area.

MR. KATZ: We are better than New York City, but we are worse than Rockland County and Orange County, which border on Bergen, Passaic and Sussex. We are a penny worse than Pennsylvania and 5 cents worse than Delaware. And we are 9 cents worse than Maryland, if you are driving up from the south.

SENATOR SKEVIN: Joe, I remember you were a heavy smoker when you worked with Governor Hughes.

MR. KATZ: Yes. I don't work for him anymore.

SENATOR SKEVIN: You are not smoking anymore?

MR. KATZ: I don't smoke.

SENATOR SKEVIN: I have no further questions.

Senator Maressa?

SENATOR MARESSA: I would just like to ask: Does the tobacco industry receive any federal subsidy whatsover?

MR. KATZ: I think so, but I don't know. I am not working for the tobacco industry, per se. I am working for one group that has been formed largely by the manufacturers and growers, the Tobacco Tax Council. It concerns itself with taxation. I can't speak authoritatively. I think there are subsidies to growers, to farmers, to maintain prices. There is also a federal tax. I can tell you about that. It is 8 cents a pack on top of the 19 cents in New Jersey.

SENATOR MARESSA: There is no question about the fact, if cigarette smoking were to be curtailed, there would be no loss of employment in New Jersey. As you indicated, cigarettes are not manufactured here: it is just a question of retailing them here.

MR. KATZ: Right, and wholesaling them. If you could curtail them through taxation, there would be that small loss. But my contention, Senator Maressa, is that we have had one of the highest cigarette taxes in the country and the only impact it has had is that it has increased illegal sales and reduced tax-paid sales, and, in effect, to a degree boomeranged.

SENATOR MARESSA: And I think you made a very profound statement when you said that increased taxation in any amount does not in any way curtail or reduce the smoking of cigarettes. In other words, cigarette sales continue to skyrocket, do they not, regardless of the price, it would seem, even if they had to pay a dollar a cigarette?

MR. KATZ: That's the way it seems to be and I think we have all seen it around us. As I said, if taxation did reduce cigarette smoking, I wouldn't be very effective in representing this group.

SENATOR MARESSA: That is all I have.

SENATOR SKEVIN: One other observation or perhaps a question, Mr. Katz: Are you familiar with the program established in Kentucky which provided a five-cent cigarette tax for cancer research?

MR. KATZ: No, I am not. What is their tax in Kentucky?

SENATOR SKEVIN: It is a five-cent specific cigarette tax on every pack sold in the state.

MR. KATZ: Did they add it on?

SENATOR SKEVIN: I am just quoting from Dr. Corielle's report here.

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MR. KATZ: Because Kentucky's tax is still substantially below ours here in New Jersey.

I do want to make this point and I hope it isn't taken to be against the operations of New Jersey's research. But last year, Dr. Howard Temin of the University of Wisconsin, who won the Nobel Prize in 1975, was testifying as a friendly witness for the Hart-Kennedy Bill which would have raised the federal excise tax, but was defeated, and said that a large and rapid increase in money for cancer research is not warranted. He said, we were not limited here in cancer research by money: we are limited by ideas. Again, if money could do it alone, who wouldn't be for getting any kind of money from anywhere to solve the problem? It has to be digested too. Perhaps we do have special needs in New Jersey because the statistics apparently show a special problem. But I wanted to make the point that just throwing money in won't do it necessarily.

SENATOR MARESSA: Dr. Corielle seems to need money every year and we - when I say "we," I mean the State - have not been able to give him all the money he needs.

MR. KATZ: That is why I have tried to differentiate between the New Jersey Cancer Institute and the problem in general.

SENATOR MARESSA: Has the tobacco industry ever made any gifts for cancer research?

MR. KATZ: From my reading, yes, there have been substantial grants on a national level, such as to universities, by the tobacco industry - not in New Jersey because it doesn't amount to that much as a manufacturing state.

SENATOR SKEVIN: Thank you, Mr. Katz.

Mr. David Goldfarb, Tobacco Distributors Association of New Jersey.

D A V I D G O L D F A R B: Good morning, Senators. I wish you would have recognized that I too was a legislator once and that you could have given me the same accolade you gave Joe Katz because I have a few clients.

SENATOR SKEVIN: We waited until Joe Katz finished though, Dave.

MR. GOLDFARB: Nevertheless, I appreciate the fact that the Commission is meeting and, as Executive Director of the Tobacco Distributors, I would just like to read this statement. I may delete some things as we go along and I hope that I will be able to answer any of your questions as they come.

I would like to refresh the minds of the Senators on the position of organized crime in "buttlegging" in the State of New Jersey. I address myself to the provision in the Skevin Bill for a penny rise in the cigarette tax. We now raise approximately $170 million in tax revenue in the State of New Jersey from the 19-cent tax, and that figure is going down, which I will substantiate in a moment. It is estimated at this time that New Jersey now loses from 10 to 15 million dollars a year, graduated since the taxes were raised some 3 or 4 years ago. To ask this industry for an additional penny, to some may be infinitesimal: but you see, members of the Commission, a penny would make the cigarette tax 20 cents. Therefore, we would estimate another 5 to 6 million-dollar loss. So we fail to see the increase of cigarette tax as a proper funding mechanism for this legislation.

We definitely go on record as favoring the Skevin Bill. We are wholeheartedly in favor of that. I think, Senator Maressa, you asked Joe Katz: Does the cigarette industry contribute anything to the fighting of cancer? In the last 12 years, this industry has contributed $57 million to the fighting of all causes of cancer in America, more than any other industry. On top of that, the federal government has spent billions of dollars in the last ten years. Just yesterday, in the overriding of the President's veto for the appropriation, HEW got $10 billion. Out of that $10 billion, it is expressly provided in the bill that cancer research will get some of those billions. So, all in all, there

RSV 0013749

is lots of money being funded for us to find the causes of cancer.

We would ask a fiscal note as an amendment to the Skevin Bill and ask that the money be taken from the general treasury. After all, as I explained earlier, the cigarette industry pays more than its fair share of taxes. It is a very simple maneuver for a New Jersey resident to cross our State line, as Joe Katz mentioned, into any surrounding state bordering New Jersey and find he can buy cartons of cigarettes for from 60 cents less in Delaware; 50 cents,in New York State; 20 cents, in Pennsylvania; and one dollar less in Maryland, as it relates to the prices he will be paying here. Although that is not bootlegging, it is another form, as you know, of revenue loss to the State. I would guarantee you that will happen, as it happened when the last tax rise was put into effect.

We know the State cigarette tax enforcement men have done an outstanding job in their endeavor to curb this loss of revenue. But more illegal cigarettes pass through this State than they could possibly catch. With the estimated loss of business, it should be noted that there will be a tremendous loss of jobs in all phases of our industry. At one time, there were over 20,000 retailers throughout the State. It is estimated at this time that there are substantially less, and that will be corroborated by a speaker for the retailers. This also adds to the loss of tax revenue due to the infiltration of illegal cigarettes into neighborhoods. Just this morning, a pack was put into my hand in the hall with a North Carolina stamp, and I almost fell through the floor because I don't want to touch it. First of all, I work for these people.

SENATOR SKEVIN: You won't cross that picket line, will you?

MR. GOLDFARB: No.

Well, cigarette smuggling is a problem in New Jersey. The current State tax rate of 19 cents per pack, the third highest state rate in the nation, encourages New Jersey smokers to buy their cigarettes from out-of-state retailers and from organized crime. Recent statistics from the Special Investigations Unit of the New Jersey Division of Taxation show that violations of the cigarette tax laws have increased. For example, the number of people arrested in fiscal year 1974 is up by 13.6 percent over fiscal year 1973. Also, the number of fines levied has increased by 21 percent over '73 and the figure for '74 and '75 is way up.

This Special Investigations Unit, which is funded by the State, has begun to make some dent in the illegal distribution of cigarettes in New Jersey. However, an increase in the New Jersey cigarette tax to 20 cents will just give organized crime a greater incentive to bootleg more cigarettes. For example, total fines levied in New Jersey in fiscal '74 amounted to $112,994 for bootlegging. To organized crime, such fines may be a nuisance, but they are certainly no deterrent to the big business of cigarette bootlegging. However, an added tax is.

Obviously, an increase in the cigarette tax rate in New Jersey will call for a stepped-up enforcement program by the Special Investigations Unit of the State. Such an expanded program will cost the State of New Jersey - both in revenue to finance the program and in additional manpower. Returns on the revenue invested in enforcement procedures, in most states are minimal compared to the costs of enforcement. This is part of what another penny increase would cause.

An indication of the seriousness of the problem in New Jersey is contained in a recent paper presented at the 46th National Meeting of the Operations Research Society of America by Professor Michael D. Maltz, a member of the faculty of the Department of Criminal Justice at the University of Illinois. Professor Maltz estimated that back in 1973, New Jersey lost $15.5 million in potential state tax revenue due to cigarette smuggling activities. In 1974 and '75, the amount was larger. Can you imagine the loss

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if you raised the tax to 20 cents?

Currently, the Commission is considering an increase in the State tax rate to 20 cents per pack. Such a move would give New Jersey the second highest tax rate in the nation, as I said. With a 20-cent tax, New Jersey residents will purchase increasing quantities of cigarettes from out-of-state retailers and from cigarette bootleggers, who are already operating in New Jersey and neighboring states. Preliminary studies indicate that if a 20-cent tax is put into effect, in 1976 New Jersey residents will purchase more than 165 million packages of cigarettes from outlets in bordering states and from organized bootleggers, resulting in an expected loss of $14,950,000. This is predicated on what we know has happened in the past.

Thus, an increase in the current New Jersey cigarette tax will encourage citizens to purchase their cigarettes from sources which by one means or another avoid the heavy burden of the State cigarette tax.

Senators, you may tell me another state or two has used this tax as a research method, but what you are not telling those who do smoke, especially the middle- and lower-income group is that these states are not up to 19 cents a pack as we are, and losing revenue each month. You referred to the State of Kentucky. Well, the basic tax rate in Kentucky is 3 cents a pack. Even if they raised it a nickel to 8 cents a pack for cancer research, what difference does it make? -- eight cents a pack compared to nineteen cents. We would recommend that that $8 million be taken from the treasury.

Each state around us is lower in tax and does not suffer the pangs of a vast buttlegging ring operation in New Jersey.

We, the distributors in New Jersey, are aware of what the Commission is trying to accomplish and we agree on the purpose. What we say to you, gentlemen, is: Consider taking your money from the $170 million we provide to the State treasury. Then you will have accomplished your purpose. To place a tax on one industry, one industry, who has had more adverse publicity than any other, is the easy way to cop out. The cause of this investigation and Commission was not to find an easy thing to tax to raise funds, but for research. And we feel this burden of finances should not be borne by consumers of one industry who not only pay the price for smoking, but who pay every other tax in this State that a non-smoker pays. In these times of unpopular taxation, I would suggest, as before, if it is the cigarette smokers you are after by taxing, then you must be aware, for every penny more you tax on cigarettes, you create a deficit in the yearly tax by causing less purchase of the product. We then ask the Commission to review its financing. There is,right at this time, a bill being put into the hopper to reduce cigarettes by 2 cents.

The cigarette tax imposed on the sale of cigarettes within this financially hard-hit State could have brought in more revenue - perhaps as much as 14 million to 20 million more - without the sale of even one additional pack of cigarettes, except for two very important reasons:

One, the disparity in the taxes on cigarettes between the State of New Jersey and the State of North Carolina - a difference of as much as 10 cents per pack - makes the bootlegging of untaxed cigarettes into New Jersey tempting and highly profitable, particularly for organized crime. We ask that the Commission not help make it more attractive.

Two, contraband cigarettes in the past have been retailed mainly through businesses which don't normally deal in tobacco products, such as beauty parlors, barber shops, shoe-shine stands, small drug-store outlets that don't handle all tobacco products, and other such business places, as a convenience to their customers and an added source of revenue for themselves - and even, as another example, from the back of a station wagon at

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a construction site. In recent months, however, the State has discovered that these untaxed cigarettes are being increasingly sold in legitimate retail outlets, as I suppose the pack I was handed was sold. The person who handed it to me out here wouldn't tell me where he bought it. In other words, more and more business men and women, who are law-abiding in other respects, have become law violators by evading the cigarette tax. Moreover, they are assisting in robbing the State of much needed revenue at the same time that they are becoming accomplices of big-time racketeers and small-time hoodlums. Perhaps, most tragic, the legitimate retailers who have become tax-evaders by selling contraband are subjecting themselves to arrest and criminal prosecution with resultant disgrace to their families as well as themselves. You will be shown a paper indicating five legitimate distributors in New York State have just been arrested for counterfitting New York stamps on cigarettes. They were considered very civic-minded citizens. Mr. Kleiner will show you that article. They did this because of the bootlegging which caused their products not to be sold from their warehouses in their distributorships.

Governor Byrne and Tax Director Glaser are well aware that cigarette bootlegging and cigarette tax evasion are a serious problem.

I make a point, gentlemen, that the cigarette tax is an important part of the revenue that helps finance our schools, our health programs and other vital public services. Every dollar stolen from the cigarette tax revenue is a dollar that must come from some other tax source. So, in the long run, the real loser is John Q. Public. The New Jersey citizen who buys untaxed cigarettes does not get a bargain; he simply helps increase the taxes he must pay in other ways. The only winners are the cigarette bootlegger and his accomplices. Moreover, cigarette bootlegging is a national problem.

The problems of the closing of tobacco businesses in this State because of the unfair competition of purveyors of untaxed cigarettes and the problem of the resultant loss of jobs, I assure you will be brought about by a raise in the tax of cigarettes.

I respectfully submit that we as one of the smallest really proven causes of cancer incidence should not bear the cost of the full funding of this program.

I would like to say in July, 1976 - just to substantiate the fact I have figures, and the figures for July 1976 are the latest figures Mr. Glaser could give me - as compared to July 1975 - and don't forget this is the summer season when all the tourists are here - the cigarette salesloss was $855,380 for that one month. I don't know yet what the figure will be for August because it takes the Tax Division 2 months to come up with figures.

I would recommend, Senator, in all fairness, that we not be asked to be bear the full cost of funding the Skevin Bill. We have supported the bill and will support the bill. We wouldn't think of doing otherwise. But our cigarette sales, due to the publicity we have received and due to the bootlegging, is way down and the Treasury is not very happy with the money that has been coming in in the last eight or nine months - or last year and a half. Bootlegging is way up.

I just wanted to make a point on the tax - that was all. If I can answer any questions, I will be happy to.

SENATOR SKEVIN: Thank you, Mr. Goldfarb, for your very enthusiastic - and also fine - presentation. You represent your clients well too and it also reflects your fine background as an Assemblyman who represented his constituents in Essex County very well.

MR. GOLDFARB: I may run again. If you say that to me, I may run again.

SENATOR SKEVIN: I have one question and maybe Senator Maressa wants to ask you something. Approximately what would be the revenue produced by an increase of one penny in the cigarette tax?

MR. GOLDFARB: I disagree with the content of the Skevin Bill. I don't think you

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would get $8 million. I think you might get closer to $6.5 million.

SENATOR SKEVIN: Six and a half million - is that your estimate?

MR. GOLDFARB: Possibly, because of the loss that we have experienced in the last couple years. It is a known fact. Sid Glaser will tell you that.

SENATOR SKEVIN: I have no further questions.

SENATOR MARESSA: I know things are tough, but as an inducement to us not to pass this bill, would your industry be willing to make any kind of a contribution at all or would you consider it? I know you can't answer that question right now. If you could get back to us with some money, maybe we wouldn't need six or eight million dollars. Maybe we would need only two million if we had the chemical people and the tobacco people contributing. We have to do something toward cancer research. Why don't you get together and maybe come up with a figure?

MR. GOLDFARB: I would like to say something off the record. Is that permissible?

SENATOR MARESSA: Ask the Chairman.

MR. GOLDFARB: It is not a big kind of a thing.

SENATOR SKEVIN: Everything is on the record here, as you know as a former legislator, Mr. Goldfarb.

MR. GOLDFARB: I would like to say that we already contribute nearly a quarter of a billion dollars - $170,000,000 - and I am not talking about any other industry contributing -- but from gasoline, alcohol and tobacco you get half a billion dollars in revenue. So what I am saying is: It is a beautiful bill. We love it. We are for it. But please, not again! This tax on cigarettes was knocked down in the last three years each time on the basis of the merits. It is hard on us now.

SENATOR SKEVIN: Mr. Goldfarb, I mentioned before the many fine clients that you represent. I notice additionally in your statement you indicated the position of organized crime. You don't represent organized crime, do you?

MR. GOLDFARB: Oh, no.

SENATOR SKEVIN: Okay. Thank you.

Mr. Ira Cucual, Retail Tobacco Merchants Association of New Jersey.

I R A J. C U C U A L: Senator Skevin, I'm the little guy.

SENATOR SKEVIN: You are a big guy.

MR. CUCUAL: Despite my bulk, I am still a little guy.

SENATOR SKEVIN: And you are important to us.

MR. CUCUAL: Senator Skevin and Senator Maressa, my name is Ira J. Cucual and I am a resident of Bricktown, in Ocean County, New Jersey.

Retailing has been my livelihood since 1937 and I have had stores in Elizabeth and Union, New Jersey. I have held office in county and state retail tobacco organizations. I am a former Secretary of the Retail Tobacco Dealers of America, a national organization, and have served as a Director of that group for almost twenty years.

I appear before you today on behalf of the thousands of tobacco retailers in the State of New Jersey. In 1948, a cigarette tax law was enacted in our State. The tax was three cents per pack. Today, 1976, the tax is nineteen cents per pack, an increase of 633 percent. At nineteen cents, the New Jersey cigarette tax is 58 percent above the national median tax rate of 12 cents per pack. Cigarette sales nationwide have increased; whereas, in New Jersey, sales have decreased more than 4 percent. I maintain the sales have dropped, but consumption has increased. Why the drop in sales?

Number one - bootlegging. Bootlegging is costing the State licensed retailer and the State licensed distributor millions of dollars in sales of cigarettes and related

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items. By "related items," I mean, candy, magazines, razors, razor blades and hundreds of other items. This loss of sales is costing the State of New Jersey many millions of dollars in taxes: that is, on cigarettes, sales tax and now also income tax. The sale of untaxed cigarettes, despite tremendous efforts by our Tax Department and State and local police, is increasing daily. Increasing the now 19-cents-per-pack tax will certainly stimulate and increase bootlegging.

Gentlemen, the State of New Jersey is hurting. The tobacco distributor is hurting. And we, the small retailers, are hurting too. We are here today to ask that you not tax us out of business. Please do not say that that cannot happen. In 1973, New Jersey had 18,185 licensed retail outlets. In 1974, there were 17,850, a loss of 335 licensed outlets. In 1973, we had 37,681 licensed vending machines; in 1974, 36,929 - a loss of 752. Add the two and we have a loss of 1,087 licensed retail outlets in two years. These figures are from our State Tax Department.

Reason number two: lower state taxes in neighboring Pennsylvania, 18 cents: New York State, exclusive of New York City, 15 cents: Delaware, 14 cents; Maryland, 10 cents. This is causing tremendous loss of sales due to commuters, visitors, truck drivers, etc., bringing cigarettes into New Jersey for friends, relatives, and too many bootleg operations. As retailers we ask that you not permit any increase in the cigarette tax. Any further increase will most certainly hasten the death of our business lives.

We retailers are proud of our position in society. We believe that we are an asset to our communities in the State of New Jersey. We are taxpayers, tax collectors - here I refer to the sales tax - lottery sales persons and active in all civic, welfare, patriotic, religious and charitable endeavors. Taxing the retailer out of business will not only affect him and his family, but will have a disastrous effect on his employees, the distributors and their employees, and will be costly to the general public in the area he serves.

Gentlemen, I ask you allow us to survive. Many thanks for the opportunity of meeting with you and I want to once again thank you.

SENATOR SKEVIN: Thank you, Mr. Cucual. You have presented your views very well representing the small guy, the retailers, which are very important to us. Your views will be part of this transcript and available to every member of the Legislature when the transcript is published.

Senator Maressa, do you have any questions?

SENATOR MARESSA: No. I can appreciate what would happen to your industry, and we are going to do everything we can to help.

SENATOR SKEVIN: Ms. Diane Graves of the New Jersey Sierra Club.

D I A N E T. G R A V E S: Thank you for the opportunity to testify.

My name is Diane Graves and I am Conservation Chairman for the New Jersey Chapter of the Sierra Club.

There must be a concerted effort by both the Federal government and the State to reduce human exposure to hazardous and toxic substances. Since New Jersey reaps the benefits of leading the nation in chemical manufacturing, it should also face the responsibilities involved in protecting both the workers and the public at large from the risks and hazards of exposure to dangerous substances.

Experts agree that chronic exposure to low-levels of pollutants poses a serious medical problem, and as this affects the greatest number of people, we cannot afford such pollutants the human right of "innocent until proven guilty." Of course, Senator Skevin has made that point very clearly. We also agree with the Commission and the DEP

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that if we err, it must be on the side of safety. We can't wait for certainty.

Environmental clean-up and protection is not a luxury that can be afforded after New Jersey's economy is straightened out - it is an essential part of our health and our economic well-being. Almost daily now, we are faced with new indicators, if not positive proof, that the environment is over-stressed: the ocean off Atlantic City is dying or dead: air-borne lead poisons urban children: fish are contaminated with PCB's, Kepone and Mirex: crops are damaged by air pollution: former drinking water supplies are laced with petrochemicals: preliminary tests indicate presently-used water supplies have traces of carcinogenic substances: dumps that have accepted chemical wastes pollute waterways and groundwater: carcinogens are in our air: workers have too-high levels of cancer-causing agents in their bodies: and we now know that 60 to 90 percent of cancer deaths are caused by environmental factors - therefore, they can be prevented.

The Cancer Commission has made clear that known carcinogens in New Jersey's water and air will be given serious attention and urgent action. As an early part of your effort, we urge that there be an evaluation of the DEP's ambitious proposed cancer program for "determining and defining the dangers which New Jersey residents face as a result of exposure to carcinogens and other environmental contaminants."

It would be useful to both the public and to the efforts underway for the Commission to conduct a thorough discussion of the proposal. The discussants should include appropriate DEP personnel, independent experts, such as Samuel Epstein, M.D., who is noted for his cancer-environment expertise, and representatives from interested groups, including environmentalists, industry, labor and health people. This could not only serve to clarify and assess the proposal, and determine specific priorities, but also help gain the public support needed to get it implemented.

The DEP proposal specifically states that it is a long-term program. We need a quick process for determining what needs to be done right now. There are a number of questions that need to be discussed. For instance, who decides what substances are toxic and which ones should have the highest priority?

If, or when, federal money, via the Case-Williams-Maguire amendment to the Toxic Substances Control Act, becomes available, interested New Jersey agencies, the Commission and the public sector ought to have thoroughly discussed, evaluated and decided where and how that demonstration program money is to be spent. Such a discussion should also include the Commission's legislative proposals prior to public hearings.

I will just add an additional comment on the Toxic Substances Control Act. It has been under way for at least five years and the final version is a compromise version, quite a bit weakened because of the industry lobbying against it.

In may 1976, 40 organizations, physicians and individuals urged that an Emergency Task Force on Environmental and Occupational Carcinogens in New Jersey be established. The Task Force should include a balance of representatives from consumer, environmental, health, independent scientific, industry, labor, medical, and public-interest groups. Industry is included on the Task Force, but it should not outnumber any other interest. The Task Force should have a professional staff, headed by a full-time staff director. We discussed briefly what the Task Force should do and, by when, its powers, the information it should obtain - primarily that of the workplace, since it is the workers who get the concentrated dose and thus suffer the most - that the Task Force be adequately funded, hearings be held, and that the findings and recommendations be made public. (I have attached a copy of that testimony.)

About the same time, the Governor established the Cabinet Committee on Cancer Control. For a number of reasons, we believe that the two entities should be merged in

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some way. At Commissioner Bardin's request, we submitted names of candidates for such a task force or advisory body. We urge your support.

We believe the people listed would be helpful not only on a task force or advisory group to the Governor's Cabinet Committee, but they would also be helpful in discussing, evaluating and deciding priorities for the DEP's proposed cancer program and the Commission's proposed legislation.

I have attached a copy of the letter to Bardin, with the list of names.

We are keenly interested in the Commission's legislative package and in learning its details. Senator Skevin's statements indicate the package would deal with carcinogenic substances in water and air emissions. We hope the package will also address disposal of hazardous and toxic wastes. Despite New Jersey's having the largest concentration of chemical plants in the nation, there appears to be little research, planning and regulation to insure the proper disposal of toxic and hazardous chemical wastes. New Jersey allocated only about $300,000 last year to regulate the more than three hundred sanitary landfills, and the thousands of waste-hauling vehicles, and to run the solid waste program. For perspective on that figure, in 1973, the National Sanitation Foundation said New Jersey ought to spend over $840,000 on solid waste management, and that was before the recognized crisis in hazardous and toxic wastes disposal. I have attached a copy of our newsletter which includes an article on this matter. I have also attached a copy of my letter to Bardin on the reported closing agreement for Kin-Buc I, Kin-Buc being the only remaining landfill that received chemical and toxic wastes. It was recently closed. And I call particular attention to item #4, Groundwater contamination.

We urge the Commission to push EPA to do thorough groundwater testing in New Jersey. A priority area might be Middlesex County because of the Kin-Buc dump and other chemical plants in the area, or perhaps initial efforts should be concentrated around landfills known to have accepted chemical wastes.

There was such short notice for this hearing that a number of people who could contribute to your deliberations and activities couldn't arrange to come today. A key person for you to contact directly for your next hearing is Louis Marinari, who represents the Oil, Chemical and Atomic Workers International Union in New Jersey. Mr. Marinari is included on our list to Commissioner Bardin. I will add here too that the Oil, Chemical and Atomic Workers Union would express quite different views, I believe, and bring you quite different information than those expressed by Mr. Hansen earlier today. I understand you have made preliminary contact with some other people on the list of the names that I submitted to Bardin, and we urge that you follow through and invite them, and others, to also testify.

I have also appended two papers by Samuel Epstein, M.D.: "Environmental Determinants of Human Cancer" and "Cancer and the Environment: A Scientific Perspective," for your additional information. (These two papers are on file with the Commission. Two letters to Comm'r Bardin submitted by Ms. Graves are on pages 33 & 35.)

Thank you.

SENATOR SKEVIN: Thank you, Miss Graves, for the very comprehensive and fine paper and submission. It reflects your devotion and concern, and the concern of your organization, in this very important matter. We certainly appreciate those suggestions and I feel confident that the Commission will follow them.

I don't have any specific questions. Your paper and your presentation cover the area well.

Senator Maressa?

SENATOR MARESSA: What is it that we should have as enclosures here? I am very interested.

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MS. GRAVES: I am sorry I only brought one package.

SENATOR MARESSA: Then I am stealing yours.

SENATOR SKEVIN: No, you have a copy of Diane's letter.

SENATOR MARESSA: She said she only had one package. Would it be possible for you to send me that?

SENATOR SKEVIN: We will have this reproduced for each member.

SENATOR MARESSA: In other words, your presentation includes copies of letters and names, etc., and you only brought one package of that.

MS. GRAVES: That's right.

SENATOR MARESSA: You know we have placed a great deal of faith in your sincerity and expertise. I intend personally to follow up on everything that you have presented here. You have some tremendous ideas and our next session should be rather interesting.

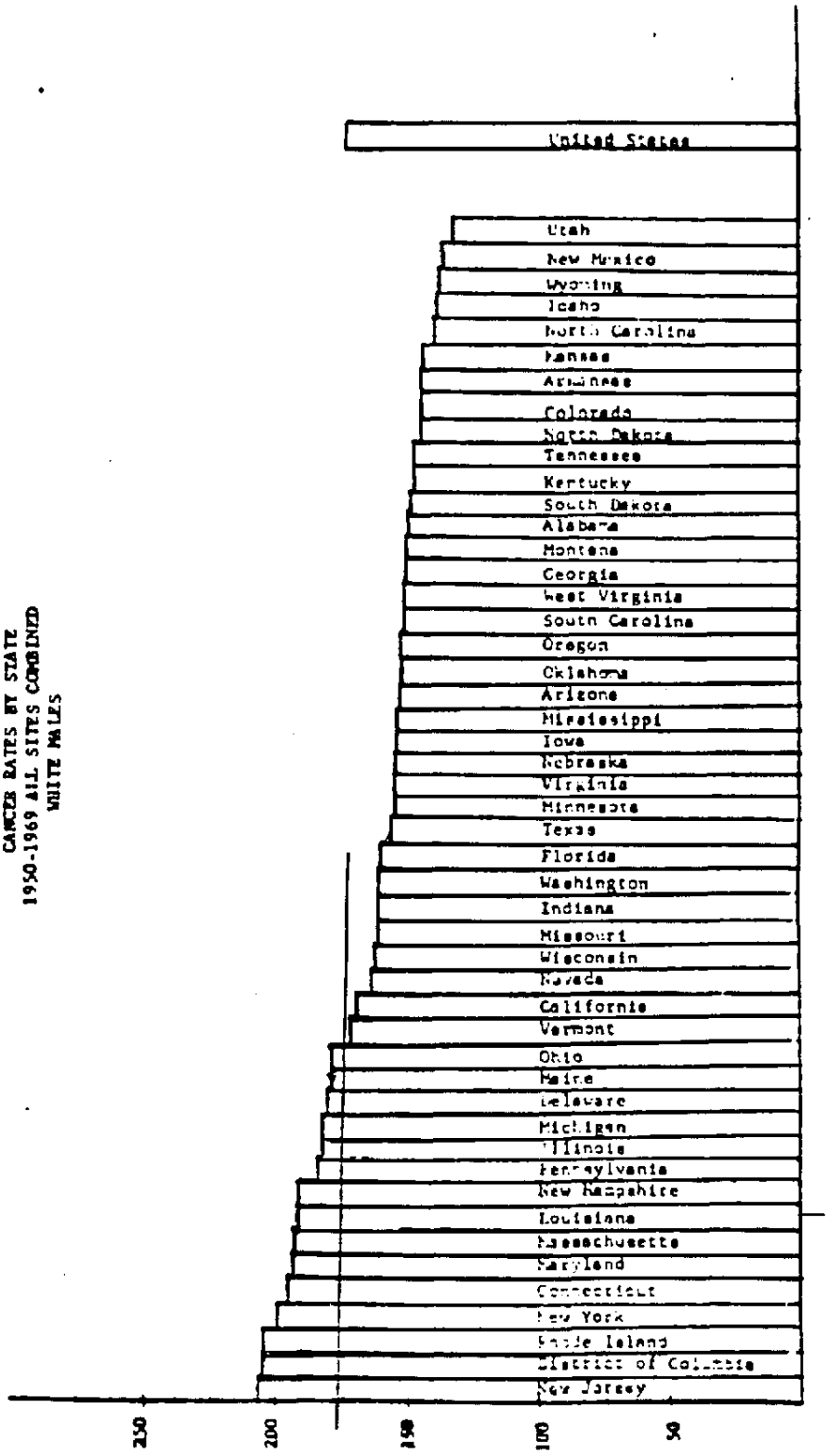
SENATOR SKEVIN: Thank you, Miss Graves.

This will conclude this hearing of the Senate Commission on the Incidence of Cancer in New Jersey. Further hearings will be schedule in the near future. Thank you.

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Submitted by C. A. Hansen, Jr.



Prepared by the Public Health Statistics Program
New Jersey State Department of Health

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Submitted by C. A. Hansen, Jr.

TABLE I

1974

	Total U. S.	Total Northeast Region*	Total New Jersey
Cancer Mortality	360,472	95,730	13,985
Rate per 100,000	170.5	193.7	190.8

New Jersey's rank in U. S. 10

New Jersey's rank in Northeast 6

* Connecticut
Maine
Massachusetts
New Hampshire
New Jersey
New York
Pennsylvania
Rhode Island
Vermont

Source: Vital Statistics Report
U. S. Dept. Health, Education, & Welfare
Public Health Service
Health Resources Administration

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SUBMITTED BY JOSEPH KATZ *Part I*

of the Report of the

NEW JERSEY TAX POLICY COMMITTEE

TRENTON, NEW JERSEY

February 23, 1972

TABLE 15

State of New Jersey

PROPORTION OF NEW JERSEY TAXES
PAID BY NEW JERSEY RESIDENTS: FISCAL 1970
(millions of dollars)

	Total Tax	Portion Exported Outside of N.J.	Net Burden on N.J. Residents	% Exported	% on N.J. Residents
Property Taxes	$1,924.4	$341.9	$1,582.5	17.6%	82.4%
Sales & Use Tax	355.6	68.3	287.3	19.2	80.8
Corporation Business Taxes	221.8	113.8	108.0	51.3	48.7
Public Utility Taxes	203.6	41.0	162.6	20.1	79.9
Motor Fuels Taxes	199.6	30.1	169.5	15.1	84.9
Motor Vehicle Fees	130.2	—	130.2	0.0	100.0
Cigarette Taxes	117.9	—	117.9	0.0	100.0
Beverage Taxes	42.5	—	42.5	0.0	100.0
Inheritance & Estate Taxes	66.6	13.3	53.3	20.0	80.0
Insurance Taxes	40.4	6.1	34.3	15.1	84.9
Spectator Sports Taxes	35.2	14.1	21.1	41.1	59.9
Commuter Benefits Tax	17.0	17.0	0.0	100.0	0.0
Railroad Taxes	7.4	7.2	.2	97.3	2.7
Other Taxes	31.9	0.0	31.9	0.0	100.0
Totals	$3,394.1	$652.8	$2,741.3	19.2%	80.8%

With regard to specific taxes, the percentage borne by the lowest income groups was highest for property and tobacco taxes. The two top income classes received about 41 percent of total income while bearing less than one-third of the tax burden. The highest income category, in particular, enjoyed a relative income share exceeding its tax burden share. Four-fifths of the consumer units in New Jersey had a tax share higher than their share of state income.

If the most important tax source is excluded from the figures, the tax burden share declines for those income groups under $10,000 while increasing for those above this level. The last row in Table 16 contains information on the distribution of tax burdens without the property tax. Under these circumstances, even the $15,000 under $25,000 income class bears a tax burden share higher than its proportion of total income.

Effective Tax Rates

Table 18 presents data displaying the pattern of effective tax rates by income class and type of tax. The effective rates were computed by expressing the absolute amount of tax paid in each income category as a percentage of total money income in that class. The manner in which effective tax rates change as one moves through the income distribution provides a basis for determining the progressivity of specific levies. A progressive tax is often defined as one in which the effective tax rate increases as income increases. A proportional tax may be defined as one for which the effective rate remains constant as income increases and a regressive levy as one in which the tax rate decreases as income increases. Unfortunately, few of the effective rate estimates for specific levies conform precisely to such simple definitions.

The total state and local tax burden on New Jersey residents amounted to over 10 percent of money income in Fiscal 1970.

This percentage represents an increase over that calculated a few years previously, implying that state and local taxes have increased at a faster rate than money income. For all income classes under $15,000, the effective tax rate exceeded that for all consumer units in the state. The rate for the lowest income group was almost twice as great as that for all families. State and local levies absorb almost one-fifth the annual income of those in the under $3,000 income class. The discrepancy between the effective tax rates falling upon the lowest income class and other units is almost solely attributable to the property tax. This levy is responsible for generating more than half of total state and local tax revenues. The effective tax rate of this levy falls twice as hard on the under $3,000 group as on any of the four income classes above $7,500.

As can be seen from the last row in Table 18, without the property tax, the discrepancy of effective tax rates is considerably narrower under this situation. Besides the property tax, public utility and tobacco taxes impose higher effective rates on the under $5,000 income groups than any other class. The liberal exemptions

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Part V

of the Report of the

NEW JERSEY TAX POLICY COMMITTEE

TRENTON, NEW JERSEY

February 23, 1972

Progressivity Approach

Progressivity of a tax system is closely related to income elasticity in that the more progressive the system the more income elastic it is likely to be. In addition, people tend to evaluate the tax system according to its progressivity, that is, in relation to its impact on the income to the taxpayer, as the measure of ability to pay.

Implicit in the use of an income measure of ability to pay is the assumption that the larger the income the less the sacrifice for each incremental dollar of tax payment. Starting from this theory, the effect of a tax system, or of an individual tax, may be described as either regressive, proportional or progressive.

Progressivity Indices A progressive tax may be defined as one in which the effective tax rate (the ratio of taxes paid to income) increases as income increases. A proportional tax may be defined as one for which the effective rate remains constant as income increases; and a regressive levy is one in which the tax rate decreases as income increases. Few taxes appear to conform precisely to such simple definitions. In many cases, the effective tax rate neither increases nor decreases continuously throughout the income distribution. Upward and downward movements of the effective tax rate between income classes cause problems in determining whether a levy is progressive or regressive as well as the degree of progressivity.

Because of the difficulties described above, we have employed a statistic which shows whether a particular tax is, *on the average*, progressive, proportional, or regressive. This statistic has been classified as a progressivity index and it measures the rate at which tax payments vary with respect to changes in income throughout the income distribution.[1] Two sets of such indices have been computed for each tax. In the first set, all income classes are weighted equally; in the second, each income group is weighted according to its relative importance with regards to population. Depending upon whether the index exceeds, equals, or is less than 1, a tax is classified as progressive, proportional, or regressive. Moreover, the higher the index, the more progressive the levy. Correspondingly, an index number considerably below 1, would imply a particularly regressive tax.

TABLE 5-11

State of New Jersey

PROGRESSIVITY INDICES OF MAJOR STATE AND LOCAL TAXES

USING FISCAL 1971 SALES TAX YIELD AND FISCAL 1970 YIELD FOR OTHER MAJOR TAXES

Tax	Unweighted	(1) Weighted by Relative Population Shares
Property Taxes	.53	.55
Corporation Business Taxes	1.16	1.06
Sales and Use Tax	.85	1.28
Public Utility Taxes	.61	.66
Motor Fuels Taxes	.67	.77
Motor Vehicle Fees	.80	.86
Insurance Taxes	.79	.92
Tobacco Taxes	.31	.31
Alcoholic Beverage Taxes	.84	1.01
Spectator Admission Taxes	.89	1.04
Inheritance-Estate Taxes	1.60	2.16
Other Taxes	.71	.99
Total Taxes	.64	.68
Total Taxes Excluding Property Taxes	.80	.86

(1) weight according to number of people in each income class

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Table 11
TAX-PAID PER CAPITA SALES
(in number of packs)
(During fiscal years ending June 30)

State	1970	1971	1972	1973	1974	1975	State
Ala.	99.0	95.4	101.1	102.9	108.2	111.7	Ala.
Alaska	131.4	123.0	130.0	126.8	130.4	150.4	Alaska
Ariz.	119.2	109.6	125.0	120.1	111.1	121.8	Ariz.
Ark.	100.3	104.1	101.9	102.0	109.2	114.8	Ark.
Calif.	123.0	121.0	123.5	124.3	126.7	127.1	Calif.
Colo.	124.8	125.5	134.1	132.9	132.8	131.0	Colo.
Conn.	120.0	117.6	110.8	101.1	117.4	110.2	Conn.
Del.	155.0	161.1	156.3	154.7	151.3	147.6	Del.
D.C.	200.4	213.0	220.6	209.4	182.7	176.5	D.C.
Fla.	123.6	123.0	126.0	132.6	136.0	131.9	Fla.
Ga.	109.9	115.7	117.8	109.8	123.7	122.9	Ga.
Hawaii	82.1	85.0	81.6	79.2	89.1	92.4	Hawaii
Idaho	102.4	108.5	126.1	121.8	125.6	123.3	Idaho
Ill.	124.8	125.6	126.6	124.4	131.9	131.8	Ill.
Ind.	134.6	139.3	149.2	156.0	159.6	162.4	Ind.
Iowa	108.5	108.4	109.4	110.6	116.1	120.5	Iowa
Kans.	114.0	102.8	111.0	115.2	118.6	123.4	Kans.
Ky.	155.8	163.5	179.4	201.9	217.4	223.0	Ky.
La.	115.9	119.8	125.3	126.7	129.9	133.6	La.
Maine	128.5	133.2	136.5	138.0	142.1	140.7	Maine
Md.	123.5	126.7	133.2	137.3	144.8	146.1	Md.
Mass.	124.3	121.4	117.9	121.2	124.3	126.1	Mass.
Mich.	128.6	126.1	131.4	133.7	138.8	136.8	Mich.
Minn.	104.3	116.4	96.8	106.8	110.6	111.5	Minn.
Miss.	93.4	105.4	112.1	115.0	117.1	116.8	Miss.
Mo.	121.3	127.6	130.0	132.1	135.4	135.6	Mo.
Mont.	111.2	115.6	122.2	119.9	121.9	123.7	Mont.
Neb.	108.1	108.6	103.9	108.6	110.5	114.1	Neb.
Nev.	189.5	190.5	198.6	203.5	204.7	205.2	Nev.
N.H.	265.7	278.0	296.2	279.0	269.8	269.1	N.H.
N.J.	120.7	125.1	128.4	120.2	121.4	122.3	N.J.
N.M.	90.0	92.6	99.3	98.9	100.3	103.1	N.M.
N.Y.	119.0	123.3	119.9	118.7	121.6	123.9	N.Y.
N.C.	172.4	187.6	214.3	226.5	227.3	226.0	N.C.
N.D.	93.8	98.5	103.8	108.7	110.5	117.9	N.D.
Ohio	121.6	124.6	124.4	120.5	122.1	122.5	Ohio
Okla.	108.4	116.4	121.7	124.1	130.5	132.9	Okla.
Ore.	157.0	157.9	160.4	155.3	155.8	154.4	Ore.
Pa.	107.3	106.3	109.0	110.7	114.2	114.6	Pa.
R.I.	123.9	123.7	134.4	142.0	136.1	154.7	R.I.
S.C.	103.6	115.0	118.7	125.5	129.7	130.5	S.C.
S.D.	92.7	96.7	103.0	103.5	108.4	113.5	S.D.
Tenn.	99.8	106.3	111.5	109.7	114.8	117.4	Tenn.
Tex.	106.4	108.9	108.6	110.4	114.7	116.0	Tex.
Utah	65.5	67.7	71.3	72.7	75.6	75.8	Utah
Vt.	122.6	124.4	138.0	146.8	151.8	155.5	Vt.
Va.	124.3	128.4	137.0	143.1	149.6	152.7	Va.
Wash.	96.7	97.0	88.5	91.0	98.6	99.5	Wash.
W. Va.	114.5	111.5	117.5	116.6	119.9	123.2	W. Va.
Wisc.	106.4	105.4	108.8	109.5	111.8	113.5	Wisc.
Wyo.	132.2	131.7	140.0	141.2	145.8	160.7	Wyo.
*All taxing states weighted average per capita	118.3	121.9	124.7	126.3	129.9	130.9	*All taxing states weighted average per capita
All taxing states (median)	119.0	119.8	123.5	121.8	125.6	123.9	All taxing states (median)
**National per Capita	126.7	132.4	131.6	135.1	141.7	134.9	**National per Capita

*Data for individual states are based on the total number of packages taxed.
The population figures used for the states are Census Bureau estimates as of July 1 of the respective fiscal years.
**Figures prior to 1960 are based upon federal dollar collections, from 1960 on figures shown are based upon "taxable removals."

RSV 0013762

Sierra Club
NEW JERSEY CHAPTER
360 Nassau Street, Princeton, N.J. 08540
(609) 924-3141

Sept. 24, 1976

Please reply to:

The Honorable David J. Bardin
Commissioner, DEP
Box 1390, John Fitch Plaza
Trenton, NJ 08625

Dear Commissioner Bardin:

The attached Newark Star Ledger article on Kin-Buc I raises questions that should be answered before any agreement on closing Kin-Buc I is signed.

1. Dike - will it be constructed; is its purpose and construction to keep the Raritan out, or leachate in, or both; how constructed; who supervises and inspects the construction; who is responsible for the dikes' long-term integrity and maintainance, and at what cost?

2. Clay cover - how constructed; who supervises and inspects; who is responsible for the long-term integrity and maintainance, and at what cost?

3. Leachate - if Kin-Buc is covered with impermeable clay, how long will leachate be produced? What is the leachate collection system, and leachate disposal procedure? Will Kin-Buc be responsible for collecting leachate and its proper disposal until it is no longer produced? If not, will DEP be responsible, and at what cost?

4. Groundwater contamination - a preliminary Geraghty and Miller report submitted to the Middlesex County 208 program shows that Kin-Buc I "has potential to cause significant pollution of the Farrington Sand member of the groundwater system serving Middlesex County." Who will be responsible for clean up, and at what cost, if contamination of the Farrington Sands is confirmed next month, next year, or ten years from now? It is known that drums containing hazardous and/or toxic wastes are buried in Kin-Buc I, and that such drums will probably leak in time. We have the Dover Twp. experience to keep in mind.

5. Performance bonds - should be required for closing implementation; another larger one should be required for the long-term, sufficient for all clean up costs, in the event of groundwater contamination.

We urge that the proposed agreement be circulated for review by all interested parties, including the Sierra Club, and that a public hearing be held on the proposal before it is signed.

Sincerely yours,

Diane

Diane T. Graves
Conservation Chm.

cc: Director Tylutki
Public Advocate
G. Hansler
R. Ricci
J. Zelikson
Middlesex 208

RSV 0013763

The Hazardous and Toxic Waste Crisis

Despite New Jersey's having the largest concentration of chemical plants in the nation, there appears to be little research, planning, and regulation to insure the proper disposal of toxic and hazardous chemical waste. New Jersey allocated only about $300,000 last year to regulate the more than three hundred sanitary landfills, and thousands of waste hauling vehicles within its boundaries. The Department of Environmental Protection (DEP) regulations on hazardous and toxic waste fill less than three pages and require only that toxic and hazardous waste be dumped at a landfill registered for such waste, that there be monitoring wells around the landfill, and that the landfill have a leachate control system.

For the first six months of 1976, the KIN BUC landfill in Edison was the only New Jersey landfill registered to receive toxic and hazardous waste. The forty acre KIN BUC I landfill on the bank of the Raritan River was receiving liquid chemical waste at the rate of 30,000,000 gallons per year when it was ordered shut by the DEP on July 18, 1976.

Numerous environmental problems were and are associated with the KIN BUC I landfill. Preliminary data gathered by the Environmental Protection Agency (EPA) showed concentrations of Benzene, a known human carcinogen, of 1,550,000 nanograms per cubic meter near the landfill. ("Clean air" contains no more than 50 nanograms of Benzene per cubic meter.) EPA data also showed a half dozen other known or suspected carcinogens in the air around the landfill. Leachate was observed spilling into the Raritan River. The Lower Raritan 208 Water Quality Policy Advisory Committee gathered data showing potential for significant ground-water pollution due to the presence of the landfill.

Despite all these threats to the environment and public health, the DEP was forced to rely solely on relatively minor technical violations to justify closing the landfill. If it were not for repeated fires, an excessively large working face, and various other relatively minor violations of the garbage-oriented regulations, KIN BUC would still be receiving toxic and hazardous chemical waste. In a sort of "compromise" KIN BUC has temporarily suspended accepting liquid chemical waste, but is still receiving solid waste.

Beatrice Tylutki, Director of the Solid Waste Administration, granted conditional approval to proposals for an eighty acre KIN BUC II and KIN BUC III site within two weeks of ordering the original KIN BUC closed. KIN BUC II is a landfill designed for hazardous and toxic waste. KIN BUC III is a reprocessing facility for toxic and hazardous waste.

A group of Edison residents called the "Citizens Committee to Close KIN BUC" banded together in July to try to halt any further dumping of hazardous and toxic waste at KIN BUC I or II.

Senator Bernard Dwyer and Assemblyman John Froude recently were able to obtain unanimous passage of bill S1493 in their respective houses of the legislature. This bill bans landfills located within 1,000 yards of a river flood hazard area from receiving hazardous or toxic waste. The bill is currently awaiting Governor Byrne's signature. One of the effects of this bill will be to keep KIN BUC I and II from accepting hazardous or toxic chemical waste.

The KIN BUC reprocessing facility is not affected by S1493. An environmental impact statement is being required by the DEP for KIN BUC II and III. A hearing will be held on the statement. Any members of the Sierra Club interested in studying the proposal for KIN BUC II and III or in studying and suggesting approaches to hazardous and toxic waste regulation are invited to contact Diane Graves at the club office or Frank Burke at (201) 572-2327. Especially needed are additional members with expertise in the areas of chemical engineering, geo-hydrology, soils, and civil engineering.

Frank Burke
Conservation Committee member

RSV 0013764

Sierra Club
NEW JERSEY CHAPTER
360 Nassau Street, Princeton, N.J. 08540
(609) 924-3141

Please reply to:

Sept. 7, 1976

The Honorable David J. Bardin
Commissioner, Dept. of Environmental Protection
Box 1390, John Fitch Plaza
Trenton, NJ 08625

Dear Commissioner Bardin:

I'm writing to follow-up on your request for names to be submitted as candidates for a task force on environmental and occupational carcinogens in New Jersey.

As you know, we first recommended such a task force in a statement presented to the Maguire hearing in May. I'm enclosing a copy of the applicable part of that statement.

We recommend the following people, listed alphabetically, for the task force. They have all agreed to serve, if asked. You'll note that the list consists of people who have specialized in different areas, and they represent a broad spectrum. We expect to add to the list shortly.

MSL 0052368

Seymour Charles, M.D. - Chief, Pediatrics, Newark Beth Israel Medical Center; member Environmental Health Committee, NJ State Medical Society; Associate Clinical Professor Preventive Medicine; President, Physicians for Automotive Safety; Moderator, Community Health weekly radio program - WNJR Newark.
(medical)

50 Union Ave., Irvington, NJ 07111, 201-926-1730

Albert Ehrlich, M.D. - Pathologist; Asst. Visiting Professor of Pathology at NJ College of Medicine; represents the Physicians Forum.
(pathology)

1 Horizon Rd., Fort Lee, NJ 07024, 201-224-6126

Barbara Eisler - President, N.J. Citizens for Clean Air; Chairman, Clean Air Council; member Clean Air Council (5 years).
(air quality)

212 Long Hill Dr., Short Hills, NJ 07078, 201-467-1310

Joan Goldstein - Ph.D Candidate in Environmental and Medical Sociology; Professor at CUNY, Bio-Medical Education Dept., Fellow, Resources for the Future '76-'77; Research Associate CUNY Graduate Center - Environmental Studies; former NJ State Health Manpower Planner; former Nat'l Coordinator Task Force Women and Health - NOW.
(sociology/ consumer)

E-14 Lakeview Terrace, Princeton, NJ 08540, 609-452-1796

"...AND PRESERVE THE NATION'S FORESTS, WATERS, WILDLIFE AND WILDERNESS..."

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Carter Henderson - Graduate in economics, Univ. of Pennsylvania, Wharton School; author, lecturer and advisor in fields such as health care, education, information handling, renewable energy, and resource recovery.

(economics)

60 Hodge Rd., Princeton, NJ 08540, 609-921-2280

Joseph Jadlocki - BA Biology; MS Environmental Sciences and Engineering, specialty aquatic biology and chemistry; with FMC Corp. since 1973 - Environmental Research Section.

(industry)

FMC Corporation, P.O. Box 8, Princeton, NJ 08540, 609-452-2300

Morris Joselow, Ph.D - Professor of Preventive Medicine; Director, Division of Environmental Toxicology, College of Medicine and Dentistry, Newark, NJ 07103, 201-456-4775

(preventive medicine)

Edward Lloyd - Staff Attorney for N.J. Public Interest Research Group (NJ PIRG); AB Chemistry, Princeton Univ.

(public interest/ legal)

32 W. Lafayette St., Trenton, NJ 08608, 609-393-7474

Louis Marinari - Vice President District 8 Council, Oil, Chemical and Atomic Workers International Union; Health and Safety Coordinator for the District; Sec'y of the Westville Local 8638 - OCAW.

(labor)

P.O. Box 291, Westville, NJ 08093, 609-845-4831

Grace Singer - Chairman of municipal Board of Health (5 years); Chmn. Environmental Health Committee of the Delaware & Raritan Lung Assn. and on Board (3 years); on State Lung Assn. Board (2 years); League of Women Voters air lobbyist; Research Assistant, Princeton Univ.

(health/ environmental)

6 Springwood Dr., Princeton Junction, NJ 08550, 609-799-0654

It has been suggested that my name be included on the list - (water quality). Please contact me or the people above for additional information.

Sincerely yours,

Diane T. Graves
Conservation Chmn.

cc: above list
Sean Reilly
Samuel Epstein, M.D.
The Honorable Andrew Maguire

enclosure

RSV 0013766

Appendix 11.

P U B L I C H E A R I N G

before

SENATE COMMISSION ON THE INCIDENCE OF CANCER IN NEW JERSEY

Held:
November 5, 1976
Senate Chamber
State House
Trenton, New Jersey

MEMBERS OF COMMISSION PRESENT:

Senator John M. Skevin (Chairman)
Senator Anne C. Martindell
Senator Joseph L. McGahn
Senator Barry T. Parker
Senator Wayne Dumont, Jr.

* * * * *

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Dr. Peter Preuss Department of Environmental Protection State of New Jersey	32

SENATOR JOHN M. SKEVIN (Chairman): Good morning. My name is Senator John Skevin. I am Chairman of the Senate Commission on the Incidence of Cancer in New Jersey. To my right is Senator Joseph McGahn from Atlantic County, of recent casino gambling fame. The other members of the Commission have other commitments on this particular day.

With today's hearing, I believe we will reach the conclusion of a preliminary stage in these proceedings, be in a position to issue an interim report to the State Senate, and submit for its consideration a series of legislative proposals designed to implement a counterattack against cancer in New Jersey. As previously mentioned in the past, these proposals will include: provision for enforcement procedures against known cancer-causing substances in the atmosphere and within industrial plants, banning consumer products which contain known carcinogens, and the creation of an early detection program. Dr. Harry Demopoulos, Director of the Cancer Institute of New Jersey, will be testifying in more detail on that aspect of the program today.

I do not intend this brief opening statement to summarize the necessary action which I believe must be taken by the State of New Jersey. These specifics will be detailed in the interim report to which I have referred.

The fact is, while a picture has been emerging throughout the course of these hearings concerning those aspects of the problem against which the State of New Jersey can act, another picture has been coming into focus concerning those aspects against which our State, by itself, is helpless.

For instance, while it is possible we could eliminate the emission into the atmosphere of all known carcinogens from sources in New Jersey, we can do nothing about those substances which enter our air from other states. Also, it seems equally obvious, we cannot expect to assume lone responsibility for the necessary scientific and medical research. Such an effort must be undertaken on the national level, and it is wishful thinking for anyone to feel that any individual state, with its limited resources, can shoulder this responsibility.

It would seem to me that in addition to New Jersey's forceful program designed to protect the lives of our citizens, we should consider the following:

(1) A coordinated, regional program which would set limits on all known cancer-causing substances and would provide the power to eliminate these substances from our atmosphere. This is an effort which requires more than good intentions and more than regional meetings with eloquent lip-service. I am suggesting that what we may well need is a regional cancer control authority to which each neighboring state grants the power necessary to enforce its standards.

If we can undertake cooperative, regional ventures to build tunnels, bridges and airports, why can't we cooperate in an effort to save the lives of our citizens? I would be interested in hearing the views of today's witnesses on this proposal.

(2) On the national level, I believe we need a commitment against environmental cancer to rival the effort which put men on the moon and a Manhattan Project. In an effort to provide impetus for such a program, I shall submit to the New Jersey Legislature, as part of our cancer program, a resolution calling upon the Congress of the United States to adopt in its own turn a "sense of Congress" resolution, which would label environmental cancer as a leading enemy of our citizens, and call upon the Executive Branch of the government to provide the leadership in a program intended to find a scientific solution to the problem. I believe such a total commitment, coupled with an effort to clean up the environment, could usher in a new era of accomplishment for mankind and would make a fitting endeavor for the new administration in Washington. Again, I welcome the comments

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of witnesses here today.

Now, let me reiterate what I have been saying all along. Here in New Jersey, we do mean business, and we intend to do everything possible to match our words with action. If necessary, we will start it alone because the battle must begin somewhere. However, as I have tried to indicate, we can also hope to set an example here in New Jersey which will inspire others, on regional and federal levels, into cooperative action.

Now we will have our first witness, Dr. Lewis Coriell.

D R. L E W I S L. C O R I E L L: Thank you, Senator Skevin. It is a pleasure to be here and I appreciate your inviting me.

I have a short statement which I will read which outlines some of the problems, as I see them, and some of the background and some of the suggested methods of attacking this problem.

First of all, control of cancer is a long-term goal and a long-term project and we shouldn't look at it as a short-term goal. Human cancer occurs in at least a hundred different forms, each of which has its typical age of onset, speed of growth, degree of malignancy, ease of diagnosis, response to known therapeutic measures, and sex, race and geographic distribution, for example. A few predisposing causes are known, such as cigarette smoke, a few chemicals, radiation, ultra violet, viruses in some animal tumors. But for most human cancers, we do not know the cause: even in those tumors where a predisposing cause is well documented, many other unknown factors are of equal or even greater importance. For example, cigarette smoke is the chief cause of lung cancer, and lung cancer is many times more frequent in heavy cigarette smokers than in non-smokers. Still only less than one of ten heavy cigarette smokers gets lung cancer. Obviously there are other conditioning factors. This is true of all cancer.

Solution of cancer will not be easy. It will require a prolonged effort. That is one of the main points I want to make. It presents a difficult and complex problem.

Excessive deaths in New Jersey may be due to increased disease, increased exposure. It may also be due to sub-optimal care. And I want to discuss both of those briefly.

Increased disease: There is no registry of cancer diagnosis in New Jersey. So we don't know whether we have more cases than other states. But, according to this Public Health Report which goes back from 1960 to 1969 - in other words, back a generation - we have more cancer deaths. New Jersey is densely populated and has an extensive chemical manufacturing industry. There is speculation, but no sound evidence, that New Jersey citizens are exposed to more of the predisposing causes of cancer than citizens of other states. We simply don't know how much and where the known carcinogenic chemicals that we do know about are used in New Jersey. More research is needed. I think we can discount that New Jersey citizens are exposed to more X-rays, more ultra violet, and more cigarette smoking than other states. These are some of the known causes of cancer: the other is speculation.

Now, a word about care: Cure of cancer is most frequent when diagnosis is made early and the best treatment applied immediately. Delayed diagnosis, inadequate treatment, partial treatment, lead to earlier and more frequent deaths. And the problems I see here, trying to look at all the problems, are these:

New Jersey, according to this Public Health Report, has the highest cancer death rate for that period of any State in the union. It is number one in cancer deaths for white males: number two,for white females: number six, for black males: and number five, for black females. New Jersey leads the nation for cancer of the bladder and urinary tract and is above average in cancer of the esophagus, stomach, large intestine,

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rectum, lung and breast. These are the big cancer killers. So it is a problem.

Some of the possible reasons: A survey we made in 1975 showed that the national average of physicians per hundred thousand population was 159: New Jersey was 126 per hundred thousand population; and South Jersey - the seven counties - where I come from, 92 per hundred thousand. That shows a deficit which we can explain by the next point.

We have not had medical schools in New Jersey. For the first 200 years of New Jersey, we didn't train any physicians. That is probably part of the reason. We started a school at Seton Hall in 1962 and, in 1970, that plus the Rutgers Medical School were taken over by the State. So they are still developing.

Since passage of the Conquest of Cancer Act in 1971, the federal government has launched a well-funded effort to control cancer through support of research in the causes of cancer, early diagnosis, cancer care, treatment, rehabilitation and education of the public and professionals for optimal application of the present knowledge. This was stimulated by the fact, as you mentioned, Senator Skevin, that cancer is the disease most feared by the American people: and this program was started in 1971.

New Jersey has gained little benefit from this national program because it has relatively few facilities and organizations that can compete for federal funding for the cancer research dollar. Take cancer research, for example. In 1975, New Jersey investigators received from the National Cancer Institute for cancer research grants, contracts and fellowships, seven and one-half million dollars: whereas, the six neighboring states - Connecticut, Massachusetts, New York, Pennsylvania, Maryland and Virginia - received five times as much on a per capita basis. To make it comparable - if New Jersey received its proportionate share of research contracts and grants, it would have received $37 million or $38 million, or $30 million more.

It should be observed that the National Cancer Institute cancer funds are not allocated by formula or by need, but rather on the basis of competitive applications which present the best prospects for completion as judged by the competence of the investigator, his plan, his facilities, his equipment, his local support, and his reputation as a good cancer researcher. So, New Jersey did not get much of that money put into cancer research and control over the past five years, and the reason is obvious. There are not many established cancer researchers with facilities and backing and local support in our Medical Schools and other parts of the State. Those grants are given on the basis of who presents the best application.

The fifth point I want to make is that we have had in the past an inadequate plan or efforts to coordinate cancer care, including early diagnosis, registry, multi-disciplinary care, follow-up, rehabilitation and education. New Jersey has not tried to do this in the past.

So, historically, New Jersey has provided inadequate State support and encouragement for solution of any of these problems I have just listed, nor has New Jersey taken official notice of the Federal Cancer Center programs nor asked for help from the federal government, industry and the public to help solve this problem.

In summary, together these unsolved problems mean that many New Jersey residents possibly have received less than optimal care, perhaps delayed diagnosis, delayed treatment, inadequate initial treatment and follow-up, all of which must be a major factor in the increased cancer deaths in New Jersey.

Environmental exposure is undoubtedly a factor in some types of cancer. But, in my opinion, this too is made worse by historical difficulties in New Jersey.

As to the solution, I think we have taken one step already by recognizing that we have perhaps an unsolved problem. It is called to mind by this Public Health Report of

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the Incidence of Cancer Deaths. I am sure that there are many defects in those data - reporting, recording, transcribing and the lack of a cancer registry. It may be higher than that: it may be lower than that in New Jersey. We have to go with what we have there. But, presumably, we do have an unusual problem for whatever reasons: and this is being recognized now by these hearings and others. Everybody is painfully aware of this problem and wondering what to do. I think responsibility for correction is ours and I congratulate you on having the hearings. And, as Lincoln said about some of the problems of his day, "We," and I quote, "even we here, hold the power and bear the responsibility." So I am pleased to contribute whatever I can to this.

The recommended State actions that I would think of - and others may have others - are:

Number one, adequate support for New Jersey Medical Schools, including the third medical school in South Jersey, to which a commitment has been made, to train more physicians and have centers of excellence for outreach into the community hospitals. This includes undergraduate and specialty training of physicians as well as paramedical sub-specialties required by modern medical practice, on-going education of physicians, and outreach programs to the community hospitals. Eighty percent of the cancer cases treated in this country are treated in community hospitals, not in big medical centers. So one of the problems of having the medical centers of excellence is to reach out and help bring the quality of care up in the community hospitals where most of the patients will probably always be treated. Talking to Lee Clark, who heads the M. D. Anderson Cancer Center in Houston, probably one of the best and most well organized, he says they treat only 9 percent of the cancer in Texas. He told Dr. Demopoulos recently that he thinks New Jersey has a wonderful opportunity to really do something by trying to coordinate the whole State and all the centers of excellence in New Jersey into a coordinated program. I think that really is a great opportunity we have and it is possibly easier than where you have established large independent medical centers, in trying to get them to cooperate.

Number two, I would recommend stable support for expanded cancer research and facilities. Senator, you mentioned that this is really the responsibility of the national government to support cancer research. I would like to give you my feeling on that. The reason we fare so poorly in getting our fair share of cancer research is that New Jersey for whatever reason, whether it is State or local government, has not provided a stable base for enough cancer investigators - high quality people. They need a stable salary for themselves, so they feel secure in building up a team of investigators, who then bring in many more times their cost in grants and contracts, and train additional people.

So, when you go to NCI, National Cancer Institute, with a grant application and say, "Give me money to help solve our problem in New Jersey - we haven't done anything in the past, but now we are ready, if you will support it," they will say, "What sort of backing do you have at home?" This is what I am saying - we need to put something into it.

The Research Committee of the Cancer Institute of New Jersey, of which I am the Chairman, recommends that the State of New Jersey encourage its cancer research capability by establishing eight to ten cancer research professorships in various institutions within the State. These are expected to attract many times their cost in additional federal and foundation research grants and other research personnel, who will, together, attract New Jersey's share of the National Cancer Research support. You don't solve cancer in one or two years. This has to be stable support.

RSV 0013772

The third recommendation is support of a comprehensive plan for cancer care in New Jersey. The National Cancer Act of 1971 postulated that eventual control of cancer will come from research. But, in the meantime, many cancer patients do not receive optimal care that is now available. The Act, therefore, provided for the establishment of a series of comprehensive cancer centers across the United States where all the latest information and skills would be available to citizens of that region. In five years since 1971, 18 comprehensive cancer centers have been funded and 3 more are authorized under the present legislation. Funding is authorized up to $5 million for each center. To qualify, a center must show implementation of a plan to provide its region a comprehensive attack on cancer. This includes basic and clinical research, public and professional education, and patient care. Optimal patient care includes both the delivery and the teaching of others to deliver or provide for early diagnosis, a cancer registry, multi-disciplinary management of each case, follow-up and rehabilitation.

Organization of each of the 18 comprehensive cancer centers that have been funded varies from a single institution, in many cases, like M. D. Anderson which I mentioned, to a consortium of institutions. And this depends on the region and what facilities are there. You build on your strengths when you do something like this. In each case, however, a prerequisite to funding was a submission of convincing evidence that a rational plan of action had the strong backing of concerned groups within the region, competent professional direction on hand, with stable support, and stable local financial support. These are really the key. Then when you go to the national groups, they can say, "They are serious about this. They have already put up some of their own money to make sure that the direction and the basic support are stable, and it is not going to fold up in one year. So, if we put some money in that, it would probably be well used."

New Jersey is now preparing a request to the National Cancer Institute for designation as a comprehensive cancer center. The application is being prepared by the Cancer Institute of New Jersey, a non-profit organization developed for that purpose. It has a Board of Trustees representing academic, industrial, research, medical groups, and a full-time Medical Director. Initial support has been provided by contributions from industry and foundations; and many professionals have contributed their time and advice, including representatives from the American Cancer Society, the Department of Health, Medical Schools, Research Institutes, hospitals, physicians, industry - and the pharmaceutical industry, which is very strong in this State, has a very strong research base and they are working with our Research Committee.

CINJ has been awarded an initial planning grant by the National Cancer Institute and two supplementary planning grants have received site visits, one, to coordinate cancer delivery of care in seven South Jersey counties; and, the other, radiologic therapy, coordination of x-ray and radiologic therapy of cancer of a number of hospitals in North Jersey. So our appeal has been answered and been awarded. Those small grants are inadequate and we need to move on and organize ourselves so that we can ask for more support to be designated.

I strongly urge that the CINJ (Cancer Institute of New Jersey) efforts to upgrade cancer care in New Jersey receive some stable support from the State to supplement its other sources. Allocation of such State funds should be by a commission that is competent in science and management skills and free of political influence, because cancer attacks saints and sinners alike, and partisan politics has no role in evaluating the scientific merit of a research project or the treatment of a given cancer. Both are long term and the stable support needs to be set up so that it can follow whatever is

RSV 0013773

best scientifically.

Secondly, the incidence of some types of cancer may be increased in New Jersey by excessive exposure to environmental carcinogens. This requires additional research and proof - again, the research element. To qualify for this federal help, the region must compete with other regions and show that it has a superior plan, staff, capability, facilities, and commitment by all involved factions of society.

For all of the above reasons, I strongly urge that bills be developed, which, when approved, will provide stable, long-term financial support for cancer research, both basic and clinical, a cancer registry, and a comprehensive cancer control program. This is the beginning.

If the State will provide basic support, I feel sure its contribution will be matched many times over from other sources, including industry, foundations, private sources, and federal funds. Some support has been received from each one of those already within the last year.

Finally, in this way, cancer deaths in New Jersey will be reduced and eventually controlled as research gradually develops new and more effective methods for early detection, treatment and prevention. I thank you.

SENATOR SKEVIN: Thank you, Doctor, for a very fine and comprehensive report.

Before we begin with the questions, I would like to welcome Senator Martindell, a recent arrival on our Commission. Senator Martindell represents the other side of the casino gambling question - no dice.

Dr. Coriell, I had the pleasure and the privilege to be with you at your Institute for Medical Research where you have a very fine and excellent program. Could you tell us whether there is any research either by your group or any group that would be in the area of a cure? You mentioned early detection as a cure. But is there research in the direction of a cure,or immunity, or that type of thing?

DR. CORIELL: Well, there is research on all aspects of cancer. Early diagnosis and treatment do cure more and more cancers. The earlier you get it, the better chance. The statistics on breast cancer are, if it is confined to the breast at the time of surgery, there is an 85 percent cure with simple surgery. If it has already metastasized to the axillary nodes, it drops to 50 percent. And, if has passed beyond the axillary nodes, it is 10 or 12 percent, five-year cure. Cure means the cancer has occurred, you detect it, and it doesn't ever come back. We are getting some cures now in leukemia, which used to be fatal within six months for small children. There are cures now in osteo-carcinoma, in Hodgkin's disease. Prevention is our eventual goal. Once you have the diease, then we go through this surgery, chemotherapy, radiation, and most people with these serious cancers still die of the cancer down the road.

So the eventual goal is earlier detection with more effective methods, where you don't wait until you have a big lump, big enough to feel. Probably the incubation period of cancer from the time it starts is ten or twenty years before it is diagnosed. We should be able - and we are working at our Institute on earlier methods of detecting. The body realizes that cancer is there and there are ways in immunology, perhaps, to test this by skin tests and that sort of thing. To answer your question, we still have a long way to go. Most people who get cancer are still dying of cancer, although inroads have been made in the last five years, very encouraging ones.

SENATOR SKEVIN: Your research in terms of immunity - are there any developments in that area? Is there any activity in that area?

DR. CORIELL: Tremendous activity, Senator, in that area. One of them is clinical research, combining surgery, chemotherapy and x-ray, with non-specific immunal

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therapy. It was observed that, in guinea pigs, you can cure some of them of guinea pig tumors by vaccinating them with BCG, which is TB vaccine. It is the live vaccine that they use in many other countries more than here. But it causes an infection. It is an attenuated strain of the tubercle bacilli. It does raise the general immune mechanism. Thousands of people with cancer, which has not responded to all the things we know, are now receiving, in addition to chemotherapy, BCG vaccination once a week or once every two weeks, because of the guinea pig response. The response in man is open to question. Some studies seem to show improvement - others, equivocal. It seems to do something: it is obviously not the answer. It is not a panacea.

More specific immunization, taking the neoantigen from the tumor and immunizing people with that - there is much work being done in experimental animals. There is still no break-through there. In our Institute, we are working on breast cancer that is caused by a virus in mice. We can immunize the mice with a vaccine and prevent breast cancer. We find traces of evidence for the same type of virus in human breast cancer, but we are not convinced yet that we have shown there is a virus in human breast cancer. Other people are not convinced we have shown it. But there is a great deal of evidence for it. Down the road, there might be something we could learn from the mouse study for earlier detection, which would be a help in itself. Dr. Cole told us last week that he thinks breast cancer in humans, the number one cause of death by cancer in women, probably in the majority starts in puberty, in the teen ages, and it is that many years before they get cancer at 30 or 35 years of age. The circumstantial evidence, the epidemiologic evidence, that we have suggests that something happens there, because the incidence is related to age of the birth of the first child and age at the onset of menstruation. Many of the factors seem to be occurring there.

So there is great possibility of advances in immunology. What you are thinking of there is: Could you prevent it entirely so you don't have to go through all this later treatment? Yes, that's the goal of many people.

SENATOR SKEVIN: And the research is in that direction?

DR. CORIELL: There is a great deal of research in that direction.

SENATOR SKEVIN: Going back to your testimony, Dr. Coriell, about the loss of federal moneys or federal grants, I recall that you mentioned something in the area of some thirty-odd million dollars that would be available if we had matching funds?

DR. CORIELL: Not if we had matching funds. You have to compete for these funds. I or Dr. Demopoulos or anybody who puts in an application to do some research for federal funds must compete against every other investigator in the United States, who also sends it to the same place. If it is on the same subject, it goes to the same committee. The Committee looks these over and they say, "Well, Dr. Demopoulos has done excellent work. He has a good place to work. He has good facilities. He has all the centrifuges he needs. He has a good back-up team. He even has some stable support from the State or from the country and a local United Fund is giving him some money. So he's solid. He has ten graduate students working with him. We think he could do a better job of this than Joe Blow out in Berkeley, California. We will award it to him." That is the way you get funding.

The obvious reason we don't compete with the other six states on both sides of us is that we don't have enough good people established in New Jersey, who can compete for this. We have some excellent people, but it is not comparable considering the number of people in the State on a per capita basis.

SENATOR SKEVIN: As a result, we have lost some thirty-odd million dollars. Is that what you are saying?

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DR. CORIELL: Well, we haven't really lost them. We haven't qualified.

SENATOR SKEVIN: You haven't qualified for them.

DR. CORIELL: We haven't qualified for them. They are available. The National Cancer Program has been increasing the amount of money that it is putting out for this purpose since 1971. This year Congress passed a bill for $815 million; last year, it was $762 million. So they upped it a little. It will at least take care of inflation, probably not much more. But, anyway, each year it has been increased because the national legislators are convinced that this is a serious problem and that putting more money into research and patient care, together, will help solve the problem down the road. So they are committed to it on a long-term basis.

We, in New Jersey, have not made that commitment in the past. The people who are doing research in New Jersey are doing it on their own.

I must thank the New Jersey Legislature for supporting my institute, the Institute for Medical Research. For eight years, New Jersey has appropriated some funds each year to help cancer research at the Institute for Medical Research in Camden. They dropped that last year in all the cutting. I hope they will put it back. I am grateful for that. That is one example and I might point out, right there, we get from national sources five dollars in grants and contracts for every dollar the State gives us. That is what I am saying. That's where the $30 million went. You didn't ask for it - you didn't qualify.

SENATOR SKEVIN: We didn't qualify for it?

DR. CORIELL: Right.

SENATOR SKEVIN: Dr. Coriell, I have used as a guide your excellent booklet, "Cancer and Cancer Research in New Jersey." With your permission, I would like to make that book a part of our hearing record today.

DR. CORIELL: You are welcome to. I brought some copies if you want them and I also brought a copy of a report that I helped write in 1968, pointing out the need for additional medical education. There are a number of statistics in there which haven't changed much in the eight years since then.

SENATOR SKEVIN: If you will hand that to the stenographer, we will make that part of the record.

("Cancer and Cancer Research in N.J. - A Report by the Research Committee of the Cancer Institute of N.J." and "A Medical-Dental School in South Jersey", submitted by Dr. Coriell, on file with the Committee.)

Dr. McGahn, do you have any questions?

SENATOR MC GAHN: No.

SENATOR SKEVIN: Senator Martindell?

SENATOR MARTINDELL: No questions.

SENATOR SKEVIN: Thank you very much, Doctor.

I will next call Dr. Hammond.

D R. E. C U Y L E R H A M M O N D: Senator Skevin, thank you for inviting me down here.

I have had long association with your Health Department, which has helped us with our research and many other people in New Jersey. It is certainly a pleasure to do anything I can to help in return for this.

I should say a little bit about my background, which will explain what I am going to say that after that. When I was in Graduate School at Johns Hopkins, my thesis work was on change in environmental conditions in relation to longevity, death rates and energy. It happened to be on fruit flies, but I was interested in man. When I got out of there, my first professional job was in the Division of Industrial Hygiene - it would now be called Occupational Health - in the National Institutes of Health. After the war, in 1946, I came with the American Cancer Society and I have been working

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primarily on environment interrelation to cancer ever since: and Vice President for Epidemiology and Statistics of the Cancer Society. We have what you might call a cooperative research project of the Society, the major collaborators being Dr. Irving Selikoff in Mount Sinai Medical School in New York, a resident of New Jersey: and Dr. Oscar Auerbach in the Veterans Hospital in East Orange, New Jersey. We have jointly what we call the environmental cancer research project and this is supported partly by the Cancer Society. My own work is supported entirely by the Cancer Society. But we also get support from the major government agencies - NIOSH, the National Institute of Environmental Health Sciences, NCI, and other groups.

I say that because,from that background, you can see that almost all of my interest of my professional life has been environment interrelation disease, not just cancer. For a very long time, this was a seriously unsupported endeavor. There was extremely little support at the state and local level; and even in medical schools about the country, this was a field seriously neglected. It is only in recent years that there has been enough public interest aroused to make the public aware of it and get the support we need, not just financial, but general public backing to carry on this work.

Now, with that statement, you may be a little surprised what I am going to say next. A thing that worries me is that in many instances we have had newspaper headlines on some terrible thing happening. Everybody gets excited. We suddenly get support from private or local sources or from the government. Then this dies down. It is almost a fade. A fade one year for one thing and another year for another thing. As the previous speaker said, this unstable support - feast or famine - is not going to help us solve the cancer problem in this or any other state. What is necessary is a realistic view of the fact that, to solve the cancer problem, we are going to have to have both public and private adequate support, and stable support over a long time.

In New Jersey, a lot of people have been worried about this book of maps brought out rather recently by the federal government, the Epidemiology Branch. I think everybody here must be well aware of it. These are the maps which have been so much quoted speaking of New Jersey as the worse place in the United States to live, or words to that effect. This wasn't said by the authors of the book, but it is the way it has been interpreted by some people. In fact, you get the impression some people think this is such an unhealthy place to live that you certainly don't want to move into New Jersey. And this is not doing your state one little bit of good in public reputation in the country.

SENATOR SKEVIN: Is that the Cancer Atlas that you are referring to, Doctor?

DR. HAMMOND: It was the Cancer Atlas that gave rise to this. Actually it was a previous report. I have a copy here. I expect you have one. But then, what was done with this by the press, was some scare headlines.

I would like to point out that this scare attitude, which is fine in so far as it will give impetus to legislators and private individuals to see the problem, is going to backfire unless it is put in proper perspective. It will only be a flash in the pan. The proper perspective is this: They say, if you want people to believe you, they will look at the way you act, not just what you say. I happen to live in New York. I am not far from retiring age and my wife and I are looking for a house in New Jersey. We intend to move here. I think that is the best indication that I am not scared of this environment. I wanted to put it on the record to allay some fears. For that matter, we have three lovely little grandchildren. They are living in New Jersey and I am not one bit worried about that fact. I would like to put that on the record.

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Let me explain what this book,which has caused so much commotion here,actually shows. It shows, and the study back of it, that in the period 1950 through 1969, death rates, particularly among white males, were among the highest in the United States. You can pull out certain pieces of it and get New Jersey to be the highest. This is what has been pulled out.

As the previous speaker pointed out, from everything that we know that causes cancer and from everything we know about animal experiments, it takes around anywhere from 20 to 50 years delay time from first exposure to those things that eventually lead to cancer until cancer occurs. What this is saying then is that somewhere between about 1900 and, oh, not later than about 1950, people living in New Jersey in this later period had at some previous time, 20 to 50 years earlier, been exposed to something that led to their cancer. I am not saying what that something is. There are many different things. There are many forms of cancer.

In so far as this applies to those who are living in New Jersey, it is reflecting conditions in the first quarter of this century, perhaps in New Jersey. We know some of them were here then. But, in this period, you also had a very large migration from other states and, for that matter, from other countries, particularly in Northern New Jersey. This data doesn't give any impression whatsoever that living in New Jersey is less healthy than living in any other state in the United States. It is conceivably so, but this doesn't say so. It is not even suggestive of that.

Now, having said that, you may get the impression that I am not for doing anything about it. This is far from true. I am most enthusiastic about doing something about it and I would like to say what this does show from another standpoint. Quite regardless of why people living in New Jersey still have a very high cancer death rate, it means you have more cases of cancer per capita than most of the rest of the country. This varies with different sites of cancer. You are in the upper level of a problem. Regardless of the cause of that problem, this is a problem that is with you. New Jersey more than many other states has a caseload of patients, people who are going to develop cancer, and, therefore, you have more of a problem of trying to prevent it here and more of a problem of patient care and treatment.

I am not going to repeat everything the previous speaker said. But I am really going to try to reenforce what he said. In my opinion, a much greater effort in cancer control than has previously been mounted in New Jersey is warranted. I also agree with his opinion that, if you do so, you will get help from outside of this State from federal sources. He has mentioned some of the things, one being a cancer registry. Twenty years ago, I tried to get New Jersey to set up a cancer registry. This is no new thing. There is more reason to set it up here than in most states - and this was apparent twenty years ago. Unfortunately, not enough support could be developed to bring this about. I agree with the previous speaker that this is one of the first things that this State should do. I would like to be a little more specific and make a suggestion on it.

The model of cancer registries on a statewide basis in this country started in Connecticut. It was started with state support, but on a voluntary reporting plan. It was actually initiated by the State Medical Society who then petitioned the state government to put up funds for it. Interestingly enough, a few years ago, Connecticut was the only state in the country where reporting of cancer was not required by law. It was the only state in the country where cancer was almost 100 percent supported. I say that because legislation, in itself, is not sufficient. There also has to be voluntary compliance in building up a background for it. But what they did was to encourage

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reporting of cancer and follow-up of cancer patients by the state appropriating a certain amount of money and also setting up an office to keep the records. This money was then allocated to the hospitals of the state on the basis of what they were doing in relation to cancer. One of the most important things for the proper treatment of cancer is proper diagnosis and this requires a histologic proof that there is cancer. This is a piece of tissue taken from the cancer or that suspected of being cancerous, which is then studied microscopically. Once the patient has been treated, he must be followed periodically because, if there is any recurrence, his life and his comfort, even if his life can't be saved, will depend on getting him back for treatment. This was woefully lacking in Connecticut before this registry started. It is, I am afraid, quite lacking in Connecticut today. What they did was to take the state funds and give a certain amount to each hospital for each biopsy and histologic specimen that they did. They paid for each follow-up - the money was divided. Now, this brought in a lot of private money as well as the state money. This was a stimulation. It was enormously beneficial. It attracted better men to Connecticut. The whole level of cancer care in Connecticut was brought up by this procedure.

Now, I am going to say two things critical of Connecticut because I am making a suggestion that to some extent you follow Connecticut, but, in another respect, depart from what they did. New Jersey at about the same time was, I think, the first state that set up a slide registry. This is the microscopic slides on cancer patients. They were not sent in, unfortunately, as well as they might have been. It wasn't well enough supported. There wasn't enough impetus back of it. But it was one of the best teaching collections of slides in the United States. Frankly, I have had no contact with it recently. It was perfectly splendid at an earlier date. Those two ideas should be combined. They did not do that in Connecticut. So the slides were not available for research.

I would suggest, respectfully, that New Jersey set up an excellent cancer registry, but with a slide program that you led the country in at an earlier date, combining those two ideas to make it much more useful for research.

They also made another mistake. What they did was have the registry under the Health Department of the State and they never allocated, after spending a great deal of money on the total registry, enough funds to attract a good man to run it. On a clerical basis, it was excellent; on a control basis, excellent. But they were limited in their salaries and they were not in a position to attract the top men they could and, therefore, I don't think they have gotten 10 percent out of it that they should have gotten out of it in terms of research. The Cancer Society offered to supplement it. We offered to pay for it and get money privately. But they would never let it out of the control of a situation where they couldn't do it professionally. They have now, finally put it for research purposes under a professor at Yale, with cooperation between the two.

I would suggest, sir, that if you take the suggestion that I have made and the speaker before me, you get the cooperation of one of your Medical Schools, preferably with an epidemiologist and a pathologist, to give the registry the professional attention it needs. If you do that, it should be splendid, both from the standpoint of benefiting the cancer patient here and now, on the one hand, and, on the other hand, making it useful for research.

I fully agree with what the previous speaker said concerning the general cancer control activities and getting federal support for them, which you can get if you put in the seed money, the stable money. He said it better than I could say it concerning these matters, so I won't repeat it.

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I would like to emphasize one thing. The control of cancer, at least in the United States, is going to be dependent upon close cooperation between official and unofficial agencies and individuals. The support and cooperation of the medical profession is required, not just orders, but their wholehearted voluntary cooperation. When it comes to the cancer education side of it, you have in this State one of the very best American Cancer Society Divisions in the country. They are doing a splendid job of cancer education and, since it is done by volunteers, the cost is trivial compared to what the State would have to pay to do the same job. And I think the very fact it is being done by volunteers makes it more effective than this aspect would be if it were taken over by the State with State funds or otherwise.

Now, could I turn briefly to the other side of this; and, that is, the research aspect. My own interest is more in research in environment than anything else. In the past, New Jersey - with, granted, inadequate funds and no medical schools - nevertheless was a leader in this field; I mean, residents of your State and corporations in your State. I wasn't speaking solely of the State government. For example, the first, and magnificent piece of work, on epidemiology of cancer was undertaken by a Mr. Hoffman who was the Chief Actuary of the Prudential Life Insurance Company in Newark. They put in a lot of company money on it. This was New Jersey money and New Jersey effort. This got us started on the type of approach that this book shows - a magnificent piece of work.

Some of your companies, but certainly not all of them, have also done it. One of the greatest efforts in the field of trying to prevent industrial cancers was carried out by what was at that time, Standard Oil of New Jersey, in their research laboratories here. This was one of the first companies that tested each new product that they were considering producing on experimental animals to see whether or not they were carcinogenic before exposing workers to them. Mind you, they made some few mistakes; everybody does. But they are to be congratulated for leadership in this field. This is again New Jersey. What they did,did not benefit just New Jersey because this is a worldwide company. As you know, they have refineries and many of them are here. They were leaders in that.

Sadly, some of your other industries tried to sweep problems under the rug. But that has not been just in New Jersey, but all over the country.

One of the most successful laboratories in the country in cancer research has been the work carried on by Dr. Oscar Auerbach in the East Orange Veterans Hospital. I will refer to what was said about support. The American Cancer Society gave him a small grant, really quite small. And we put it to work. As a result of that, the Veterans Administration put in several times the amount of money. Now, they say, money begets money; and excellence begets excellence. In the research field, if you do not give basic, long-term support in a way that you can attract a corps of good men here, you are not going to get support from the federal government or private donors. That is the key to it, as was said by the previous speaker.

Are there opportunities here? Yes, there are. This was really the first industrial state in our then young nation, and, particularly, around Paterson much of the industry developed. Along with industry with all its benefits, you also do have some risks. This is certainly true in New Jersey. If we look at the environmental causes of cancer, one of the best techniques to do so is to determine what has happened to people who have been exposed to various substances in industry. You may know the work Dr. Irving Selikoff and I have done on asbestos from that awful plant that operated in Paterson some years ago. This was during World War II. We are trying now to find out whether the dust created around Paterson within about a half a mile radius of the plant was sufficient to cause cancer in

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the general population other than the workers. For the workers, it was disastrous. We are tracing everyone who lived within a half a mile of that plant during the time it was open during World War II. All accounts indicate that there was terrible exposure to the general population in that area. You can still find asbestos dust in attics and places that haven't been cleaned since then. It is still present - it was so heavy.

In this case, we know there was a huge hazard to the exposed workers. The question is whether this carries over to the general population to a lesser degree. This is a national problem. It was a specific problem in Paterson, but it is a national problem as to whether we must make strenuous efforts to reduce the possible exposure to asbestos which occurs in every city in the United States. This is the best laboratory, as it were, to test it. We don't know the answer yet. This same sort of thing which we may call local air pollution can occur in the vicinity of almost any industrial plant. And, depending upon the nature of the substance, whether it be a chemical, and the wind direction, it can spread over a wide area. The techniques to find out the possible hazards are first to study the people heavily exposed, and these are people who are occupationally exposed. If heavy exposure doesn't hurt them and does not result in later cancer, then we need not worry about that substance for the general population. If it does, then we need to be worried about it. So we are studying industrially-exposed groups, both for themselves - obviously we want to prevent industrial cancer - and also for the general public as well.

I would like to make one suggestion as to this, besides that which you are doing. Incidentally, the State Health Department here has been enormously cooperative with us for years. They have made possible studies we have done which we could never have done without them, and I would like to thank them. But one of the problems is that while there are some companies that are very cooperative, there are other companies who try their level best to keep investigators from getting information to find out whether or not there is a hazard and to see what can be done about it. My own feeling is that this is very foolish. From a purely selfish standpoint on their side, they are hurting their workers and eventually the law suits are going to be far worse than anything else that could happen. To my mind, it is utterly stupid. But there are some stupid people in this world. I would think that legislation in this State which would make it possible, with or without the consent of a company, to get information from company records and make tests in those plants as to what the exposures are so they can be studied, would be the greatest contribution that New Jersey could make to the cause of cancer control for the country and the world as a whole. I think, in doing this, you would have to put in some safeguards and not just have anybody walking in any plant. There would have to be safeguards so it would be confined to competent, qualified investigators.

My guess is that nine times out of ten, the company will be glad you did it because it will be found that there is no hazard. If there is a hazard, then the workers and humanity should be glad you found it out. I think this is possibly the greatest contribution you could make in thia State. Thank you, sir.

SENATOR SKEVIN: Thank you, Dr. Hammond, for a very fine and comprehensive report.

I would like to welcome here, Senator Parker, a member of our Commission, who is sitting to my left.

In your testimony, Dr. Hammond, are you really telling us the statistics reflect exposures 20, 30 or perhaps 40 years ago and that these people were doomed in terms of the cancer involved by that early exposure?

DR. HAMMOND: That's right. Let me give as an example this very terrible asbestos plant in Paterson. The people who worked in that plant in later life had 8 to 10 times

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the rate of lung cancer that the general population had. They also had a very high rate of two related diseases that simply hardly ever occur in anybody but asbestos workers - pleural mesothelioma and peritoneal mesothelioma. They are horrible diseases - both absolutely incurable. There is nothing you can do about them. They also had asbestosis. This is not cancer, but that didn't help them much because it kills them. Far before it kills them, it disables them.

Dr. Selikoff and I traced these people from the time they were first exposed. We were able to get a list of every man in the plant and a number of them had been Dr. Selikoff's patients in the early days. We traced all of them. You couldn't see any appreciable increased risk of cancer until roughly 20 years after the time they were first exposed. Now seeing that and looking back, well, you can see maybe there was a slight increase earlier. But, as far as determining it, practically speaking, it was 20 years up to 30 years. In some other situations where we have been able to examine people longer, it persists for as much as 50 years after inital exposure.

So, what I was saying is that in so far as these cancers occurring in the period of this report in New Jersey were caused by the environment - some of them may have been virus - these are a result of things that happened, as I said, 20 to 50 years earlier. Some of those people were living in New Jersey and I know that some of them were due to occupational exposure in New Jersey. Many of them were living in Jamaica, South America, Alabama - you name it.

If you take this report, second to New Jersey in cancer mortality in this period reported here among white males was the District of Columbia. The District of Columbia simply had no industry at all during the period 20 years earlier. It didn't exist. There is hardly any now. The residents of the District of Columbia were, of course, from all over the United States and it wasn't things they got in the District of Columbia that caused that high cancer rate. For some reason - it is hard to imagine why - there must have been some selective migration of people with this exposure into the District. So you can't take these things too seriously.

I will say about the District of Columbia the same thing I did about New Jersey. From the standpoint of the problem of caring for the patients and providing them with hospital beds, surgery and treatment, it doesn't make any difference at all why this occurred. It still means the District of Columbia has a problem, although the cause of it might have been due to somebody else's neglect conceivably. So, in both cases, from the standpoint of treatment, early diagnosis and cancer registry problems, you had the problem regardless of the cause.

SENATOR SKEVIN: In terms of environmental factors, exposures to cancer-causing substances, what you are saying then is we really couldn't be effective in that area, if we started today, until perhaps 20 to 30 years from now. Is that what your conclusion is?

DR. HAMMOND: That's right, in so far as we know from all our experience. There may be some exceptions, but we just don't know anything about them at the present time.

SENATOR SKEVIN: Dr. McGahn.

DR. MC GAHN: No questions.

SENATOR SKEVIN: Senator Martindell.

SENATOR MARTINDELL: No.

SENATOR SKEVIN: Senator Parker.

SENATOR PARKER: Just one question: Is there any correlation in any information you have between the high exhaust emissions and the incidence of cancer from automobiles? I am thinking of Washington, D. C. and the Paterson-New York area.

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DR. HAMMOND: Yes, I do have information on it. Extending the study, I can tell you what we have up until this point. In 1959, we enrolled through volunteers of the American Cancer Society 1,078,000 men and women, all at that time over the age of 30, most of them over the age of 45. When they were enrolled, they each answered a detailed questionnaire, giving their family history of cancer, what their parents died of, their physical complaints, their habits, their occupation, etc. We also knew where they lived. We knew their residence histories and,from federal sources and city sources, we were able to classify these 1121 counties by various indices of air pollution.

When you know one thing is important as a cause of cancer, then you have to control on that before you look at things that may be of lesser importance. In this study, we knew their smoking habits, so we could analyze the data, holding smoking habits constant -- I mean, comparing people with the same smoking habits. I can say that up through 1965, we found no correlation whatsoever between lung cancer death rates in any measure to degree of air pollution, even in Los Angeles County. We also, however, had to control on occupational exposures. In Los Angeles County, they did have a slightly higher rate of lung cancer, but this was confined entirely to people who had occupational exposures, which raised the rate by about 13 percent or thereabouts. Since, again, growing air pollution over a long period of time could have increased with time, we have now followed the people for another 6 years. We will reanalyze the data and see whether the earlier conclusions have in any way changed.

SENATOR SKEVIN: Thank you, Doctor.

Dr. Harry Demopoulos, Director of the New Jersey Cancer Institute.

D R. H A R R Y B. D E M O P O U L O S: Good morning. I would like to thank you for inviting me. I think when these proceedings are finished sometime in the future and you look back, you will probably find they are among the most significant events taking place in the war against cancer. I think your proceedings here are a logical continuum of the national cancer plan: that is, an attempt to develop legislative programs at a state level. I want to thank you for inviting me.

As the Director of the Cancer Institute of New Jersey, we are developing a comprehensive cancer center program, which is statewide. Now, there are two things in that statement that need definition. A comprehensive cancer center-- Dr. Corirll has touched upon the definition and Dr. Hammond has described some of the many events that take place in a comprehensive center. I would like to amplify what the Cancer Institute of New Jersey is, so you may have a more clear idea about what kinds of legislative programs are necessary. In order to do this, it is necessary to discuss what is a comprehensive cancer center program. These have been, in part, defined by the National Cancer Institute and the National Cancer Advisory Board when cancer centers were set up under the aegis of the National Cancer Act in 1971.

A comprehensive center means just that. It is supposed to attack every facet of the cancer problem. I know of no other disease wherein the disease is so completely intertwined with the fabric of society. It involves molecular research. It involves business, industry, money, legislators and long-term support. And, to try to attack the cancer problem piecemeal is not very productive. You must attack it on a broad front. With New Jersey having the scope of cancer problems that it does, it is clear that you cannot attack it piecemeal. Hence, the only answer is a comprehensive cancer center program that will meld together all of the research in the State and all of the clinical activities.

The interesting thing about New Jersey - and I am sure many of you know it - is the fact that you have the resources in this State to build a comprehensive cancer

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center program. I am not talking about a monolithic institution like M. D. Anderson or the Memorial Sloan Kettering. They don't have that many people. They serve rather as models of expertise to be copied and replicated in part. Ultimately what has to be done is to treat cancer patients in their communities and you have a number of excellent community hospitals. You have gotten a good start in the development of medical schools. And there are a number of very interesting research resources that can be pulled together to build a comprehensive cancer center program.

The Cancer Institute of New Jersey through the funding of the National Cancer Institute, as well as through private foundations, has initiated a number of steps to gain recognition as a comprehensive cancer center from the National Cancer Institute. We have gotten together into programs approximately 300 physicians and scientists throughout the State. Starting from the south, Dr. Coriell at his Institute for Medical Research, and, coming up further north to Rutgers University, the College of Medicine and Dentistry of New Jersey, as well as pharmaceutical research houses, we have been able to gather together approximately 120 basic scientists who are building collaborative programs. Similarly, we have gotten together 15 hospitals that dot the State, which will build networds to disseminate comprehensive cancer care.

The Cancer Institute of New Jersey in building these programs is not anything other than a conduit organization that aids and abets collaborative enterprises. We, at the Cancer Institute, neither treat patients nor do research. We simply try to help the participating organizations do a better job, so that researchers in Camden and New Brunswick and Princeton can work together better to attract more funds from the National Cancer Institute as well as from private foundations. We have done the same thing with hospitals in building collaborative projects, in trying to get them to cooperate in getting linear accelerators in a sensible and rational manner which is not contentious. Ultimately, what we hope to do - and it is somewhat down the pike, but it is ultimately the magic of a comprehensive center - is take the research advances and apply them into the clinical programs.

Research advance includes not just molecular research and chemistry, but it includes research in human behavior, which is essential in understanding what motivates people to take up certain life styles that predispose them to cancer. It includes pain research. It includes nutrition research. It includes epidemiologic research - numbers games, computers. This kind of comprehensive research should be and must be applied to clinical cancer care in order to bring about cancer control. That is the ultimate goal of a comprehensive cancer goal and it is our ultimate goal in the Cancer Institute of New Jersey.

In addition to the support of the 300 physicians and scientists throughout the State, we have institutional backing from the New Jersey American Cancer Society, which has become a blood brother in this program. Rutgers University, the College of Medicine and Dentistry of New Jersey, Dr. Coriell's Institute, a number of business and industrial concerns, the New Jersey Medical Society, the New Jersey Oncology Society, the Hospital Association, the Nurses Association, the Pharmacists Association, as well as the New Jersey Chamber of Commerce,have pledged their backing in developing comprehensive programs.

That is the scope of a comprehensive center and the scope and aims of the Cancer Institute of New Jersey. What we would like to do specifically in New Jersey is to set a priority of programs that are aimed at early detection as well as prevention. The reason for this priority in that order, early detention being more important than prevention, is as the previous speakers have noted, you can eliminate virtually every carcinogen known today and what will happen for the next 20 years is that you will

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continue to see as much if not more cancer than we have been seeing. Whatever we have done, whatever we have done to ourselves, whatever exposures we have had, we will pay for it in terms of developing cancer for the next 10 to 20 years. The only way, therefore, to attack the mortality, morbidity and incidence of cancer is to mount an early detection program.

Amongst the reasons for doing this is that the numbers are right. If you look at the big cancer killers - breast, colon, uterus, lung - there are for most of these rather simple, cost-effective, early detention programs that can be mounted. If you were to do early detection successfully in the uterus, the alimentary tract and the breast, as well as the urinary bladder, you could decrease the mortality rate by 40 percent in this State within a couple of years if you did it wisely and uniformly. That is a 40 percent decrease in mortality. There are currently 14,000 cancer deaths a year in this State. If you added some rather simplistic educational prevention programs aimed primarily at cigarette smoking and alcoholism, you could add another 20 percent decrease to the mortality rate. So, by a combination of prevention and early detection programs, with the knowledge that is at hand now, we could decrease mortality rates in New Jersey, as well as throughout the country, by 60 percent. That is not a pipe dream. That really just means management of resources and education. There is no big mystery in curing cancer. There is no big mystery in the causes of cancer.

We know, in fact, the causes of many forms of cancer. We also know the life styles and things that bring about cancer for the overwhelming majority of the cancers that kill, as well as for cancers that do not kill but maim and add costs to society. For example, if we start with lung, we know that the majority of these cancers are caused by cigarette smoking. There are another devastating set of tumors that are very deadly. These include cancers of the mouth, cancers of the larynx and cancers of the esophagus. These are caused or correlated with individuals who do two things: They smoke and they drink. Now, it isn't a mystery as to how to prevent this. It requires regulation. It requires the elimination of smoking lounges in high schools. It requires the elimination of billboard advertising and other advertising that tends to promote cigarette smoking. I do not agree, for example, with the stance taken by elements of the American Cancer Society that it might be okay to have a cigarette with lower tar and nicotine because, if you have been following the story at all, what cigarette companies do when they lower the tar and nicotine is add flavor enhancers. If you look at these compounds, these compounds are absolutely devastating. I predict that they are deadly. They are not under the regulation of the Food and Drug Administration. They are added quite liberally to cigarettes and they are being burned and oxidized. So the so-called safe cigarette may be even more deadly than its precursor. But smoking must be attacked firmly and irrevocably with no punches pulled.

Similarly the consumption of large quantities of the distilled liquors, which are what we see in the individuals who get cancers of the mouth, larynx and esophagus, has to be discouraged, particularly in conjunction with cigarette smoking. If you add up the total deaths caused by cancers of the mouth, larynx and esophagus, it is almost as many as cancers of the lung. It is a substantial number of cancer patients and they are in deadly locations. The treatment for cancers of the esophagus, larynx or head and neck is extremely costly. It runs society an enormous amount of money. It runs insurance carriers into the red because the surgery is extensive, the radiation therapy is extensive, the chemotherapy is extensive, and we lose the battle anyway in those areas. They are extremely deadly forms of cancer. But we know how to prevent them and we are not doing anything about it.

We also know some of the life styles associated with cancer of the large bowel,

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which is the next largest killer after lung. Epidemiologically, it has been well demonstrated that diets that are high in fat, particularly cholesterol, coupled with a low-fiber content, which characterizes much of the American diet, markedly and greatly predispose to cancer of the bowel. There are people who live in this country who have very low rates of cancer of the bowel. The Seventh Day Adventists who follow certain dietary restrictions enjoy very low rates of cancer, even though they live in Southern California where environmental pollution is horrendous.

We know what causes cancer of the cervix or, at least, we know what correlates with it. The age of onset of intercourse is the best correlation with cancer of the cervix.

We know that cancer of the endometrium is correlated with misuse of Premarin, a conjugated set of estrogens that are given by gynocologists to women who are in the menopausal period. We know that uncontrolled use of Premarin is associated with increasing incidences of endometrium, another common cancer killer.

Cancer of the skin, which doesn't kill many people, costs society a great deal of money, particularly since many of them occur on the head and neck and require not only extensive curative surgery, but extensive plastic surgery, and result in patients feeling like hermits and not going to work. Cancer of the skin is caused by ultra violet light. Simple preparations that act as sun screens could very effectively decrease the costs of skin cancer. This includes three forms: the squamous cells, basal cells, as well as the more deadly form, the melanoma. But we don't do much about that. We do not teach children. We do not teach adults. We do not promote these preventive mechanisms.

So I think emphasis on these approaches might be productive and not necessarily costly. I think when you get into the area of prevention, one of the first things that should be done is to clearly define what we mean when we say environmental cancer. I think it is necessary to distinguish between environmentally-related cancers and industrially-related cancers. They are not the same thing.

Individuals like Dr. Cooper,who is the Assistant Secretary for Health, recently appeared on the Rutgers campus a couple of weeks ago and referred to the fact that four out of five cancers are caused by the environment. That leads to a lot of misunderstanding. I know Dr. Cooper. I have heard him talk before and I have had discussions with him. His definition of environmental cancer is everything that we do to ourselves. It is the personal environment, the micro-environment, what you smoke, what you drink, what you eat, the age of onset of intercourse: just the total life style is what Dr. Cooper means by environment. I am afraid that it is easy to misunderstand environmental cancer and blame that and interrelate it with industrially-related cancers.

Industry-related cancers form a very small and rather insignificant percentage of our cancer statistics. If you go through the cancer mortality rates that have been published and subtract out the national average from the excess that New Jersey has - and I have done these analyses --- In other words, there is a certain set rate of cancer that we will keep getting in this country, separate and aside from anything that relates to industry. There is a base line due to other aspects of the personal environment that will produce cancer. If you look at those statistics and go on the basis of what we know may cause cancer that is industrially-related, you look at cancers of the stomach, cancers of the lung, liver, bladder and skin. Those are cancer sites wherein industry-related agents have caused problems. Cancers of the breast, cancers of the colon, cancers of the uterus, prostate, and pancreas are not and have never been shown to be related to industry. There are no industrial exposures that have been proven to relate to things like breast cancer, prostate cancer or cancer of the colon or pancreas.

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So, if you restrict yourself and look at the data for the excess stomach cancers that New Jersey has over and above the national average, and the excess of lung, liver, urinary bladder, and skin cancers, you come up with a figure of somewhere about 600 excess cancer deaths in those areas where you might try to blame industrial factors.

Now, even with asbestos exposures, where the correlation is great and the causitive agent is unquestionable, even there it is not just exposure to the asbestos; the conjoint exposure to cigarette smoking markedly enhances the development of lung cancers. So, we have the spectrum of coexposures and multiple exposures to many types of carcinogens. Many workers will both smoke and drink, as well as be exposed to industrial carcinogens.

So, out of that is a maximum that I have figured out of about 600 excess deaths in New Jersey every year that you might blame on industry, or industry-related factors. That is 600 deaths out of 14,000. And, I submit that given a choice of where to focus, I would rather focus on the 13,400 deaths where we have no known relationship to industry and where we know some of the other factors that we can control through education and early detection. That is the place to make the greatest gains in terms of human life, as well as ultimately decreasing the cost of medical care.

I do not advocate an increasing health care dollar. I think that if we pull together as a comprehensive cancer center, we can not only solve the cancer problem, we can bring about a reversal in the spiraling health care costs. One example will suffice. If you detect cancer of the cervix early, all you have to do is a simple hysterectomy. If you detect cancer of the cervix late, you have to do a radical hysterectomy. The surgeon's fee is much greater for a radical hysterectomy. The number of days in the hospital is at least doubled. In addition, you have to give expensive radiation therapy. And, furthermore, you turn out a patient who is going to die, who will seek multiple hospital readmissions, all at the expense of society. She is going to have to have help at home, again at the expense of society. And, she will not be able to go back to work if she is working. And, she is not going to be a taxpayer.

The difference between these two is $1.67: that is the cost, the raw cost, of a pap smear to detect cancer early. And, if you prorate these two types of individuals and add in all the costs, there is about $100 thousand in cost difference between these two individuals. You can save $100 thousand for $1.67.

I can make the same argument with cancer of the bowel. For 50c, I can detect occult blood in the stool. If I do that four times, which costs $2.00 - if I do that on everybody over the age of 40 in this State, I can save you money. If we do simple things like this, there are cost defective mechanisms, and they need not only be done by doctors. We can train paramedics under the supervision of doctors to do these early detection tests.

Cancer of the breast can be detected early. We don't have to do mammography. You can pick up just as many cancers of the breast by a triplet of activities. If you do breast palpation, plus thermography, plus the use of ultra sound in a technique called echo tomography. You can pick up 95% of early cancers of the breast. You don't need mammography, which appears to be dangerous and is also costly. But, every agrument that I have made about cancer of the cervix applies to cancer of the breast and cancer of the bowel. Those are huge killers and they add enormous cost and we have to gradually shift over the health care dollar into prevention and early detection

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programs.

Currently, the biggest retardant in carrying out early detection stems from multiple factors. Number one, medical schools do not teach early detection. Secondly, nobody pays for early detection. Most insurance carriers, and most third party payers are regressive or atavistic and do not understand early detection costs. They amount to something like well-baby care, except it is for adults and nobody understands that paying for prevention or early detection really can save money in the long run. It just isn't being done. Hospitals, in general, are encouraged to have facilities for early detection and, certainly, the doctor who donates time to supervise an early detection clinic in this State will not be compensated for his time. So, if we look at the total scope of the problem of what potential you have, I think it is enormous. There is no other state that is doing a better job than you, so you need not feel bad. All states are very far behind in applying the knowledge that we have gained in the past number of years.

Frank Raucher, the former Director of the National Cancer Institute, who has joined the American Cancer Society, has summed it up rather nicely and it is particularly relevant because he is a Phd. in virology. He is a basic scientist and you wouldn't expect him to make the comment that he made, that at the helm of the National Cancer Institute, for the past several years, he has spent literally billions of dollars in research and the most profound thing that he and others have learned is that 90% of cancers are caused by external agents that can be largely controlled. We cause cancer and we bring it upon ourselves. It is possible, I think, to legislate against it and to develop programs against it.

At the Cancer Institute of New Jersey, as I think I indicated to you last week, Senator Skevin, together with the American Cancer Society, we are sponsoring a series of workshops that will pull together a number of experts on a series of comprehensive programs that might be developed into legislation at the State level. This would include workshops on controlling the environment of school children, or relative innocents, who innocently go up to the candy machine and get out a package of red licorice which contains about a gram of known carcinogen. I don't trust the new red dyes either. We feed them bacon sandwiches which contain known quantities of nitrosamines. We do nothing to control that.

There are other needs like protection of the job of the cancer patient. The prevalence rate at any one time - there are about 150 to 200 thousand cancer patients in the State of New Jersey walking around at the present time. Their jobs are at risk because their life is not understood by the employers. There are a series of needs and I think that the tip of the iceburg has been touched in your hearings and we would endeavor to hold these workshops and invite you to be a co-sponsor or a participant in these because they may amplify the speed with which you may be able to progress with your legislative programs.

SENATOR SKEVIN: Thank you, Dr. Demopoulos for that excellent presentation on your Institute and also your views in this area.

In terms of environmental cancer, you mentioned Dr. Cooper. By his definition he would say that four out of five of our fourteen thousand deaths are attributable to environmental cancer. The question between what is involved in terms of industrial factors or substances, and other factors, is open to question, is that correct?

DR. DEMOPOULOS: No, they are not open to question. Dr. Cooper has firmly enunciated what he means by environmental cancer. He means cigarette smoking, alcoholism, artificially colored foods, bacon, nitrosamines, lifestyles, high fat in a diet with low

fiber content. He has clearly defined what he means by the four out of five cancers.

SENATOR SKEVIN: Isn't it a fact that those lifestyles, or those factors that you mentioned, accelerate cancer with industrial exposure? Isn't that a fact?

DR. DEMOPOULOS: I don't think that it is a fact. There is a possibility that we may see synergism and I don't think that we ought to ignore the possibility of industrial exposures and I would not want to give that impression. I think that we must firmly address ourselves in a comprehensive program to all causes of cancer, even the 600 industrially-related deaths are important - even one death is important if it can be avoided.

There is a grim specter that is unknown, so it is not a fact. There is the grim specter that industrial pollutants could conceivably act synergistically. Now, that is an area that needs research and as Dr. Coriell has pointed out earlier, there is a great need to have research support to answer questions like that. You cannot assume synergism. It is a possibility. We have seen it in a number of situations that do not involve chemical carcinogens. We have seen it in a few instances like cigarette smoking and asbestos where there is synergism proven.

SENATOR SKEVIN: There is a higher increase, right there, isn't that a fact?

DR. DEMOPOULOS: Yes. I think four out of the five asbestos workers who got lung cancer - I think four out of every five were cigarette smokers.

SENATOR SKEVIN: And their rate was 90% greater than non-smokers with asbestos exposure, isn't that correct, Doctor?

DR. DEMOPOULOS: That is correct. So, I would not advocate letting pass the question of environmental pollutants. I think it is a major topic but I think it should be approached with facts and one of the ideas about these workshops is, since we would be sponsoring it as an objective resource, that neither legislates nor makes a profit, not treats patients, nor does research, we can be fairly objective and host them and bring together elements in this State from industry, from the Department of Environmental Protection, your own offices, the Governor's people, to discuss really the scope and the problems of industrial cancers and industrial pollutants and what might be done about them. What are the facts?

I think there is a great danger in developing legislation if it is not based on the latest facts. They get overturned subsequently in courts of law when their constitutionality or their regulations are challenged. And, if the facts are otherwise and if the legislation is not based on good science, you run that risk of actually losing time if you pass a law which is not scientifically based.

We can provide the scientific basis and, indeed, your proceedings here have pulled in a considerable number of some of the world's best experts in cancer and you are to be congratulated on the wisdom and foresight you have had in doing this. But, I think that even more has to be done.

SENATOR SKEVIN: Even if you assume that the 600 deaths that you talked about, the 600 fatalities due to industrial cancer - we assume that figure is correct - you by no means feel that we should not direct our efforts in that area to control that, do you?

DR. DEMOPOULOS: You should direct your effrots at that area but you should do it in a comprehensive manner. You should attack all causes and there are things that we can do that I think if you had to attach priority to - and I think this is the question that has to be raised - if we are limited in funds and if you are limited in capacity of what to do first - what should you do first - I think it is tragic if we have to attach priorities; I would rather not. I think that there isn't

any reason why we can't do this across the board in a comprehensive manner to attack all the other facets of cancer-causing compounds in a personal environment and in a personal lifestyle.

SENATOR SKEVIN: In terms of early detection, you projected a reduction of . fatalities, 40% to 60%.

DR. DEMOPOULOS: That is correct.

SENATOR SKEVIN: How long would that take if we implemented a program immediately?

DR. DEMOPOULOS: Well, if this was Uganda and you or I were Idi Amin, you know, we could do it in two years. But, that is not the case. I think it is just a question -- we could do it in probably three to four years, realistically, if we started working together at this point, using all of the elements that have agreed to cooperate already - the doctors, the hospitals, etc. There are leaders in industry and business who have agreed to add early detection programs, for example, at their cost to pre-employment physicals and to continuing annual physical examinations. This is an agreement that I worked out in private with some of the major employers of the work task force in this State. That is not going to cost the State any money. It is not going to cost us any money either. They are going to pay for it. And, it is not for their specific industrually-related cancers: these are general cancer screening programs that are being developed.

But, I think if we proceed with dispatch, we might do it in a few years - two years if I were really a cancer czar.

SENATOR SKEVIN: Senator McGahn, do you have any questions?

SENATOR MC GAHN: No questions.

SENATOR SKEVIN: Senator Martindell?

SENATOR MARTINDELL: Dr. Demopoulos, I was interested in what you said about the schools because I am acting Chairman of the Education Committee. Has anything been done about the smoking lounges? Is anything being done?

DR. DEMOPOULOS: Well, I think they have been allowed to proliferate. I don't think anything has been done.

SENATOR MARTINDELL: Nobody is trying to stop it?

DR. DEMOPOULOS: No. We have reviewed the legislation of 40 other states in the country that relate to cancer and most of these are amateurish programs aimed at controlling quackery and laetrile treatments and things of that ilk. Nobody has really taken a comprehensive crack at this. Certainly, the only state that I know of that has done anything about smoking is Florida, where an individual - Mr. John Regan, who is somebody we are trying to recruit at the present time - has instituted anti-smoking educational programs, statewide. At first he started with the high school students and failed miserably. He then went down to junior high school and failed miserably. And, he is now working with, and he thinks he will be successful, the 5 and 6 year olds in kindergarten and first grade. He is turning them into snarling little anti-smoking monsters that tear through their houses and through their parent's belongings destroying cigarettes on sight. Maybe that is the way to go, I don't know. But, certainly, smoking lounges are not to be encouraged. I don't think you necessarily need legislation against them.

When I went to high school, the principal in my school used to run through the boy's bathrooms chastising, violently, any cigarette smoker that he found. I am frankly chagrined that we aid and abet cigarette smoking through smoking lounges.

SENATOR MARTINDELL: I think so too. You spoke about the sandwiches, what

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about that?

DR. DEMOPOULOS: Well, I think more attention ought to be paid, epidemiologically, to this. The data is as clear as it is for cigarette smoking and lung cancer. I think that the State has an obligation to realize this and to exercise its regulatory powers over at least school lunches, as well as what is available in snack bars and candy machines that inhabit the school corridors. I think artificially colored juices and things like grilled bacon, ham and hot dogs, where the nitrites and nitrates that are present in preserved red meats are heated, nitrosamines are produced, which are powerful carcinogens. That is not myth and that is not a possibility; that is proven. The amount of carcinogen present in a bacon sandwich far exceeds that present in a carton of cigarettes.

SENATOR MARTINDELL: What about water - treated water?

DR. DEMOPOULOS: Well, I don't know about polluted water. I think that there are not sufficient studies that have been done in this area and that too is one where more research is needed. Again, amplifying what what Dr. Coriell said and amplifying what people in the Department of Environmental Protection said in your own State, there is a need to expend more funds to do research in the control of pollutants in water, as well as in the air. These are very complicated areas. A lot of the chemicals undergo changes when they are introduced into the air or into the water or into the soil.

Many of the known carcinogens, for example, are inactivated in sunlight. Ultraviolet light will destroy some known carcinogens so that the release of certain industrial pollutants during the daytime may not be so bad but you would have to worry about them at night if they have a nighttime shift. Furthermore, other kinds of compounds that are innocent when they first come out of the smokestack, or out of a truck, or bus exhaust, are converted into carcinogens after photoactivation.

But, just on the point of these pollutants and water and air, I have not seen enough attention paid to bus and truck emissions. They just seem frightening as heck, particularly with the turnpike passing through some of the most densely urbanized areas of the State. I think they probably pose a greater threat than most of the industrial sites that are fixed in this State.

SENATOR MARTINDELL: Could you jot down for me the names of those breast cancer tests. I didn't write them down fast enough, other than mammogram.

DR. DEMOPOULOS: Oh, the breast palpation, plus thermography, plus the last one is the complicated one, it is echo tomography. If you do all three - and these are non-hazardous - you can do them five times a day and there is no problem.

SENATOR MARTINDELL: Thank you.

SENATOR PARKER: Are you saying that the auto emissions -- are you differing in the comment made by the previous speaker about auto emissions and truck emissions? He indicated that there is no problem, or at least I understood him to say that.

DR. DEMOPOULOS: Well, I think he was talking about some Los Angeles studies wherein thousands of animals, rats and mice, lived in cages right alongside the Los Angeles Freeway where you could expect that they might get enormous exposures. In those studies the yield of spontaneous cancers amongst the animals was extremely low and it pointed away from any kind of significance of air pollutants causing cancers in mice and rats living along the Freeway in Southern California.

I think if you look at truck and bus emissions here-- I have lived in both places and I know the studies along the Los Angeles Freeway very well. I helped to train the pathologists that examined those lung slides and those animals. I think,

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just taking a guess, that there is more emission uncontrolled from trucks and buses in this State, particularly buses. There are not many buses on the freeways in Los Angeles.

SENATOR PARKER: I understand that, but what is there in those emissions that give rise to the cancer? Is your answer different from the other speaker?

DR. DEMOPOULOS: No. It is not different. He was citing a series of studies that were concluded and dealt largely with automobiles. I am talking about trucks and buses, particularly buses where unburned or incompletely combusted fuels are liberated through the exhaust. Those kinds of hydrocarbons are quite dangerous because they can be photoactivated into more dangerous compounds. They do not have many buses, as I said, on the freeways in Southern California. They just introduced them this year.

We have tons of buses on the New Jersey Turnpike and I think that we might have a different situation here.

SENATOR PARKER: You are basically talking about diesels when you talking about buses, is that correct?

DR. DEMOPOULOS: Yes, I am.

SENATOR PARKER: Is that different from auto emission, which is--

DR. DEMOPOULOS: There are different compounds that come out. The total hydrocarbon emission is much greater out of a bus and the unburned diesel fuels.

SENATOR PARKER: And are you saying to us that they do cause - or can cause - cancer, or put carcinogens into the air?

DR. DEMOPOULOS: They can put compounds into the air which are subsequently converted into carcinogens. The stuff that comes out of the tailpipe is not carcinogenic but what may happen to it later is carcinogenic.

SENATOR PARKER: Well, the reason I ask that is, it has always been my impression, from everything I have read, that it is the auto emissions which were more dangerous than were the diesel emissions.

DR. DEMOPOULOS: Not for cancer. You have to distinguish between air pollutants that are viewed as dangerous to the health in general because they might produce emphysema or cause pneumonias, versus pollutants that are suspect of causing lung cancer.

SENATOR PARKER: To the layman emphysema is really no different from cancer of the lung, as far as I am concerned. You get it one way or the other.

DR. DEMOPOULOS: Most automotive emission controls were aimed at the oxides of nitrogen and carbon monoxide and very little attention has been paid to total hydrocarbon emission. What I am saying is, truck and bus hydrocarbon emissions need to be explored. I think that if you were to do a study, I suspect - but it is not a fact, okay? - that you might find that more hydrocarbons are emitted from the New Jersey Turnpike truck and bus traffic than all of your industrial plants in this State, which already have hydrocarbon and other smoke emission controls in place.

SENATOR PARKER: We do have controls and we will look into that. Let me ask you - you mentioned two others that are somewhat unknown to me. I am somewhat familiar with kepone and just one press release described what it will do to the fishing industry on our coast.

But, you talked about bacon and also alcohol. Now, let's just take alcohol - what is there in alcohol that gives rise either to a carcinogen and/or accelerates the procedure? This is new to me, that is why I ask.

DR. DEMOPOULOS: It is largely the alcohol itself. Alcohol and the distilled liquors reach a high concentration. Alcohol is a toxic substance which, aa far as I

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am concerned, from the research that has been done throughout the world, as well as in my own laboratories, is a deadly toxin and it should be treated as such. There are, in addition to the distilled liquors, occasionally trace contaminants and metals that might add to it. But, as far as the alcohol itself is concerned, alcohol damages very sensitive parts of the cells and epidemiologically, as I said, about 90% of the patients who have cancer of the mouth, larynx, and esophagus have the history of strong alcoholism plus cigarette smoking.

SENATOR PARKER: Just alcohol in and of itself--?

DR. DEMOPOULOS: Yes, if you were just drinking Vodka or Gin it would be just as bad.

SENATOR PARKER: It would be just as bad and you say that causes cancer?

DR. DEMOPOULOS: In conjunction with cigarette smoking.

SENATOR PARKER: In conjunction with cigarette smoking?

DR. DEMOPOULOS: All by itself, alcohol will cause cancer of the liver, but only in a small percentage of people. It certainly will pickle the liver.

SENATOR PARKER: That is cirrhosis, isn't it?

DR. DEMOPOULOS: That's correct. That is a small number of people, but with mouth, larynx, and esophagus we are talking about large numbers and there you have synergism between two personal lifestyles.

SENATOR PARKER: Now, you talked about bacon and hot dogs.

DR. DEMOPOULOS: Grilled.

SENATOR PARKER: Grilled?

DR. DEMOPOULOS: Grilled.

SENATOR PARKER: As opposed to boiled?

DR. DEMOPOULOS: Possibly. The trick is the direct heating of the items.

SENATOR PARKER: Well, all bacon is pan fried.

DR. DEMOPOULOS: That's right, you can't get away with it with bacon. Hot dogs are grilled. And, when you make ham and eggs in the morning, you fry the ham a little bit. There are very simple inorganic compounds called nitrites and nitrates that are added to a number of preserved meats, like corn beef, pastrami, balogna, salami, ham, bacon, hot dogs. In many cases, the nitrites and nitrates are added for purposes of preserving a redish color to make it more appetizing. In other cases, some of the nitrites and nitrates are added in order to decrease the growth of bacteria that might produce the deadly poison, botulism. So, there is some degree of meat preservation associated with the use of nitrites and nitrates.

SENATOR PARKER: Even smoked? For instance, bacon is smoked.

DR. DEMOPOULOS: Bacon without nitrites and nitrates is called salt pork. A hot dog without nitrites and nitrates in it is called bratwurst.

SENATOR PARKER: And it is a lot better than a hot dog too.

DR. DEMOPOULOS: The point is that in meat that is so preserved, there are other compounds that are called amines. When you heat, under the high temperature of a frying pay, such meat that has nitrates, or nitrites, plus amines, there is a chemical reaction that is produced and it causes the development of a compound, or series of compounds known as nitrosamines. These are known, proven, carcinogens.

SENATOR PARKER: Can you use another type of preservative in these red meats?

DR. DEMOPOULOS: I would imagine - but is just imagination because I am not a food technologist - that you could. You certainly wouldn't-- I think the preservation of the red color is aesthetically stupid. It is not necessary and I think there is a serious question about how do you prevent botulism. I think there must be other ways.

maybe by more careful preparation of the meat products in the processing plant.

SENATOR SKEVIN: Thank you, Doctor. Dr. Wagner.

D R. B E R N A R D M. W A G N E R: Good afternoon. Because of the lateness of the hour, I will not repeat what everybody else has said.

SENATOR SKEVIN: Thank you very much.

DR. WAGNER: I will just simply--

SENATOR SKEVIN: On behalf of everyone here I thank you, including the stenographer.

DR. WAGNER: Just let me say that I thank you for inviting me to come before you. I consider myself here, first as a citizen and resident of the State of New Jersey and, secondly, as a scientist. The question is, should I be a concerned citizen of the State of New Jersey? The answer is, yes, I am concered. Having been born and raised in Philadelphia and then after working many years in New York, it has been a standard joke - as you probably know - that New Jersey for too long was a medical vacuum between Philadelphia and New York. I think what we are really addressing ourselves to is to a correction of a vacuum.

Working in a major community hospital, Overlook Hospital in Summit, New Jersey I see far too many patients, or far too many citizens of the State of New Jersey, still going to the medical centers in New York and Philadelphia for cancer care and I ask myself, why? What has this State done, or not done, in this area. I believe that the requests that have been put forward by all of the previous speakers are quite clear. New Jersey needs a comprehensive cancer center approach, broad and across the State, but rather unique to the needs of the State of New Jersey, where we have our population spread out in many, many small communities.

Touching, very briefly, on some of the problems that I see that I think we can address ourselves to is the one of early detection and the exact diagnosis. Dr. Hammond speculated on the quality of the raw data and I would too. This means that New Jersey needs a much better registry in this State. We really don't know how many times the diagnosis of cancer is made incorrectly and the patient is labeled. By this I would reinforce a stable funding mechanism by the State, as in many other states, that would establish, clearly, a state cancer registry, but it would expand in an out-reach fashion to all of the hospitals and eventually the practicing physicians in the State of New Jersey. There is no reason why a physician in a small community in this State, faced with a diagnosis of an unusual cancer, can't immediately have access to the best opinions available for therapy in the State, and, yet, this is not available.

Dr. Hammond spoke on specifics, namely a slide program, wherein the tissue can be reexamined and so that the diagnosis can be correctly made by other experts who see this type of rare tumor all the time. This is not available and as newer techniques through research become available for application to everyday medical care, for example, the electron microscope-- The electron microscope was discovered in the State of New Jersey many years ago. It is one of our most valuable new tools in making the accurate diagnosis of cancer. Yet, it is not available to the doctors and the pathologists of this State because it is so costly and they don't know how to use them. But, in other states they have centers where they can get this back-up information.

Touching on the high cost of care once the diagnosis is made, means we ought to take a look at our reimbursement policies for the cancer patients in the State of New Jersey. This is a very serious problem because of the tremendous cost to the patient and his family. Dr. Coriell cited the statistics. An inadequate number of physicians in many counties in the State of New Jersey means inadequate patient care and inadequate patient care means increasing patient deaths.

Now, the State of New Jersey is blessed with the fact that it has one of the

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highest concentrations of biological scientists living in the State and they probably work out of the State, either in Philadelphia or in New York. If one takes a look, through accepted criteria for a national recognition as cancer scientists, we find that a large segment of those in the State of New Jersey are in our industries, especially pharmaceutical industries. So, there is a need to pull all of these together. The State has a responsibility to the people who live in this State, that they are assured they can have the best quality care in terms of cancer detection and ultimately treatment.

Secondly, industry has to be made part of the dialogue. They are interested. They want to participate. We have to find ways of including them.

Finally, there is an enormous body of good will on the part of scientists, as you have already heard, in and out of the State, to work with New Jersey. I think the mechanism that allows you to do this best is through the Cancer Institute of New Jersey. Plus, the necessary supportive legislation would enable funding that is required to carry out some of these activities.

Just one brief problem that we constantly run into and this can't be legislated - that has to do with the lack of information as to what really constitutes a true carcinogen. I cite for you the problem with the cyclamates. We have banned it. Canada uses it. England uses it. The question of red dye 2, this is not clear. And for those of you who might remember old Hires Root Beer, its flavor was due to a material called safrole, which was shown to produce cancer of the liver in rats when used in high concentrations. But, it was calculated that to do this in a human, someone would have to consume a case of Hires Root Beer, daily, for about 20 years. Nevertheless, the FDA took the position that there might be somebody, somewhere in the United States who might do this and, therefore, as a protective agency it was taken off the market.

So, there is some sense and nonsense in our present look at the definitions of carcinogens and potential carcinogens, simply because we don't have enough information. This, again, I think is the responsibility of State's to see that on-going research is supported.

I would simply like to close by saying that New Jersey has excellent community hospitals. Because of financial considerations, they are unable to render the kind of detection activities and support activities that are necessary. I believe the State should look at this question.

We need far better support of our embryonic medical schools to make them the kind of leaders in the health, education, and research areas that befit the State of New Jersey.

Finally, I would hope that we just don't keep talking about the need for these programs. Dr. Coriell, Dr. Hammond have histroically cited for you that this has come before the State Legislature over the last 10 or more years and nothing has happened. I am hoping that this combined effort and awareness on the part of the citizens in the State of New Jersey will lead to the kind of broad, effective programs supported in long-term fashion that we need. Thank you.

SENATOR SKEVIN: Thank you, Doctor, for your remarks. Brief as they are, they cover the wide sphere of activities and certainly we appreciate them.

In terms of your concluding remarks, Doctor, about the fact that the problem has been before the State Legislature, is it correct to say that the information that we know about the danger of various factors involved has been available to State Government for the last 10 years or more?

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DR. WAGNER: Yes. The information has been available.

SENATOR SKEVIN: And, essentially, the information - environmental, industrial - has been available and to this date nothing has been done, is that what you are saying?

DR. WAGNER: Well, let's put it this way: The awareness of the information has been quite obvious, but inplementation has been non-existent.

SENATOR SKEVIN: And in terms of wringing our hands about the problem, as Dr. Hammond pointed out and you so well pointed out, we should start rolling up our sleeves and start to do something here?

DR. WAGNER: That is correct.

SENATOR SKEVIN: Okay, thank you, Doctor.

Dr. Cohen.

D R. F R E D E R I C K B. C O H E N: Senator, I think I will be brief because I have two statements. First of all, this is my first experience with a legislative body, except for Senator McCarthy, Senator Kefauver, and Senator Ervin on T.V., and seeing Mr. Smith Goes to Washington seven times. Since this is the only experience I have had, I didn't know that a statement was necessary.

Second, I guess the reason you called me was because I blurted out at one of your recent hearings what all the other speakers have said in the last two hours. I told you that in five minutes.

Finally, I guess you recognize that I probably Chair the Department in New Jersey which treats more patients with cancer than any other department in the State, and that I have been interested in cancer, and dealing with these problems, since 1958.

So, if you have any questions, I will be glad to answer them. I certainly can define the cancer problem in terms of the individual cancer patient and in terms of the individual cancer doctor, if you will, in the State, for anyone who is interested.

SENATOR SKEVIN: Well, we appreciate your coming here to give your comments, Dr. Cohen. I recall that we met at the Clean Air Council meeting at Middlesex Community College a week or so ago. You impressed me with your ability and your background, in terms of cancer in New Jersey and that is why I asked you to come today.

Dr. McGahn, do you have any questions?

SENATOR MC GAHN: No, I don't.

SENATOR SKEVIN: Senator Parker?

SENATOR PARKER: No.

DR. COHEN: I can only say, for things to say, that really the thing that I think should be emphasized - and that the experts here have emphasized with statistics which we in the medical profession, or at least in the oncological profession, have recognized - is, when you deal with a cancer problem you are dealing with a problem which is multi-faceted. It consists of diseases which are at least 100 in name and at least that many in differences of pathology, ideology, etc. Any attempt to attack it on a small basis, on a narrow basis, would be futile, expensive, and probably cause more trouble and more aggrivation to the people you neglect than any benefit you might derive from it.

I think that the Cancer Center Program that we are trying to establish in New Jersey will probably make available to the Legislature, at least, information and expertise which they can use in establishing any fight on cancer.

Certainly, one thing that has not been stressed here is the thing that I deal with, namely the treatment of the patient who already has cancer. Remember, you asked me the question once, "Would early detection mean anything?" Despite what the

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other experts say, I said I wasn't sure because it is something that is apparent to me that may not be apparent to those who do not treat patients. Very often, when a patient has an early cancer it is already too late. What you mean by early diagnosis may not be feasible. Cancer may be a disease which starts in many areas at the same time in many cases. Although early detection in terms of certain things such as some cases of breast cancer and some cases of colin cancer and some cases of prostate, etc., is very, very essential and I think it is most important.

One of the things that has been neglected in this State, as it has in most other states - except where they have large, functioning clinical cancer centers - is the treatment of patients with cancer, primarily in terms of educating physicians, many of whom don't, even to this day, know or appreciate or recognize the value of present-date, modern treatment of cancer. Number two, we have to make available to patients the places where they can be treated in their own neighborhoods, or in their own cities so they don't have to travel. They either become too sick to travel or they fear going to other places and they don't have the respect for the physicians, nor contact with their local physician who can help in the management of their disease. Finally, we have to help them pay for this treatment, or we have to be helped in order to provide this care.

At the Beth Israel Medical Center, it now costs approximately $15.60 per day for the drugs to treat cancer patients, whereas for all other patients in the hospital, the cost is only $3.50. Patients have to pay the physician sometimes as much as $150 per week for the cost of the drug, or drugs. These are things that no one has talked about and I think it is very important, so that as you proceed on your way to define the cancer problem to determine what causes cancer and prevent it, I think someone along the line ought to help us to treat the patients.

The 14,000 patients who are going to die this year, have their cancer today and I can predict that most of them can be kept alive for at least twice the length of time they are doomed to now if we were given the proper tools and help.

SENATOR PARKER: In that regard, it may well be that after Tuesday we are going to have a national health program. But, if we don't, is there any way that we can increase our policies or write some form of catastrophic insurance that would be helpful in the policies to provide sufficient funds for the appropriate care of cancer patients?

DR. COHEN: Yes. I must say because I am a physician and not a member of medical organizations - I am not a medical politician - that when I defend doctors, if you will, I am defending myself and the people that I deal with - my colleagues - who are really primarily interested in making people better. You make a good income and you do all kinds of things but your sign of success is when you make somebody better.

The cost of the delivery of the care, the touching of the doctor - and I am not talking about a surgeon now because that is not my business - is the least expensive part of medical care in this country. The $10 or the $6 or the $5 or the $15 that the patient pays for the doctor to touch them - there is more benefit derived from that; it is the best bargain you can get. It is the cost of the diagnostic machinery, the cost of the drugs, the cost of the laboratory tests that is so expensive. And, the reason they are expensive, of course, is because so many other people derive income from that - labor, etc.

There may be some program to provide just the drugs themselves. Maybe the State of New Jersey, for instance, could have a pool whereby they have in certain

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areas all the Adriamycin, or all the 5 Fluorouracil for so-called indigent patients and you could determine that and it could be given to them. The American Cancer Society does it. They pay for a certain amount of it. Other people do.

SENATOR PARKER: Do they have a program of special assistance for catastrophic diseases for cancer patients?

DR. COHEN: Yes. I don't know about catastrophic. They have a program for needy people which meets some of the needs. It is not available to all of the people becuase of funds, I guess, and also because many people don't know about it. You have to apply for it. They have to have a meeting. They have to agree to it. They pay for some of the medications.

There are many private organizations in our area, which provide funds for indigent patients. But, it is just not enough.

SENATOR PARKER: Well, it is not necessarily the indigent patient--

DR. COHEN: That's exactly right. You ought to know that even the Medicaid - even Medicaid in the State of New Jersey - does not pay for cancer drugs unless the patient is given a prescription and sent to a drug store who never heard of the drug and then maybe they will get paid for it.

But, we, in our institution, treat hundreds of Medicaid patients and we administer the drugs, whether they can pay for them or not. The Beth Israel Medical Center in Newark pays for it. Now, whether they get reimbursed or not - that always happens down in Trenton, I guess - the fact is, people walk into our clinic, they have Medicaid, the hospital - or somebody - is paid for the visit but when it comes to the drugs, there is no reimbursement for the drugs. I inject, personally, into the patients, every day, drugs that are worth on the market - that would be charged by the druggist or the pharmaceutical house - $150 to $200 per injection and nobody is paying for it.

SENATOR PARKER: Let me ask you a question. In No-Fault for automobiles they pay 100% of all cost. For Workmen's Compensation, if it is a work-related cancer or emphysema, they pay all the cost. Here we have private policies, which most people have for their health care services and they are limited in the amount of benefits to be derived under the terms of the policy.

DR. COHEN: Of course, for cancer. Of course. And the other thing that was mentioned was the cost of diagnosis. As you all know, if you want to perform a diagnostic test on a patient, it says in his Blue Cross Policy, it says in his Medicare Policy, it says in many of the other policies that they do not pay for diagnostic tests.

Now, of course, there can be abuse. There is no question that there are unscrupulous people. But, the majority of doctors, I would say, or people who would recommend a diagnostic test, do it to prevent or to diagnose a disease, and people don't get these things done because no one pays for it.

SENATOR PARKER: That will be an area, Mr. Chairman, that we will have to look into.

SENATOR SKEVIN: I think so. I agree. Thank you very much, Doctor.

DR. COHEN: One other thing I just want to go on record with one other thing. That 90% I mentioned was repeated here, even though the New York Times said, "Where did I get that figure" - or Dr. Paulson said it - I heard it repeated here.

SENATOR SKEVIN: Right.

SENATOR MC GAHN: Dr. Cohen, since we have you here, and I think for the record, you were talking about treatment. I don't want to put you on the spot but would you discuss the use of laetrile?

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DR. COHEN: Yes.

SENATOR PARKER: Well, can you tell me what laetrile is?

DR. COHEN: That is just what I am going to do. I always do this. My wife told me that. I define what I am going to do and then I will tell you.

Laetrile is a name for a chemical compound, the exact nature of which I don't think anyone has defined. It is said to be produced -- it is a product of the crusing up of apricot pits by somebody. Whoever thought of crusing them up, I don't know. One of the things that probably caused it to come into being is, some scientist probably looked at this chemical because it has a cyanide radical in it. Cyanide, as you know, is a deadly poison and a cellular poison, if you will; it interfers with the oxygenation - the oxygen cycles - in the cell and someone probably looked at this cyanide and found it came from this laetrile. Someone then named it Vitamin B 17. Now, as far as I know, there is no such thing as Vitamin B 17. I am not sure there is a 15 or a 16, that I know of. But, there is no B 17. This was obviously a ploy to get around the laws that prevented the use of medicines that were not approved, by saying it was a vitamin, or a food supplement - as you know. They thought they could get away with selling it that way.

At any rate, somewhere along the line, someone thought they demonstrated that laetrile, or Vitamin B 17 was an anti-cancer agent. Now, this was then, as all anti-cancer agents are that have any hope of success, investigated. I must say in my personal experience, which now is a long one in this field - this happens to be a young field so that a fellow like me can become an expert in it in a very short time - this single agent, whatever you want to call it, has had more testing and more involvement by Federal and private good scientific experts than any other drug I know of. If any other drug had come to the attention of the National Cancer Society or Roswell Park in New York or Memorial Hospital in New York with the slight credentials this has, it would have been thrown out. But, because there was a tremendous emotional excitement engendered by the people who sell it: because there were national legislatures involved - just like other drugs in the past - there were people involved who were not scientists and who had a tremendous amount of pressure to bring to bear and several studies were undertaken at the National Cancer Institute on human beings and on animals - but human beings also. At Memorial Hospital, at Roswell Park, and several other places these tests were also undertaken, and not one study has ever shown there to be any benefit, whatsoever, for a cancer patient.

So, as far as we know, there is not even a question in any oncologist's mind that this is a completely useless - harmless probably, but useless - drug and there is no basis in fact that would even suspect it would be useful. Whether or not the patients who claim to have been cured by it ever had cancer - one of the statements I heard made just yesterday by some specialists that we had at Beth Israel, was that most of the patients who were supposed to have been cured by laetrile never had - as you heard before - the tissue diagnosis of cancer made. So, I would go on record, myself, as saying that laetrile is a hoax on the public and people who take it at the expense of good chemotherapy or radiation or surgery are really committing suicide.

SENATOR MC GAHN: Doctor, I was happy to hear you make the statement that in most instances a tissue diagnosis had not been established and a lot of people were under the assumption that they did have a malignancy when, in truth, they did not.

DR. COHEN: Yes.

SENATOR SKEVIN: Doctor, one question and maybe you would comment on it. You indicated you were essentially in the treatment of cancer and not a medical politician.

DR. COHEN: Yes.

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SENATOR SKEVIN: You are not talking about people like Dr. McGahn here.

DR. COHEN: Oh, no, no, no. These are my friends. No, these are my friends.

SENATOR SKEVIN: You are talking about people - medical men who are--

DR. COHEN: No.

SENATOR MC GAHN: John, since Bergen County went for casino gambling, you can say anything you want to.

(laughter)

SENATOR SKEVIN: Okay. Thank you, Doctor.

Dr. Preuss will be our next witness.

D R. P E T E R P R E U S S: Senator Skevin, members of the Commission, I thank you very much for inviting the Department of Environmental Protection to participate once again in the important work of this Commission. I appear sort of as a coda, I guess, after the medical testimony that we have heard. I would like to say that I very much agree with some of the things that I have heard today, especially the fact that any attack that we make on cancer and on the high rates of cancer must be very broadly based and must have a broad focus.

I think that when Commissioner Bardin testified before you at your first meeting and discussed, somewhat, the executive order that Governor Brendan Byrne had issued in May, I think it was very clear that the intent was to have this kind of broad focus.

Now, at the Commission's second hearing, Dr. Paulson and I discussed how cancer-causing agents are identified, how Federal agencies move to control exposure to these chemicals and, in particular, we discussed our Department's overall approach toward the goal of reducing the exposure of New Jersey residents to such agents in the environment.

Now, I belive the reason that you have invited us again today, Senator Skevin - the reason I am here, very specifically - is because as part of the work of the Cabinet Committee on Cancer Control, Governor Byrne, yesterday, released a draft list of environmental carcinogens that our Department has compiled. You requested that we come and discuss this list with the members of the Commission.

Now, I would like to point out that this list is, in fact, a draft. It is being widely disseminated for review and comment and a final version will be prepared after careful consideration of all comments that we receive.

Now, I would like to just take one minute, again, to discuss some of the background to this list. When we were here last time, we mentioned that there were a very large number of substances that had been identified as potentially carcinogenic, that is, there were about 2,000 substances that have now been identified as possibly being involved in causing cancer - or that had been shown to cause cancer in experimental systems and in animals.

From the beginning of our efforts, we have recognized the need to develop a method to narrow our regulatory focus considerably so that we could zero in on those carcinogens that were both potent and also likely to be in common use in New Jersey and, thus, potentially reach large numbers of people.

By way of background, I think we have to consider the general pattern of the introduction of new chemicals into the human environment. Each year, scores - or hundreds - of new substances are put into commercial use. Until now, in most cases only relatively simple laboratory tests were generally performed to determine the short-term so-called acute effect of each new substance. These tests have included measurement of acute toxicity over hours or days, skin irritation, and others.

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In contrast, very few, if any, new substances have been adequately tested for long-term so-called chronic effects, such as cancer-causing potential, the ability to affect the genetic materials in the cells nucleus, or the potential to cause malformed, or abnormal offspring. Such drastic effects have usually come to our attention after the fact, for example when consumers of a product reported ill effects, or when environmental damage became apparent.

I think that one classic example, which is illustrative of this situation is the family of chemicals known as PCB's, or, more accurately the polychlorinated biphenyls. PCB's were introduced into commerce in the 1930's. They became widely used all over the world because of certain specific properties that they had. The greatest use came in the electrical industry, where they rapidly replaced many other products. PCB's became the substance of choice for use as insulators in electrical transformers and capacitors and were responsible for sales of 72 million pounds, per year, in the United States alone, during 1970.

It was not until 1966, about 35 years after their original commercial use, that the first indications were obtained that PCB's might be widespread environmental contaminants. A scientist in Sweden, who was looking at DDT residues in birds and certain other wild life, found a number of substances that he could not identify. They were ultimately identified as PCB's by him. Since that time, PCB's have been shown to be present wherever they have been looked for, in all parts of the environment, in all parts of the globe.

Since that time also, PCB's have been shown to cause birth defects in monkeys - given at very low concentrations, I might add. A fairly recently report, which appeared about one month ago, indicates that they may be correlated with human cancer in industrial settings.

Now, to complete this little example, I must say that they Monsanto Company, who is the sole producer in the United States of these PCB's, has acted, voluntarily, to restrict the use of these substances and has said that they would phase them out when substitutes became available.

Now, the new Federal Toxic Substances Control Act, which was just recently signed into law, mandates the phasing out of these substances and the use of these substances in this country.

I went into this for a minute because I think that the history of these compounds, these PCB's, well illustrates the many problems that we are facing in dealing with environmental contaminants that may be associated with cancer or other serious disease conditions. If we paint this picture broadly, we are faced today with a situation that thousands of substances already in every day commercial use have not been adequately tested for long-term effects, such as the potential to cause cancer. We are faced with a situation that there are hundreds of new chemical substances being developed and produced annually which are also not subject to adequate testing. We are faced with a situation that there are an unknown number of such substances present in the environment in unknown quantities and we are faced with a situation where there is a lack of scientific evidence as to the effects on human beings, or other creatures, to long-term exposures to these substances, both alone and in combination with others.

Now, some of these inadequacies will be dealt with under the new Federal Toxic Substances Control Act. In particular, this law requires the pre-market testing of newly-developed substances for certain toxic properties. Other parts of this complex problem will be approached by Federal and State agencies, although only some

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parts of this effort have so far been defined.

As I said at the beginning, in his Executive Order, Governor Byrne assigned responsibilities to various State agencies for a broad, comprehensive cancer program. Two of the areas that Governor Byrne has identified as appropriate for a State role are the monitoring of the environment in order to determine the types and amounts of substances present and, two, determining the use patterns of carcinogenic substances in the State of New Jersey.

The basic reason for moving in these two areas is to obtain information as to the actual and potential exposure of New Jersey residents to cancer-causing substances, or, in other words, to find out more precisely what is happening in New Jersey.

In order to conduct these programs, DEP has prepared a draft list of substances that we believe may well be of concern in terms of their potential long-term effects on people in New Jersey. This list has already been sent to a large number of people and organizations for review and comments. And, after review of the comments received, we will prepare another version for use as a basis for the two programs that I have mentioned, as well as a guidance document for other parts of the State's cancer control effort.

The material that you gentlemen received now, and which you have coming in the mail to you again, contains the list as well as the testimony. I would like to discuss with you now what the list is and, just as importantly, what the list isn't and how we prepared it.

When we approached the job of compiling this list and of putting it together, we first had to choose which information sources to use. We decided not to do original laboratory research of our own and also not to rely on original research reports from the primary scientific journals. Rather, we chose to use scientific documents prepared by respected national and international scientific organizations which review and summarize the results of such original research.

One of our starting points was the compilation of known and suspected carcinogens published by the National Institute of Occupational Safety and Health, which I belive many of you are familiar with already. It is called Suspected Carcinogens, A Sub File of the NIOSH Toxic Substances List. This volume lists all those substances which have been shown to cause cancer either in animals or in man. There are upwards of 2,000 substances in this volume.

This, however, was not sufficient for our needs in that there was no attempt made in this book to evaluate the scientific evidence. Fortunately, such an evaluation has been carried out and has been published in the nine volume work called, "Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man." I have one such example here. There are nine such volumes. And, basically, it is an evaluation of the cancer-causing potential of the substances that have been published elsewhere.

Now, this nine volume work was prepared by an international committee which evaluated the available scientific literature from around the world in order to determine whether the current state of research permitted a sound conclusion as to whether any given chemical was carcinogenic. This evaluation is accepted as definitive by many agencies of the U. S. Government dealing with cardinogens, including the National Cancer Institute, the National Institute of Occupational Safety and Health and the Occupational Safety and Health Administration.

Now, I would like to state that this compilation is not meant to cover all possible types of environmental carcinogens. It is limited to chemical substances

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that are used or produced by industrial facilities. As a result, a number of important types of carcinogens are not included. And, examples of those - some of which have been discussed already - are those that may be formed through secondary reactions in the environment, that is, when one substance is omitted and it reacts to form a carcinogen in the environment.

The chlorinated hydrocarbons are another example that may be formed during the chlorination of water, and food additives, drugs and substances produced as a by-product in industrial or fuel-burning processes are other examples.

Now, to further home these international and national reviews, we imposed a further set of criteria to reduce the number of chemicals to those of greatest potential concern. There were two. The first one was that each substance must be a demonstrated carcinogen in either animals or humans and, two, in adddition, each substance must be in widespread use in this country or each substance had to be a particularly potent cancer-causing agent.

Using these criteria, we were able to extract from the list of 2,000 substances, a list of about 80. Again, this does not mean that these are the most toxic substances in the environment, nor that these substances may result in the greatest detrimental health effects to humans. We are currently working on ways to supplement this list of 80 with other substances that may be toxic to human beings and that may cause other detrimental effects, not cancer. In other words, it has been stated very frequently that just the same way that we were able to prepare these cancer maps that were shown to you before, we could do the same thing for maps of heart disease and show that there are substances that are clearly related to the development of heart disease, as well as many other toxic and medical problems.

What this list is, is, it specifies our current judgment as to those substances which the scientific literature indicates are linked to cancer, which may well be present in the New Jersey environment and which should be looked at in more detail.

More specifically, this list and its supplements will be used as the basis for DEP's monitoring efforts and for a survey of the industrial use of such chemicals. Now, the monitoring will be of several kinds. We will try to determine which of the substances on the list are present or absent in the ambient environment and the quantities that are present. We will focus on certain substances to determine in greater detail where they are and how they change over time throughout the State. We will monitor the emissions from industrial facilities of cerain of these substances in order to measure how much is going into New Jersey's environment.

Now, the industrial survey we have discussed with this Commission previously. It is planned and will be implemented in conjunction with the Department of Labor and Industry.

In brief, though, what we will try to determine through this survey is the use, production and emission of the substances of this list into the New Jersey environment. It is our belief that this compilation and the two programs based on it, together with other programs in DEP and in other State agencies will permit the determination of New Jersey populations at high risk from cancer, as well as help define the regulatory programs necessary to reduce this risk.

Through our testimony at previous Commission hearings, Senator Skevin, and by other means, we have kept this Commission informed of our efforts and we plan to continue to do so.

If you have any questions, I would be very happy to answer them.

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SENATOR SKEVIN: Thank you for your fine report, Dr. Preuss. In terms of carcinogens, do we have a definition in this report and who provided that definition?

DR. PREUSS: It is very difficult to define. Basically, what we have chosen are those substances that the International Committee of the World Health Organization decided there was sufficient scientific evidence available to show that these substances caused cancer in either animals or in humans.

SENATOR SKEVIN: This is an academic list in other words, Doctor. These are known to be carcinogenic, is that what you are saying?

DR. PREUSS: They have been demonstrated to be carcinogenic, yes.

SENATOR SKEVIN: But no State tests were made to determine any substances to be included in this hearing?

DR. PREUSS: No, absolutely not.

SENATOR SKEVIN: Do we know if any of these substances are in New Jersey?

DR. PREUSS: We do know that some of these substances are in New Jersey. The list is divided into two parts. The first part are those substances which are used in quantities greater than 400,000 pounds in the United States - that is, the high-use chemicals in this country which are also carcinogenic.

SENATOR SKEVIN: Do we know the actual origin in New Jersey?

DR. PREUSS: No, that is exactly what our industrial survey will attempt to determine, Senator.

SENATOR SKEVIN: I see.

DR. PREUSS: What we do know is that when the Environmental Protection Agency came up here to sample the ambient air several months ago to determine what organic compounds were present in the air, that a number of the substances that are on this list were, in fact, found in the New Jersey air.

SENATOR SKEVIN: Do we know what industries were likely to produce these substances?

DR. PREUSS: I think we have listed for each of these substances the type of industry, yes. The specific industry, again, would be the information we would be trying to obtain through our survey.

SENATOR SKEVIN: It is likely that we could find any of these substances in combination in any given area?

DR. PREUSS: I think it is highly likely, Senator.

SENATOR SKEVIN: And has the Department of Environmental Protection, to your knowledge, ever acted against any specific industrial plant because it was emitting any carcinogenic substance into the atmosphere?

DR. PREUSS: I would say that the answer is yes, although in most cases we have not acted because of the fact that they were carcinogens. What we have done is act because they were in violation of one or another of our environmental standards. Although in recent times there have been several actions taken by the Department aimed specifically at situations where the substance was a carcinogen, yes.

SENATOR SKEVIN: And you feel you have the authority to do that under the present statute?

DR. PREUSS: In certain cases, yes.

SENATOR SKEVIN: Do you need any additional authority?

DR. PREUSS: Not being a lawyer, I have difficulty answering that, Senator.

SENATOR SKEVIN: In terms of the testimony we heard from Doctor Demopoulos on the relationship of industrial pollutants to the fatalities, do you agree with that conclusion, Dr. Preuss?

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DR. PREUSS: Partially, I do. I agree with the fact that when we say that 80% or 90% of cancers are environmentally induced, we definitely do not mean that these 90% are caused by industrial contaminants. We are, in fact, using the word "environmental" very broadly. It is not clear to me what percentage of that 90%, though, is related to industrial contaminants or to other factors.

I think, as you pointed out with the connection between smoking and asbestos workers, for example, the factor was ninety times as high among those who smoked and were exposed to asbestos. These are the kinds of things that we are not really very sure about at this time. I think there are many cases--

SENATOR SKEVIN: Excuse me, may I interrupt you? When you say "we are not very sure", is that the scientific community?

DR. PREUSS: Yes, I meant that at the general "we."

SENATOR SKEVIN: The scientific community?

DR. PREUSS: Yes, not the Department.

I think we have reached the point where, in a general sense, it is clear to us what some of the things are that are correlated to cancer, such as smoking. At the other extreme we have, for example, vinal chloride, which is an industrial pollutant and has been used in industry to produce many types of plastic, mainly polyvinalchloride plastic, where, again, it has been shown to be connected to cancer. But, again, you have to remember - and I think this is the crucial point here - that we have been able to correlate cancer and environmental contaminants only in very rare cases because it has been a very rare type of cancer. I mean, to look at an environmental contaminant and say, "Does this environmental contaminant cause lung cancer?", is very difficult because of the great prevalence of lung cancer and our inability to differentiate amongst several types of lung cancer.

We spotted vinal chloride because it caused a very, very rare type of liver cancer. We spotted asbestos. We spotted benzidine. We spotted other chemicals because they caused a very rare type of disease. In Salem County, for example, we had such a high incidence of bladder cancer that it stood out because it was so unusual. But, when we are trying to pick it out from the mass of other cancers, it is very often very difficult. So, epidemiological studies may not be able to do this for a good long time.

Now, again, the question is, what percentage of cancer is related to what. Dr. Demopoulos came up with the figure of 600. I recall at a conference in Camden not too long ago, Dr. Louria from the medical school came up with a number of perhaps 1,400. Again, depending upon how we do this and depending upon the type of cancer, we are talking about some percentage, whether it is 5% or 30%, I don't think is clear at this time.

SENATOR SKEVIN: In terms of the list that you submitted through the Governor's office, Dr. Preuss, do we have anything new there that we didn't have, let's say, for the last 10 years, in terms of information as to cancer-causing substances?

DR. PREUSS: Oh, absolutely. I don't recall which one of the witnesses mentioned that we have had this information available for 10 or 15 years. I think I would disagree with that point very strongly. We have known that certain substances cause cancer for very many years, there is no question about that. What we have not realized is the dimensions of the problem - that so much cancer is, in fact, related to environmental factors. We have simply not looked for many of these things in the past. So, I think this list and the work of, for example, the World Health Organization are quite new and are quite significant contributions in our thinking about this problem.

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SENATOR [illegible] I have no further questions. Dr. McGahn?

SENATOR [illegible] No.

SENATOR [illegible] Senator Dumont?

SENATOR [illegible] Do you think the publicity you have given with regard to smoking has really made any great amount of difference? I notice that some men have given up smoking but I don't notice any difference among women, with all due respect to them.

DR. PREUSS: Yes, as a matter of fact I understand from the statistics that there is an increase in smoking. I feel very strongly about this point, Senator. I don't think we have done enough and this is a point where I agree with every single one of the previous speakers here. I am convinced that if we could eliminate smoking in the State of New Jersey, we would reduce the number of cancer cases by more than any other thing we could do. So, I think this is a subject we have to pay much more attention to, much greater attention to. I think it runs the gamut from regulation to education, just as the previous speakers have mentioned.

SENATOR DUMONT: It would probably reduce a great many heart problems too.

DR. PREUSS: I am sure of it, yes.

SENATOR PARKER: Does that include smokers as opposed to non-smokers? In other words, do you find the incidence of cancer in non-smokers because he has to inhale this smoke?

DR. PREUSS: The epidemiology of that is quite difficult. There seems to be some indication that non-smokers are affected as well, but it is certainly nowhere the rate of the smoker. Again, among smokers, I think you have some very fine evidence now that as the number of cigarettes that you smoke increases, the chances of your getting lung cancer are much higher. Again, for those people who stop, the rate decreases. There is nearly no question about it. How this affects the non-smoker, is a little bit more difficult to say.

SENATOR PARKER: Smoking doesn't bother me, it is the alcohol that has me worried.

SENATOR DUMONT: Is smoking actually the number one cause of cancer among all of the different [illegible]?

DR. PREUSS: If we break cancer down into the component types, I would say yes. Or, I should say the one that we have identified, yes.

SENATOR DUMONT: Thank you.

SENATOR MC[illegible]: Barry, has your skin always been yellow, or is that just--

SENATOR PARKER: Excuse me, do I smoke? No, I do not smoke.

SENATOR MC[illegible]: No, I heard something about drinking. I was talking about drinking.

SENATOR PARKER: I don't partake of alcohol either.

SENATOR MC[illegible]: I think, if I may make one statement, John, that the testimony that we have been receiving almost shows one thing, the more the scientific community learns about cancer, the less it actually knows. We certainly recognize the fact that there are a tremendous amount of other factors - family incidence, if you will; the unusual circumstance of spontaneous remission of a tumor, which has been definitely proven to [illegible] very beginning, so that then you have immunological factors actually working here, susceptibility of certain individuals and, of course, the resistance of others. I think as doctors mentioned this morning, the area of synergism in these particular things is really the unknown quantity and it is easy

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to deal in terms of statistics, but when you are dealing with one single individual patient, oftentimes that patient does not conform to the statistics.

DR. PREUSS: Absolutely. I would just add to your point, Senator McGahn - and I absolutely agree with that - and say that our efforts, as some of the other speakers have said, should be in those areas where we can have some concrete, positive effect as quickly as possible. I think that is either in the area of early detection or prevention. I think that is the direction we have to go in and those areas where we really are not sure, I think we may be able to defer.

SENATOR SKEVIN: You would agree with the prior speakers who want to do something here rather than to wring their hands and talk about it, like we have for last 10 or 20 years?

DR. PREUSS: Well, in all of the conversations we have had, Senator, you know that is exactly how I feel. I feel very strongly about that.

SENATOR SKEVIN: Thank you very much, Doctor.

This will conclude our hearing. We will have Dr. Demopoulos give a rebuttal.

DR. DEMOPOULOS: No, it is not a rebuttal. I happen to have Dr. Louria's reprint of the talk he gave in Cherry Hill and his calculations came out to almost precisely what we concluded. It is 600 excess deaths that might be industrially related. I have it right here. It is not 1,400.

SENATOR SKEVIN: We were trying to get that transcript, Dr. Demopoulos. If we could make it a part of our record, I would appreciate it.

SENATOR MC GAHN: Dr. Louria was in Atlantic City last Saturday also at the cancer meeting down there and he gave essentially the same presentation.

SENATOR SKEVIN: Thank you. The hearing is concluded.

(Hearing Concluded)

SENATE, No. 1540

STATE OF NEW JERSEY

INTRODUCED JUNE 14, 1976

By Senator SKEVIN

Referred to Committee on Revenue, Finance and Appropriations

AN ACT to amend and supplement the "Cigarette Tax Act," approved April 29, 1948 (P. L. 1948, c. 65).

BE IT ENACTED *by the Senate and General Assembly of the State of New Jersey:*

1. Section 301 of P. L. 1948, c. 65 (C. 54:40A-8) is amended to read as follows:

301. Tax imposed; rate. A tax is hereby imposed on the sale, use or possession for sale or use within this State of all cigarettes at the rate of [$0.09½] *$0.10* for each 10 cigarettes or fraction thereof.

2. Section 401 of P. L. 1948, c. 65 (C. 54:40A-11) is amended to read as follows:

401. Director to provide revenue stamps. The taxes imposed and levied by this act shall be paid through the use of stamps, except as provided in section 205 (Consumers) of this act. The director shall secure stamps of such designs and denominations as he shall prescribe, suitable to be affixed to packages, and provide for the sale thereof to licensed distributors. Only licensed distributors shall affix and cancel stamps and no distributor shall affix or cancel any stamp except at the tax rate in effect on the date of such affixing or cancellation; except that on the effective date of a tax rate increase, licensed distributors and wholesale dealers must take a physical inventory of cigarettes on hand at the close of business prior to the date of the tax increase and must pay an additional tax of [$0.05] *$0.01* per package of 20 cigarettes, for all cigarettes bearing stamps at the rate in effect prior to the tax increase. The director shall prescribe the method of collecting the additional tax. The director shall not authorize any person to sell revenue stamps except his duly constituted agents and assistants.

EXPLANATION—Matter enclosed in bold-faced brackets [thus] in the above bill is not enacted and is intended to be omitted in the law.

On sales of revenue stamps the director shall allow, as compensation for the services and expenses of the distributor in affixing and handling of such stamps, a discount of [1.46%] *1.38%* of the face amount of any sale of 1,000 stamps or more; provided, that the distributor has complied with all the provisions of this act. No discount shall be allowed on any sale of less than 1,000 stamps and stamps shall not be sold in blocks of less than 100 stamps.

3. (New section) From the effective date of this amendatory and supplementary act, in each fiscal year, an amount shall be set aside from the revenues collected pursuant to the "Cigarette Tax Act," which amount shall be equal to the amount raised by levying a tax at the rate of $0.005 for each 10 cigarettes or fraction thereof. This amount shall be used exclusively to finance scientific and medical research into the causes, diagnosis and treatment of cancer in human beings. At least one half of such amount shall be paid to the State Department of Health, which shall use such funds to provide matching State funds for Federal grants to New Jersey institutions conducting such cancer research. Institutions which may receive a portion of the remainder of such amount shall be limited to cancer research institutes located in the State of New Jersey and certified by the Commissioner of the Department of Health to receive such funds, to the College of Medicine and Dentistry of New Jersey, and to Rutgers, The State University. Any moneys paid to such institutions pursuant to this section in any fiscal year shall be in addition to any other funds paid by the State to such institutions. Each institution receiving funds pursuant to this section shall annually submit a report to the Legislature and to the Commissioner of the Department of Health, setting forth the manner in which such funds were used for that fiscal year and projecting the manner in which funds which may be so received in the coming fiscal year shall be used.

4. This act shall take effect at 12:01 a.m. following the date of enactment.

STATEMENT

This bill would increase the cigarette tax by 1¢ per package. It would raise approximately $8 million a year. The bill provides that the amount so raised shall be used exclusively to finance scientific and medical research into the causes, diagnosis and treatment of

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cancer. One half of such amount shall go to the State Department of Health to provide matching funds for Federal grants. Institutions to receive the remainder of such funds are limited to cancer research institutes located in New Jersey and certified by the Commissioner of the Department of Health to receive such funds, to the College of Medicine and Dentistry of New Jersey, and to Rutgers, The State University. The bill requires such institutions to annually report to the Legislature how such funds are used.

The cancer rate among New Jersey residents is the highest in the United States for many of the most frequently fatal types of cancer. The incidence of cancer in New Jersey is 14% higher than in the United States as a whole.

Yet, New Jersey has qualified for relatively little of the $600 million the National Cancer Institute made available for cancer research last year. In 1975, New Jersey received $7,598,000.00 or $1.04 per capita. New York State received $107,251,000.00 or $5.92 per capita; Massachusetts received $42,361,000.00 or $7.27 per capita; Pennsylvania $34,788,000.00 or $2.94 per capita; and Maryland, $75,211,000.00 or $18.35 per capita. Figures show that if New Jersey were to attract its proportionate share of the NCI cancer grants and contracts in relation to neighboring states, it would have received in 1975, $37,458,000.00 which reflects a loss of about $30 million in 1 year.

To correct this situation and thereby receive its fair share of cancer research funds from Federal and other sources, New Jersey must provide stable funds to attract cancer research scientists and provide facilities and services where productive cancer research can be conducted.

Cigarette smoking is one of the major causes of lung cancer and there is a measure of propriety in financing New Jersey's cancer research capability by imposing a small tax on cigarettes.

SENATE, No. 1758

STATE OF NEW JERSEY

INTRODUCED NOVEMBER 15, 1976

By Senators SKEVIN, McGAHN, MARESSA, MARTINDELL and PARKER

Referred to Committee on Institutions, Health and Welfare

AN ACT concerning the control of cancer in New Jersey, providing for a registry of the incidence of cancer and other tumorous and precancerous diseases, authorizing the State Department of Health to require certain persons to report such cases, and making an appropriation therefor.

BE IT ENACTED *by the Senate and General Assembly of the State of New Jersey:*

1. The Legislature hereby finds and declares:

a. That New Jersey is currently suffering from the highest overall mortality rates for cancer in the Nation;

b. That certain forms of cancer are now believed to be attributable to environmental factors which, if controlled, can significantly reduce cancer incidence in this State;

c. That more complete and more precise statistical data are necessary to determine the correlations between cancer incidence and possible environmental factors and to evaluate cancer treatment and prevention measures that are currently in progress; and,

d. That a cancer registry would thus provide a vital foundation for a concerted State effort to reduce the incidence of environmentally related cancer in this State.

2. The State Department of Health shall establish and maintain an up-to-date registry which shall include a record of cases of cancer and specified cases of tumorous or precancerous disease that occur in New Jersey, and such information concerning these cases as it shall deem necessary and appropriate in order to conduct thorough and complete epidemiologic surveys of cancer and cancer-related diseases in this State and to apply appropriate preventive and control measures.

3. a. The Commissioner of Health, in consultation with the Public Health Council, shall require the reporting of cases of cancer and other specified tumorous and precancerous diseases, and the submission of such specified additional information on reported cases or control populations as he deems necessary and appropriate for the recognition, prevention, cure or control of such diseases.

b. Pursuant to subsection a. of this section, the Commissioner of Health is hereby authorized to adopt and promulgate, in the manner prescribed by the applicable provisions of the "Administrative Procedure Act" (P. L. 1968, c. 410; C. 52:14B-1 et seq.), rules and regulations specifying the health care providers, individuals, and other organizations obliged to make the report and submissions required by subsection a. of this section, the related information to be included in such reports, and the methods for such reporting.

4. The reports made pursuant to this act are to be used only by the State Department of Health and such other agencies as may be designated by the Commissioner of Health and shall not otherwise be divulged or made public so as to disclose the identity of any person to whom they relate; and to that end, such reports shall not be included under materials available to public inspection pursuant to P. L. 1963, c. 73 (C. 47:1A-1 et seq.).

5. No individual or organization providing information to the State Department of Health in accordance with this act shall be deemed to be, or held liable for, divulging confidential information.

6. Nothing in this act shall be construed to compel any individual to submit to medical or health department examination or supervision.

7. There is hereby appropriated to the Department of Health the sum of $200,000.00 to establish and administer the registry created by section 2. of this act and to process and analyze the information recorded therein.

8. This act shall take effect 30 days following its enactment.

STATEMENT

The purpose of this bill is to require the establishment and maintenance of a cancer incidence registry. Virtually all medical authorities agree that such a cancer data bank would serve as an invaluable tool in discovering patterns of cancer incidence and thus direct preventive efforts to those environments and geographic areas where they are likely to prove most effective.

RSV 0013812

SENATE, No. 3034

STATE OF NEW JERSEY

INTRODUCED JANUARY 24, 1977

By Senators SKEVIN, McGAHN, MARESSA, MARTINDELL, PARKER and DUMONT

Referred to Committee on Institutions, Health and Welfare

AN ACT establishing an early detection program for cancer and making an appropriation.

BE IT ENACTED *by the Senate and General Assembly of the State of New Jersey:*

1. The State Commissioner of Health shall initiate, develop and administer a program for the early detection and treatment of cancer. The State Commissioner of Health is hereby authorized, in conjunction with other concerned public or private agencies to:

a. Appoint and employ such professional and other personnel as may be deemed necessary for the efficient operation of such a program;

b. Arrange for, establish and maintain a series of clinics throughout the State for the early detection, diagnosis, and treatment of cancer;

c. Inform the general public and interested persons and groups of methods for detecting, preventing and treating cancer;

d. Conduct or arrange for continuous basic research, so that New Jersey contributes its share to the advance of scientific knowledge in respect to: (1) the causes, prevention and early treatment of cancer, and (2) the effectiveness of public and private cancer control programs;

e. Initiate a training program for paramedics in cancer detection, in conjunction with the State's medical schools and to utilize said trained paramedics in the early detection of cancer;

f. Enter into lease, rental or similar agreements, in the name of the State, with respect to suitable clinic or hospital facilities, and to accept and engage for the use without cost of such facilities for the purposes expressed herein;

g. Utilize, for the purposes of this act, the available services and asistance of any persons, groups, organizations or institutions, professional or otherwise, and to contract for the services of any qualified organizations or groups;

h. Formulate, promulgate, amend and repeal reasonable rules and regulations for the administration of the program for the early detection of cancer.

2. There is hereby appropriated for the administration of this act the sum of $500,000.00 to the Department of Health.

3. This act shall take effect immediately.

STATEMENT

This bill is one of a series of bills developed by the Senate Commission on the Incidence of Cancer in New Jersey. It provides for and funds a program for the early detection and treatment of cancer by the State Commissioner of Health.

RSV 0013814

SENATE, No. 3035

STATE OF NEW JERSEY

INTRODUCED JANUARY 24, 1977

By Senators SKEVIN, McGAHN, MARESSA and MARTINDELL

Referred to Committee on Energy and Environment

AN ACT concerning the control of carcinogens and the protection of the public from cancer by the Department of Environmental Protection and by the Department of Health and establishing a Cancer Control Council with certain duties, and making an appropriation.

BE IT ENACTED *by the Senate and General Assembly of the State of New Jersey:*

1. This act shall be known and may be cited as the "Cancer Control Act."

2. The Legislature finds and determines that New Jersey has one of the highest overall mortality rates for cancer in the Nation; that for a large number of different cancers, the State of New Jersey as a whole and many counties within the State rank significantly above the National average; that some cancers are troublesome, painful and treatable while most others are serious and potentially fatal; that the economic costs of cancer are immense, including the cost of hospital care, doctor bills, medication and the loss of production from and salaries of affected workers; and that the social and psychological costs to the victims of cancer and their families are enormous.

The Legislature, therefore, declares that the cumulative losses of the State's citizens shall be counteracted by a major research and regulatory program administered by the State Departments of Health and Environmental Protection in conjunction with the Cancer Control Council established herein.

3. For the purposes of this act, unless the context clearly requires a different meaning:

a. "Cancer" means a malignant tumor characterized by potentially unlimited growth with local expansion by invasion and systemically by metastasis.

b. "Carcinogen" means a substance or agent producing or inciting cancer and shall include every substance or agent identified as a probable or proven human carcinogen by the National Institute of Occupational Safety and Health pursuant to Public Law 91-596.

4. The Commissioner of Environmental Protection, with the approval of the Cancer Control Council, shall formulate, promulgate, amend or repeal rules and regulations prohibiting, conditioning and controlling the release of carcinogens onto or into the atmosphere, waters or lands of this State in such quantities which cause or may tend to cause adverse effects on man or the environment and the production, manufacture, sale and labeling of products containing carcinogens which cause or may tend to cause adverse effects on man or the environment.

5. The Commissioner of Health, with the approval of the Cancer Control Council, shall formulate, promulgate, amend or repeal rules and regulations prohibiting, conditioning and controlling the use of products containing carcinogens which cause or may tend to cause adverse effects on the health of their users or on any other persons.

6. No person shall produce, manufacture or use or release onto or into the atmosphere, waters or lands of this State any of the following carcinogens: 4-nitrobiphenyl, alpha naphthylamine, 4, 4-methylene bis (2-chloroaniline), methyl chloromethyl ether, 3, 3-dichlorobenzidine, bis-chloromethyl ether, beta naphythylamine, benzidine, 4-aminodiphenyl, ethyleneimine, beta propiolactone, 2-acetylaminofluorene, 4-dimenthylaminoazobenzene, N-nitrosodimethylamine, asbestos, vinyl chloride.

7. a. There is hereby created in the Department of Environmental Protection a Cancer Control Council which shall consist of seven members, three of whom shall be the Commissioner of Environmental Protection, the Commissioner of Health, and the Commissioner of Labor and Industry, or their designees, who shall serve ex officio, and four citizens of the State representing the general public to be appointed by the Governor, with the advice and consent of the Senate. The members shall select a chairman and vice chairman of the council from the members representing the general public.

b. Of the four members first to be appointed, one shall be appointed for a term of 1 year, one for a term of 2 years, one for a term of 3 years and one for a term of 4 years. Thereafter all appointments shall be made for terms of 4 years. All appointed members shall serve after the expiration of their terms until their

respective successors are appointed and shall qualify, and any vacancy occurring in the appointed membership of the council shall be filled in the same manner as the original appointment for the unexpired term only, notwithstanding that the previous incumbent may have held over and continued in office as aforesaid.

c. Members of the council shall serve without compensation, but shall be entitled to reimbursement for expenses in attendance at meetings of the council and in performance of their duties as members thereof.

8. The Cancer Control Council shall be empowered to:

a. Approve or disprove of any rule or regulation or any alteration thereof, proposed by the Commissioners of Environmental Protection or Health pursuant to section 4 or 5 of this act.

b. Study the State programs which may relate to the cause or cure of cancer and to make those recommendations to the concerned commissioner and to the Legislature which it deems necessary for the proper conduct and improvement of such cancer related programs.

c. Hold public hearings at least once each year in regard to existing or proposed cancer control statutes, rules, regulations and programs and upon the state of the art and technical capabilities and limitations in cancer control and report its recommendations thereon to the concerned commissioners and Legislature.

d. Recommend that the concerned State department grant any moneys made available to it by future legislation or by the Federal Government for cancer research projects to the most qualified applicants and to those applicants proposing to perform the highest priority research.

9. The Departments of Environmental Protection and Health shall have power, in addition to those granted by any other law, to:

a. Conduct and supervise research programs for the purpose of determining the causes and cures of cancer and the effects and hazards of the distribution and use of carcinogens on man and his environment; and in furtherance of this research effort the commissioner shall consider, as a primary source of assistance the American Cancer Society, the College of Medicine and Dentistry of New Jersey and Rutgers, The State University;

b. Conduct and supervise Statewide programs of cancer control education including the preparation and distribution of information relating to cancer control;

c. Enter and inspect any building or place, except private residences, for the purpose of investigating an actual or suspected

violation of law relating to carcinogens and ascertaining compliance or noncompliance with any rules, regulations or orders of the department and for the purpose of investigating suspected health hazards, documenting exposures, conducting epidemiological studies to establish a casual relationship between a suspect carcinogenic substance and cancer and to cooperate with the National Institute of Occupational Safety and Health and the Occupational Safety and Health Administration;

d. Receive or initiate complaints of violations of applicable laws, rules, regulations and orders relating to carcinogens and institute legal proceedings for the prevention of such violations and for the recovery of penalties, in accordance with law;

e. Cooperate with, and receive money from, the Federal Government, the State Government, or any county or municipal government or from private sources for the study and control of cancer.

10. If any person violates any of the provisions of this act or any rule or regulation promulgated pursuant to the provisions of this act, the concerned department may institute a civil action in a court of competent jurisdiction for injunctive relief to prohibit and prevent such violation or violations and the said court may proceed in the action in a summary manner.

Any person who violates the provisions of this act or any rule, regulation or order promulgated pursuant to this act shall be liable to a penalty of not more than $3,000.00 for each offense, to be collected in a civil action by a summary proceeding under the Penalty Enforcement Law (N. J. S. 2A:58-1 et seq.) or in any case before a court of competent jurisdiction wherein injunctive relief has been requested. The Superior Court, County Court and county district court shall have jurisdiction to enforce said Penalty Enforcement Law. If the violation is of a continuing nature, each day during which it continues shall constitute an additional, separate and distinct offense.

The concerned department is hereby authorized and empowered to compromise and settle any claim for a penalty under this section in such amount in the discretion of the department as may appear appropriate and equitable under all of the circumstances.

11. Whenever an agent of either department finds, or has probable cause to believe that any carcinogen is being produced, manufactured, sold, labeled, released or used in violation of any provision of this act or any rule, regulation or order promulgated pursuant to this act, he may affix to such substance a tag or other appropriate marking giving notice that such substance has been

detained or embargoed, and warning all persons not to remove, dispose, or use such substance until permission is given by his department or a court. It shall be a violation of this act for any person to remove, dispose, or use any detained or embargoed carcinogen without such permission.

12. The powers, duties and functions vested in the State Department of Environmental Protection or the State Department of Health under the provisions of this act shall not be construed to limit in any manner the functions, powers and duties vested in either department under any other provisions of law.

13. If any provision of this act or the application thereof to any person or circumstance is held invalid, the remainder of the act and the application of such provision to persons or circumstances other than those to which it is held invalid, shall not be affected thereby.

14. This act shall be liberally construed to effectuate the purpose and intent thereof.

15. There is hereby appropriated for the administration of this act the sum of $500,000.00 to the Department of Environmental Protection and $500,000.00 to the Department of Health.

16. This act shall take effect immediately.

STATEMENT

Mortality and morbidity rates for cancer in New Jersey are among the highest in the Nation. The high incidence of cancer in New Jersey must be reduced to protect the health and welfare of the affected persons and our society as a whole. This bill authorizes the Commissioners of Health and Environmental Protection with the approval of the Cancer Control Council, to regulate the production, manufacture, sale, labeling and use of carcinogens and the release of carcinogens into the environment in order to protect the environment and the health of our people.

RSV 0013819

SENATE CONCURRENT RESOLUTION No. 3002

STATE OF NEW JERSEY

INTRODUCED JANUARY 11, 1977

By Senators SKEVIN, McGAHN, MARESSA, MARTINDELL, DUMONT and PARKER

Referred to Committee on State Government, Federal and Interstate Relations and Veterans Affairs

A CONCURRENT RESOLUTION memorializing the Governors and the Legislatures of the States of Connecticut, New York, Pennsylvania and Delaware, in conjunction with the State of New Jersey, to form a Regional Cancer Control Commission.

WHEREAS, The mortality rates for cancer have increased at an alarming rate throughout the United States and estimates indicate that one out of every four Americans now living will develop some form of cancer; and

WHEREAS, The Legislature of the State of New Jersey has expressed its commitment to the prevention and control of environmental cancer by the establishment of the Commission on the Incidence of Cancer in New Jersey which is currently considering the institution of several State programs designed to facilitate the solution to this grave problem; and

WHEREAS, The metropolitan States of the Mid-Atlantic region have some of the highest rates of cancer mortality in the nation; and

WHEREAS, New Jersey and each of her immediate neighbors has had a long history of cooperative efforts to construct and operate bridges and tunnels to protect the public interest; and

WHEREAS, Cooperative efforts by the various States located in the Mid-Atlantic megalopolis is absolutely essential to achieve the prompt and comprehensive identification, control and elimination of carcinogenic agents; now, therefore

BE IT RESOLVED *by the Senate of the State of New Jersey (the General Assembly concurring):*

1. That the Senate and General Assembly of the State of New Jersey do hereby memorialize the Governors and the Legislatures of the States of Connecticut, New York, Pennsylvania and Delaware, in conjunction with the State of New Jersey, to form a Regional Cancer Control Commission in order to promptly identify, control and ultimately eliminate all carcinogenic substances.

2. That duly authenticated copies of this resolution be transmitted to the Governors and the presiding officers of both houses of the Legislatures in the States of New York, Pennsylvania and Delaware.

STATEMENT

This resolution would memorialize the Governors and the Legislatures of the States of Connecticut, New York, Pennsylvania and Delaware, in conjunction with the State of New Jersey, to form a Regional Cancer Control Commission in order to promptly identify, control and ultimately eliminate all carcinogenic substances.

RSV 0013821

SENATE CONCURRENT RESOLUTION No. 3003

STATE OF NEW JERSEY

INTRODUCED JANUARY 11, 1977

By Senators SKEVIN, McGAHN, MARESSA, MARTINDELL, DUMONT and PARKER

Referred to Committee on State Government, Federal and Interstate Relations and Veterans Affairs

A CONCURRENT RESOLUTION memorializing the President and the Congress of the United States to indicate their concern about environmental cancer and to take certain appropriate actions to protect the health and well-being of the citizens of the United States.

WHEREAS, The mortality rates for cancer have increased at an alarming rate throughout the United States; and

WHEREAS, It is now estimated that one out of every four Americans now living will develop some form of cancer; and

WHEREAS, The State of New Jersey has one of the highest rates of cancer mortality in the nation; and

WHEREAS, The Legislature of the State of New Jersey has expressed its commitment to the prevention and control of environmental cancer by the establishment of the Commission on the Incidence of Cancer in New Jersey; and

WHEREAS, Said commission is currently considering the institution of several State programs designed to facilitate the solution to this grave problem; and

WHEREAS, The coordination of State and Federal efforts is absolutely essential to the prompt and comprehensive identification and control of carcinogenic agents; now, therefore

BE IT RESOLVED *by the Senate of the State of New Jersey (the General Assembly concurring):*

1. That the Senate and General Assembly of the State of New Jersey do hereby memorialize the President and the Congress of the United States to resolve that environmental cancer is one of

the leading problems facing this nation today and to provide the leadership and support necessary for the maximization of State and Federal efforts to find a coordinated solution thereto within the next decade.

2. That duly authenticated copies of this resolution be transmitted to the President of the United States, the President of the United States Senate, the Speaker of the House of Representatives and to each member of Congress elected from this State.

STATEMENT

This resolution would memorialize the Congress of the United States to resolve that environmental cancer is one of the leading problems facing this nation today and to provide the leadership and support necessary for the maximization of State and Federal efforts to find a coordinated solution thereto.

QUANTITATIVE RISK ASSESSMENT

FOR COMMUNITY EXPOSURE

TO VINYL CHLORIDE

Arnold M. Kuzmack
Office of Planning and Evaluation
U. S. Environmental Protection Agency
Washington, D. C. 20460

and

Robert E. McGaughy
Office of Health and Ecological Effects
U. S. Environmental Protection Agency
Washington, D. C. 20460

December 5, 1975

RSV 0013825

EXECUTIVE SUMMARY

Vinyl chloride has produced liver angiosarcoma, a very rare form of cancer, as well as other cancers and serious liver damage in occupationally exposed populations. Since it is generally considered prudent to assume that there is no threshold for chemical carcinogens, the small concentrations of vinyl chloride observed in the ambient air in the vicinity of plants producing vinyl chloride monomer (VCM) or polymerizing it to polyvinyl chloride (PVC) involve some public health risk. This paper reports the results of an analysis to estimate quantitatively the risk resulting from vinyl chloride emissions and assess the reliability of the estimates. In addition, a search was initiated of the residence of people who have died of liver angiosarcoma in order to detect evidence of adverse effects among people living near plants.

The analysis involves three steps: estimation of the number of people exposed, the concentrations of vinyl chloride to which they are exposed, and the number of cancers and other health effects resulting from this exposure. Of these, the last is by far the most difficult to make and involves the most conceptual problems.

The number of people living at various distances from each VCM and PVC plant was determined by an analysis of census data. All told, some 4.6 million people live within 5 miles of these plants.

Ambient concentrations of vinyl chloride in the vicinity of plants were determined using standard methods of diffusion modeling. Exposures at each location were adjusted to allow for the types, numbers and sizes of plants present and the meteorological conditions which affect the dispersion of pollutants. It was found that the 4.6 million people living within 5 miles of uncontrolled VCM or PVC plants are exposed to an average concentration of about 17 parts per billion.

Quantitative estimates of health effects likely to result from this exposure were made by predicting from animal data the probability that highly-exposed workers would get angiosarcoma, checking that prediction by calculating the same probability using epidemiological studies of workers, and then projecting the results to ambient concentrations around plants. Cancers other than liver angiosarcoma were assumed to occur at the same relative rate as at the higher doses. Birth defects have been reported in communities with VC and VCM plants. These effects are not considered in detail here because quantitative estimates of these effects can not be made at this time.

Projections from incidence rates in rats to incidence rates in highly-exposed workers were made by assuming that a lifetime exposure to rats would produce the same number of effects as a lifetime of exposure to humans, which means roughly that one year of exposure to rats yields the same incidence rate as 30 years of exposure to people.

It was found, using a linear dose-response model extrapolating to zero, that the probability of getting angiosarcoma due to a year of continuous exposure is 71 cases per year of continyous exposure per ppm of vinyl chloride per million people exposed. The corresponding probability of getting cancer of all sites is 150 cases per year of continuous exposure per per ppm of vinyl chloride per million people. On this basis the probability of getting liver angiosarcoma in workers exposed to 350 ppm for 7 hours per day, 5 days per week would be 0.0052 per person per year of exposure.

From epidemiological studies of exposed workers, it was estimated that the probability of getting angiosarcoma at some time in the worker's life per year of exposure is 0.0031. To arrive at this rate, the analysis considered the distribution of exposure durations among the 14 known occupational cases of angiosarcoma, as well as the time distribution of man-years of exposure in the industry. Two other important facts about vinyl chloride carcinogenesis resulted from this analysis: 1) at some time in their lives about 7.5% of all highly-exposed workers are expected to get liver angiosarcomas due to vinyl chloride exposure with double this rate of primary cancer at all sites combined. 2) Of all the cases of liver angiosarcoma which have this far been produced by vinyl chloride, only 38% of them have been diagnoses as of 1974.

After considering the error associated with the separate estimates based on animal and human data, it is concluded that the two approaches give results that are indistinguishable.

The projection to low doses was done using two separate mathematical models of the animal data: 1) the linear model, which assumes that the response rates are directly proportional to dose with no threshold; 2) the log-probit model, which assumes that the susceptibility of the population is log-normally distributed with dose. The former model is widely used for low-dose extrapolation in radiation carcinogenesis and is generally considered to be conservative for chemical carcinogens, and the latter is usually used for describing the dose-response relationship in the dosage range of animal experiments.

It was found that the rate of initiation of liver angiosarcoma among people living around VCM-PVC plants is expected to range from less than one to ten cases of liver angiosarcoma per year of exposure to vinyl chloride. The log-probit model gives predictions that are 0.1 to 0.01 times this rate. This wide range is an indication of the uncertainties in extrapolation to low doses. The cases initiated by exposure this year will not be diagnosed until 1991 or 1995. Vinyl chloride is also expected to produce an equal number of primary cancers at other sites, for a total of somewhere between less than one and twenty cases of cancer per year of exposure among residents around plants. The number of these effects is expected to be reduced in proportion to the reduction in the ambient annual average vinyl chloride concentration, which is expected

to be 5% of current levels after the proposed EPA regulations are implemented.

As part of this analysis a survey of the known cases of liver angiosarcoma diagnosed in the last 10 years was initiated in order to see whether any evidence could be found that living near VCM or PVC plants results in higher rates than expected for the general U. S. population. No conclusion can be drawn from the survey at its present stage of completion. It is expected that if the true rate is as high as 10 cases per year of exposure among residents near plants, which is the highest rate our analysis would predict, higher than average rates of case occurrence might not yet be observable in such a survey but should become detectable in the next 5-10 years, due to the increase in vinyl chloride production rates since 1955-1960.

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Quantitative Risk Assessment for Community Exposure to Vinyl Chloride

Vinyl chloride is a known human carcinogen; it has produced liver angiosarcoma, a very rare form of cancer, as well as other cancers and non-cancer effects in occupationally exposed populations. It is also known to be emitted into the atmosphere from plants which produce vinyl chloride monomer (VCM plants) and plants with polymerize the monomer to polyvinyl chloride (PVC plants). Although concentrations of vinyl chloride in the ambient air are much less than those which caused cancer in workers, it is generally considered prudent to assume that there is no threshold for chemical carcinogens, so that any exposure involves some risk. In conjunction with EPA consideration of rulemaking action to regulate emissions of vinyl chloride from VCM and PVC plants, the Administrator of EPA requested that an analysis be performed which would estimate quantitatively the risk resulting from VC emissions and assess the reliability of the estimates. This paper reports the results of that analysis. The details are presented in the Appendices A through E.

The analysis involves three steps which are discussed in turn below: an estimate of the size of the exposed population, an estimate of the concentrations of vinyl chloride to which they are exposed, and an estimate of the number of liver angiosarcomas and other health effects which would result from this exposure. Of these, the last estimate is by far the most difficult to make. In addition an investigation was made of the places of residence of all people known to die of liver angiosarcoma in the last ten years in an attempt to detect clustering around PVC and VC plants.

Size of exposed population

A study by the American Public Health Association (APHA), performed under contract to EPA's Office of Toxic Substances, determined the number of people living within various distances, up to 5 miles, from each VCM or PVC plant. The study was based primarily on census tract information. The validity of the methodology used was confirmed by a more detailed analysis of the population living around a few plants, performed by EPA's Office of Planning and Evaluation.

The total population living within 5 miles of all PVC and VCM plants is shown in the following table:

Distance (mi)	Population
0 - 1/2	47,000
1/2 - 1	203,000
1 - 3	1,491,000
3 - 5	2.838,000
TOTAL	4,579,000

Thus, a total of 4.6 million people live in the vicinity of these plants. To calculate the average exposure, detailed population data around each individual plant was weighted to reflect differences in the type of plant (PVC or VCM), size of plant (average or large) and meteorological conditions at the plant site.

The use of residence data involves some error, of course, since people spend part of their time away from their homes and hence exposed to varying levels of vinyl chloride. There does not seem to be any practical way around this problem, short of a detailed study of travel patterns of 4 million people in over 40 separate communities.

Ambient vinyl chloride concentrations

Annual average ambient concentrations of vinyl chloride were calculated by standard diffusion modeling techniques. Two independent studies were made, one by EPA's Office of Air Quality Planning and Standards (OAQPS) and one by Teknekron, Inc. The agreement among the two studies was good, with differences generally less than 25%. It was decided to use the Teknekron results in the actual calculations since they included data on variations in meteorological conditions from location to location.

For an average uncontrolled PVC or VCM plantin an area with average meteorological conditions, the annual average concentration of vinyl chloride in each annulus around the plant is shown in the following table:

Distance (mi)	Vinyl chloride concentration (ppb)	
	PVC plant	VCM plant
0 - 1/2	323	113
1/2 - 1	57	20
1 - 3	15	5.2
3 - 5	5.7	2.0

It can be seen that concentrations around VC plants are significantly smaller than around PVC plants. This fact combined with the much smaller population living near VC plants, implies that by far the greatest part of the public health risk is from emissions from PVC plants.

In calculating total population exposure, it is necessary to take account of factors that lead to variations from the average. Information from OAQPS and from the APHA study was used to determine areas where more than one plant was located, and OAQPS characterized the size of each plant as "average" or "large." The Teknekron study was used to categorize the meteorological and topographic conditions at each location. Details are given in Appendix A.

The net result of these calculations is that the average exposure faced by a person chosen at random from the 4.6 million people living within 5 miles of plants is 17 ppb.

Unfortunately, it has not been possible to make a systematic comparison of the diffusion modeling results with data obtained from actual monitoring, although they appear generally consistent. It is therefore difficult to estimate the uncertainty of these estimates. Lacking anything better, we can take the difference between the two diffusion modeling efforts of up to 25% as an estimate of that uncertainty.

Health effects resulting from exposure

What are the results of exposing 4.6 million people to an average of 17 ppb of vinyl chloride? The first major decision to face in answering this question is to arrive at some combination of two basic approaches. One approach is to rely largely on human data (which exists for vinyl chloride but not for many other chemicals of concern to EPA): the secons is to make projections from animal experiments. Both involve difficulties. Use of human data eliminates the uncertainties that result because we do not know the differences in response between the test animals and humans. On the other hand, with the data on human (occupational) exposure, it is necessary to guess at exposure levels over the past 30 years and approximate the total number of workers involved and the number of cancers caused by past exposures for which symptoms may not appear until many years in the future. By using animal data we can avoid these problems, but only at the price of uncertainty in the relevance of animal experiments to human exposures. The approach taken in this analysis is to use animal data to predict the probability of human liver angiosarcoma, and then use the human data to the greatest extent possible to interpret those predictions.

A second major decision that must be made is how to project the results observed at high doses in animal experiments (and in the occupational exposures) to the much lower doses encountered in the environment. Two alternative assumptions are frequently made in the scientific literature. The first is a straight-line projection to zero dose, assuming no threshold (the "linear model"). This is also referred to as the "one hit" model, since it would follow logically from the assumption that each minute increment of exposure to a carcinogen has the same independent probability of causing a cancer, regardless of the dose level. This assumption is generally accepted as prudent in radiation carcinogenesis. For chemical carcinogenesis, the model is usually considered to provide an upper limit to the level of effects likely at extremely low doses, because the existence of detoxification mechanisms would render small doses less effective in causing cancer and would therefore result in a threshold of at least fewer effects.

The second commonly used projection method is based on the assumption that the observed changes in response with dose are the result

of variations of susceptibility in the population, which is assumed to be log-normally distributed with dose. For convenience, we refer to this as the "log-probit" model because it forms a straight line when the logarithm of the dose is plotted against the proportion of responses expressed in probability units (probits). The log-probit model is closely related to the Mantel-Bryan procedure.

In this analysis, both models are used. For technical reasons, the log-probit model is difficult to apply to this case. Therefore, the basic calculations were done using the linear model, but a sensitivity analysis was done to show how the results would change under the log-probit assumption.

A third decision that must be made is how to predict human incidence rates from animal data. Again, there is little hard data to provide guidance. The assumption used here is that a lifetime exposure of humans to a given concentration of vinyl chloride would produce the same number of effects as a lifetime exposure of rats. Thus, the one-year exposure in the animal experiments would be equivalent to about 30 years of exposure for humans.

A fourth decision to make is how to use the available human data on liver angiosarcoma cases among highly-exposed workers to calculate the probability per year of exposure that cases will eventually develop in people. This calculation is needed for comparisons with the animal model. There are three aspects to this problem: 1) to find in the literature a realistic estimate for the fraction of highly-exposed workers who have contracted liver angiosarcoma at some time in their lives; 2) to account for the fact that the currently-observed rates underestimate the actual incidence because they do not include workers who have been exposed more recently than 15 to 20 years ago; and 3) to account for the fact that people can die from other causes before a latent case of liver angiosarcoma becomes manifest.

These issues were treated as follows: Of the four occupational epidemiology studies from which it is possible to estimate an incidence rate, the two with the smallest number of subjects and the best separation of highly-exposed workers from the group of all workers had the largest incidence of angiosarcoma. This incidence was assumed to be valid for all highly-exposed workers. The latency time for liver angiosarcoma and the growth in the number of person-years of exposure since 1940 are two factors which affect the number of cases we have observed through 1974. These factors, along with the distribution of exposure durations for people who have been diagnosed with liver angiosarcoma, were included in an analysis presented in Appendix D. The result of the analysis is an estimate of the probability per year of exposure that a person will get angiosarcoma sometime in his life. The remaining problem of multiple risks competing for mortality was not treated because of its complexity.

A fifth decision which had to be made was how to quantatively describe the other effects of vinyl chloride exposure besides liver angiosarcoma. This problem was handled by estimating from the literature ratios of the number of people with other cancers and the number of people with liver damage compared to the number with angiosarcoma. As an index of liver damage, the bromsulphalein (BSP) test is used because it, among all liver function tests that have been used, correlates best with vinyl chloride exposure and because an abnormal BSP test indicates that severe damage has occurred in the liver, either because the liver cells are not able to assimilate the intravenously injected BSP dye from the blood and excrete it into the bile passages, or that the bile passages are no longer structurally intact enough to carry the dye out of the liver.

The results of these five aspects of the problem are presented below in reverse order. The approximate ratio of severe liver damage cases to liver angiosarcoma cases is about 30, the result being consistent for two independent occupational studies. It was also found that about twice as many cases of cancer of all sites are caused by vinyl chloride as cases of liver angiosarcoma alone. The animal experiments have shown approximately the same ratio of all cancers to liver angiosarcoma, after background incidence is taken into account.

In calculating the probability per year of exposure that a highly-exposed worker will get angiosarcoma some time in his life, we found that the fraction of highly-exposed workers who have been currently diagnosed is 0.02; they have been exposed for an average of 17 years before diagnoses. The analysis, which required assumptions about the time distribution of man-years of exposure and the distribution of exposure durations preceding diagnosis, showed that only 38% of the highly exposed workers who are expected to get angiosarcoma some time in their lives have been already diagnosed. Therefore the probability that one of these people will get angiosarcoma some time in their lives is $0.02/(17 \times 0.38) = 0.0031$ per year of exposure. In Appendix D the calculation is explained in greater detail.

The 17-year average concentration to which these workers were exposed was estimated to be 350 ppm on the basis of one study. Only one company has reported measurements of vinyl chloride for the jobs in their plant. These measurements, started in 1950, showed the highest exposure jobs ranged from 120 to 385 ppm before 1960, when the exposures were reduced because of suspected toxicological problems with vinyl chloride. In estimating the average, it was assumed that the other factories, most of which probably did not monitor the concentration of vinyl chloride, were less concerned about industrial hygeine and therefore took fewer pains to keep the levels low.

In predicting the human angiosarcoma rate from the animal dose-response data, it was projected, from the linear model, that exposure to vinyl chloride would cause 0.071 cases of liver angiosarcoma and

0.15 cases of all types of cancer per million people per year per ppb of continuous exposure. Details of these calculations are given in Appendix C. Converting to a 7-hour per day, 5 days per week work schedule of exposure to 350 ppm, the model predicts an incidence rate of 0.0052 per person-year exposure. It is shown in Appendix D that this rate is numerically indistinguishable from the rate of 0.0031 calculated from the human data, considering the known quantifiable errors of estimating the parameters of the animal and human data. It can be concluded that the slope of the linear animal dose-response relationship for angiosarcomas is consistent with the human data.

The extrapolation of the animal dose-response relationship to a concentration of 17 ppb (the average concentration around the uncontrollec plants) yields the following predicted number of cases in the 4.6 million people living within 5 miles of the plants. For details see Appendix B.

	Cases per year of exposure	
Type of effect	Linear model	Log-probit model
All cancer	11	0.1 - 1.0
Liver angiosarcoma	5.5 -	0.05 - 0.5

This is the expected number of cases projected to be caused per year at current levels of emissions; the people exposed now will not be diagnosed for another 15-20 years. Similarly, any cases observed now would have been caused 15-20 years ago (if they were in fact caused by vinyl chloride) when production was about 10% of current levels.

In order to arrive at a final estimate of the number of people adversely affected by vinyl chloride emissions, the important results of this analysis to consider are as follows: 1) the number of cancers at all sites caused by vinyl chloride is twice the number of liver angiosarcomas; 2) the number of people with severe liver damage is 30 times the number of liver angiosarcomas; 3) the animal model predicts that the number of liver angiosarcomas in the population around plants is 5.5 cases per year of exposure; 4) the number of cases calculated from the human data is 60% of the number predicted from the animal model; 5) the use of a log-probit model for extrapolation to low doses gives predictions of 0.1 to 0.01 times the number predicted by the linear model; 6) the error in the estimate of 5.5 cases per year ranges from + 55% to -10%. This error includes statistical uncertainty in estimating the dose-response, uncertainty in ambient concentration estimates, and errors resulting from not considering exposures beyond 5 miles or decomposition of vinyl chloride in the atmosphere. It is not symmetrical because it includes possible effects beyond 5 miles from the plants, which were not explicitly considered in the analysis. It does not account for our uncertainty about the appropriateness of using a

linear model extrapolated to zero dose; or of extrapolation from animal data; 7) the quantifiable error in the rate calculated from the human data is about ± 67%. This includes uncertainties in the 17-year average dose received by the workers, uncertainty in the number of hours per day of actual high exposure, and uncertainty in the fraction of highly-exposed workers who have been diagnosed with liver angiosarcoma. Other errors can not be quantified, and are discussed in Appendix D.

When all these uncertainties are considered, our judgement is that the number of liver angiosarcoma cases produced per year of exposure in people residing near vinyl chloride plants is somewhere between less than one and 10 cases. The cases produced by this year's exposure will not be diagnosed until 15 to 20 years from now. If the EPA regulations are implemented, the number of cases is expected to be reduced in proportion to the reduction in the ambient annual average concentration, which is expected to be 5% of the present level.

The vinyl chloride exposure around plants is also producing somewhere between less than 1 and 10 cases of primary cancer at other sites, mainly lung, brain and bone. Assuming no threshold for liver damage somewhere between less than 1 and 300 cases of serious liver damage would be predicted. The number of liver damage cases is likely to be less than this because the assumption of no threshold is likely to be wrong.

In order to find out whether people living near VC-PVC plants have, as of 1974, had higher rates of liver angiosarcoma diagnosis than the overall U. S. population, a search of the residence records of all known liver angiosarcoma cases in the last 10 years was performed. Out of 176 cases where residence at time of death was known, 3 people lived within 5 miles of a plant. Unfortunately, the diagnosis of these cases has not yet been confirmed by the National Cancer Institute. In addition one infant whose parents lived within one mile of a plant died of a relatively common liver tumor. It was shown in Appendix E that this rate of occurrence is not higher than the national average. However, the survey is too incomplete to draw any conclusions at the current time.

Considering the results of the foregoing analysis, one would only now expect to be seeing some evidence of vinyl chloride exposure. If the highest rate in our range were actually occurring, 10 cases of liver angiosarcoma per year of exposure are being produced; 15-20 years ago when the vinyl chloride production was 10% of current levels, one case would be expected per year of exposure (with constant population). This is to be compared to a background rate of 0.6 cases per year that are occurring now.

The survey of liver angiosarcoma cases would probably detect the existence of 10 cases over the 10 year period. Since this was not observed we can conclude that the real incidence is not significantly greater than the predicted upper limit of 10 cases initiated per year

of exposure unless migration of people in and out of the regions around plants has been excessive. If the lower rates in the range of the above analysis were to be true, increased incidence of angiosarcoma would not be observable.

Appendix A. Population and Exposure Estimates

This appendix describes the approach taken in estimating the population at risk and the levels of exposure. The task is made easier by the use of a linear dose-response model to project health effects at very low exposures. This model implies that, for example, a given number of people exposed to emissions from two plants, each at the same distance from the people, would suffer the same number of health effects as twice as many people at the same distance from one plant.

More formally, with a linear dose-response relationship, the number of health effects is proportional to the sum, over all plants, distances, etc., of population times dose. This discussion shows how this quantity was estimated.

Population Estimates

The population estimates used in this analysis are all based on a study performed by the American Public Health Association under contract to the Office of Toxic Substances (APHA, Population Residing Near Plants Producing Vinyl Chloride, Aug. 1975, hereinafter cited as "APHA Study").

The APHA Study is based on the 1970 Census. For each PVC or VCM plant (or group of plants located close together), APHA determined the number of people residing within 0-1/2 mi, 1/2-1 mi, 1-3 mi and 3-5 mi. Further breakdowns by age and sex and by direction from the plant were also determined; however, as is discussed below, these detailed breakdowns were not used in this analysis.

For those plants located in Standard Metropolitan Statistical Areas (SMSAs), where Census Tract data is available, APHA asked a local respondent to allocate each tract to one of the distance ranges. Census tracts were not split. Independently, the Office of Planning and Evaluation, EPA, made a similar analysis for several selected plants in which Census tracts were split and one for which city block data was used. A comparison with the APHA data for those plants indicates that no significant errors were introduced by the APHA procedure. For untracted areas, population was assumed to be uniformly distributed over the area. For a more detailed description of the methodology used to estimate population, the reader is referred to the APHA report.

The total population residing at the indicated distances from PVC or VCM plants is shown in the following table:

Distance (mi)	Population
0-1/2	47,000
1/2-1	203,000
1-3	1,491,000
3-5	2,838,000
Total	4,579,000

Of course, some of these individuals are exposed to VC from more than one plant or are exposed to emissions from plants that are larger than average. These variations are taken into account in calculating exposure estimates, as described below.

There are several sources of error which result from the methodology used here. First, by estimating the exposed population from Census statistics on place of residence, we do not take into account the daily mobility of people, some of whom travel into areas of higher VC concentrations than their residences while others do the opposite. There does not seem to be any way of estimating the net effect of such travel or even whether it increases or decreases overall exposure, short of an analysis of commuting and travel patterns in the 42 communities included in this study. In general, it might be noted, however, these plants do not appear to be located in large central business districts to which large number of non-factory workers commute. Given the much greater uncertainties that are inherent in the estimation of dose-response relationships, it does not appear to be worth the effort to do such anayises.

Second, the overall U.S. population has grown about 5% since the 1970 Census, but it is not known whether the areas covered in this study have grown similarly. The error here again seems small compared with other uncertainties.

Third, people living further than 5 mi from plants are not included. The effects of this error are considered below in the context of the overall calculation.

Fourth, even though information was available on the distribution of exposed populations by age, sex, and direction from the plant, this data was not used. The data on age and sex was not used because there is no information on how susceptibility to the effects of exposure to VC would vary with these factors. The data on distributionn of population by direction from the plants could have been used, together with data on the distribution of the ambient VC concentration by direction, to develop a more accurate estimate of the overall population exposure to VC. An examination of several communities with large exposed populations suggested, however, that the populations were either distributed in many directions or did not appear to be correlated with the angular distribution of VC concentration. Since the more detailed analysis would require 16 times as much calculation, it was judged not be worth the effort, particularly given the much greater uncertainties in other parts of the analysis.

Ambient Concentration Estimates

It was decided early that the measure of exposure to be used is the long-term (annual) average ambient concentration. At one level, this assumption is a consequence of the linear (single shot) dose-response relationship. But regardless of the specific form ot the dose-response relationship, it appears reasonable to assume that the probability that

any given individual suffers an adverse health effect would depend on the long term integrated exposure of the individual, independently of the functional relationship between the probability and the exposure level. Indpendent of the actual truth of this assumption, however, is the absence of any data on responses to peak exposures, either from the occupational experience or animal experiments. Thus, there is no alternative to this assumption if a quantitative projection of effects is to be made.

The estimates of average annual concentrations were derived from diffusion modeling, using standard techniques. Two independent modeling efforts were made. The first was performed by the Monitoring and Data Analysis Division of EPA's Office of Air Quality Planning and Standards in Research Triangle Park, N. C. This analysis took one representative set of meteorological conditions and devoted considerable effort to investigating the effects of different plant source configurations for controlled and uncontrolled PVC and VCM plants. In general, for average annual concentrations, the results for uncontrolled PVC plants were quite similar for the plant configurations analyzed, as were those for uncontrolled VCM plants (although, of course, emissions from PVC plants are very different than those from VCM plants). The results for typical PVC and VCM plants are shown in Figs. A-1 through A-4.

The second independent effort was performed by Teknekron, Inc. under the contract with EPA's Office of Planning and Evaluation. In this analysis, plant configurations were fixed (except for size and the difference in pattern of emissions between PVC and VCM plants), but an effort was made to estimate the distribution of meteorological conditions and, to some extent, topography for each site.

The results of the two sets of calculations were extremely close. The following table shows the average annual concentration of VC emitted from a typical PVC plant as a function of distance (that is, averaged radially through all directions), as determined by the two studies.

Distance (mi)	Vinyl Chloride Concentration (ppb) OAQPS	Teknekron
0.25	301	323
0.75	76	57
1.00	--	37
1.50	26	--
2.00	--	15
3.00	10	8
4.00	--	5.7
4.50	5.6	--
5.00	--	4.0

A dash (--) in the table indicates that a value was not determined for precisely that range. The results appear generally to be within 25% of each other, with the OAQPS results lower than the Teknekron results at very close ranges and higher at longer ranges.

In general, the closeness of the two sets of independent results serves as a confirmation of both. Of course, since both use similar methodologies, it is possible that they share some of the same errors. It has not proved possible to compare these modeling results with actual monitoring data in a systematic way, and this is an important shortcoming of the analysis as it stands at present. Summaries of the ambient measurements have been examined, however, and they appear to be generally consistent with the modeling results.

For the actual calculations, it was decided to use the Teknekron results, since these showed how ambient concentrations would vary with meteorological conditions (although, in retrospect, it turned out that these variations made little difference in the final result). The sites listed in the Teknekron report divided naturally into four categories, which we designate as Low (L), Average (A), High (H), and Very High (VH), based on the ambient concentration projected to result from the same level of emissions. (Variation among sites due to the number and size of the plants are factored into the analysis separately and are discussed below). Table A-1 shows the category to which each location is assigned. The relative ambient concentrations for the four categories are:

Low	0.56
Average	1.00
High	1.55
Very High	2.55

CAQPS provided information which categorized each plant by size as "Average" or "Large." The typical "Average" VCM plant had a production of 700 million lb/yr, and the typical "Large" VCM plant 1300 million lb/yr. For PVC plants, the typical "Average" plant used 150 million lb/yr and the typical "Large" plant 350 million lb/yr. It was assumed that large plants would have proportionately greater emissions. It is estimated that uncontrolled PVC plants emit about 4% of the vinyl chloride processed and VCM plants, about 0.3%. Table A-1 also shows, for each location, the number and size categories of plants located there. The letter "L" indicates a "Large" plant; thus, for example, the designation "2" means two "Average" plants and "2 & 2L" means two "Average" and two "Large" plants.

Overall Exposure Estimates

Table A-1 also shows the populations residing at the indicated distances from individual plants. To determine the equivalent exposure (compared to exposure from a single average size plant at a location with average meteorological conditions), each of the population figures must be multiplied by two factors, one reflecting meteorological conditions at that location and one reflecting the number and size of plants at the location. Thus, for example, in Texas City, Texas, each individual is exposed to emissions from two average size plants and one large plant (equivalent to 2.3 average plants) but under "Low" meteorological conditions (leading to 0.56 of the concentration that would be expected under average conditions). Thus, these individuals are exposed to the equivalent of 0.56 x 4.3 = 2.4 average plants' emissions under average

conditions. Similar calculations were performed for each location in Table A-1 and the results summed to obtain the values shown as "Weighted total population" in Table A-2.

The weighted total population figures are then multiplied by the average annual concentration of vinyl chloride, as derived from the diffusion modeling. The sum of these figures is the total population exposure. These results are shown in Table A-2. The total exposure is estimated to be 76.4 million people x ppb. Thus, the total exposure of the U.S. population to vinyl chloride due to emissions from these plants is equivalent to 76.4 million people exposed to 1 ppb, or 7.64 million people exposed to 10 ppb, etc. Probably the simplest way to understand this figure is in terms of the average exposure of those who are exposed. Since there are a total of 4.6 million people exposed within 5 mi of plants, the average exposure of these people is 76.4/4.6 or 17 ppb.

We can also use these figures to gain some insight into the distribution of the aggregate exposure (and hence of the resulting adverse health effects). We can see from Table A-2 that only about 3% of the exposure is a result of emissions from VCM plans, with the remaining 97% coming from PVC plants. It also appears that the bulk of the health effects will be occurring from 1 to 5 miles from the plants, rather than within one mile, since much larger numbers of people live at the greater distances.

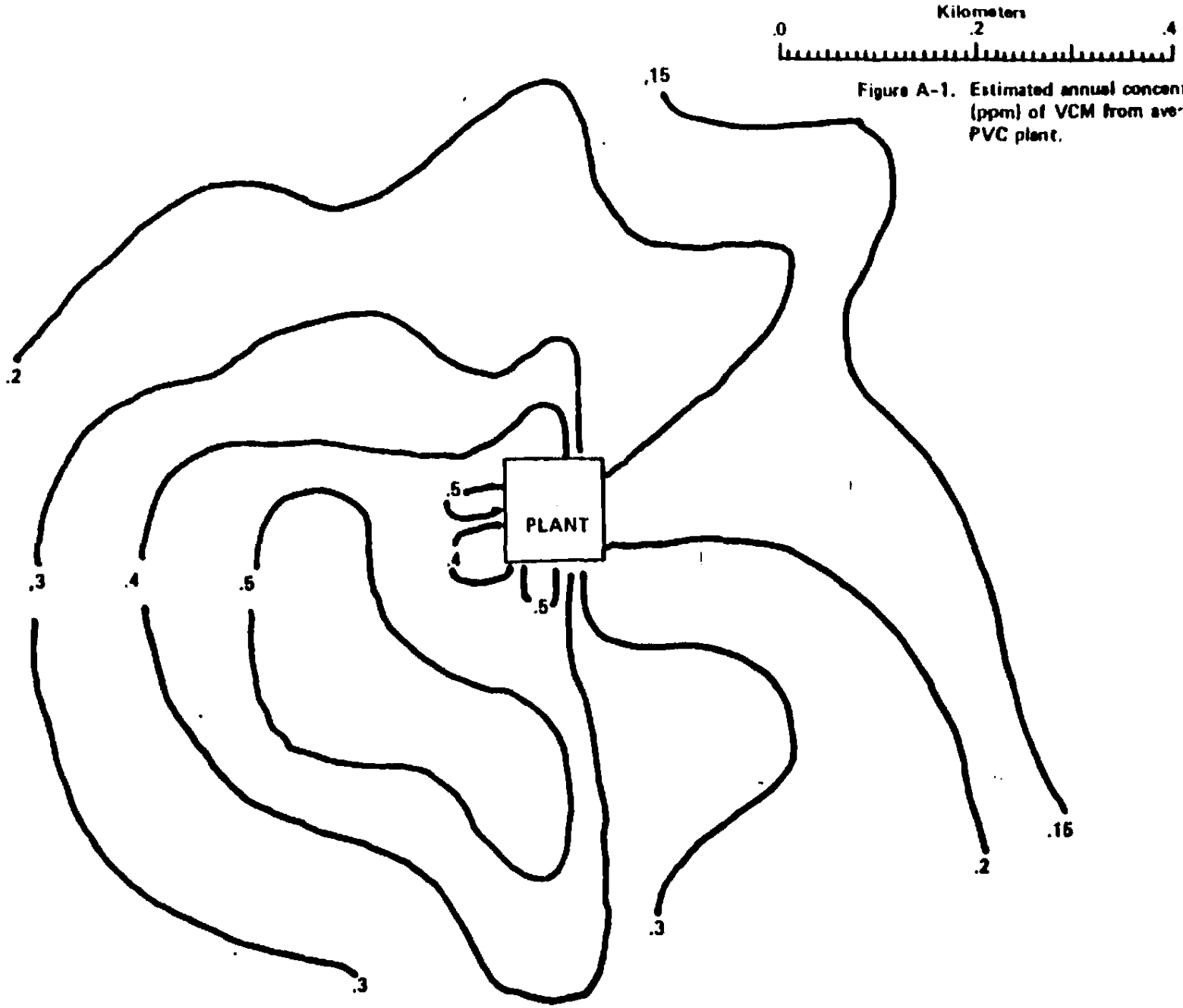


Figure A-1. Estimated annual concentrations (ppm) of VCM from average PVC plant.

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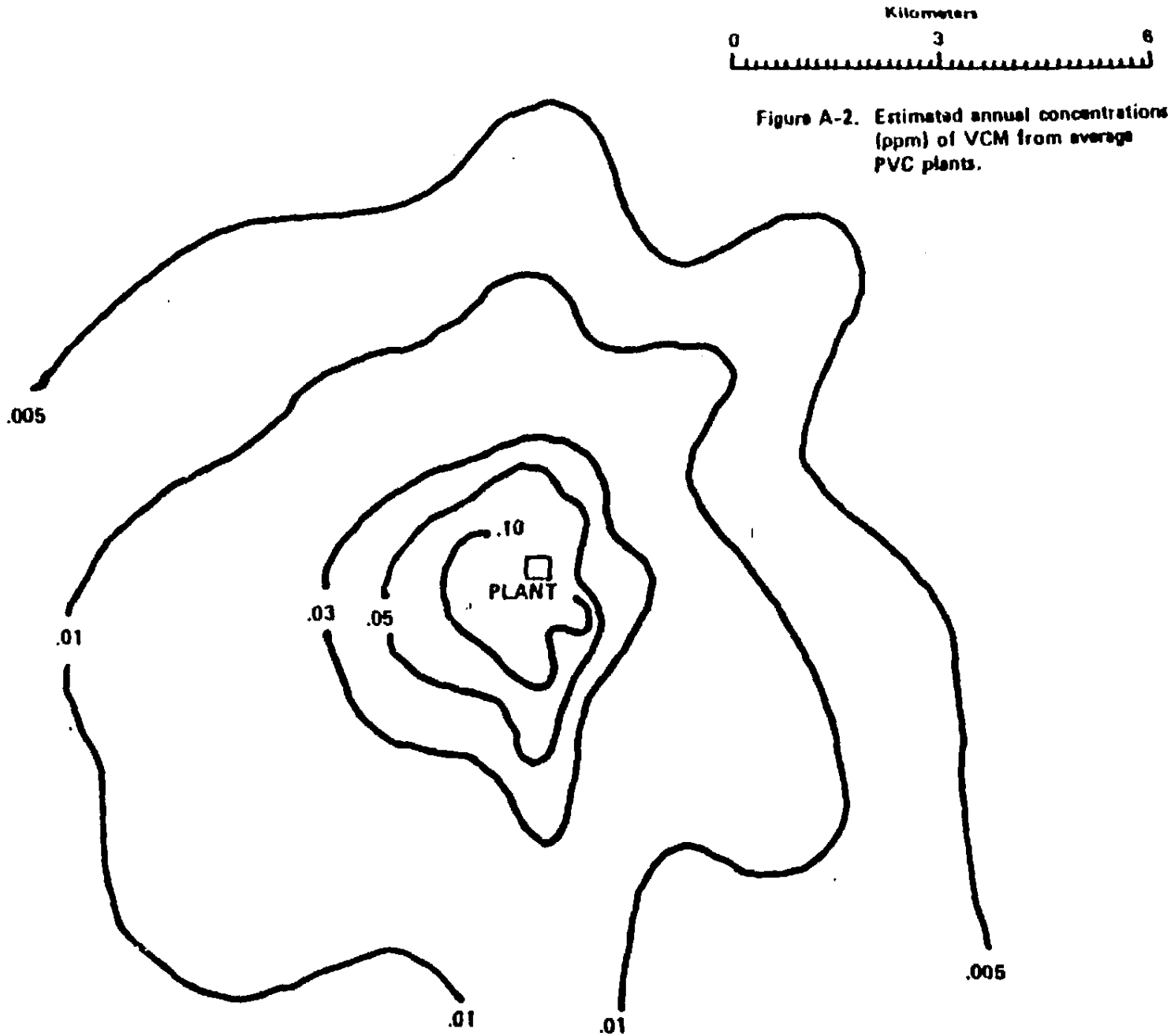


Figure A-2. Estimated annual concentrations (ppm) of VCM from average PVC plants.

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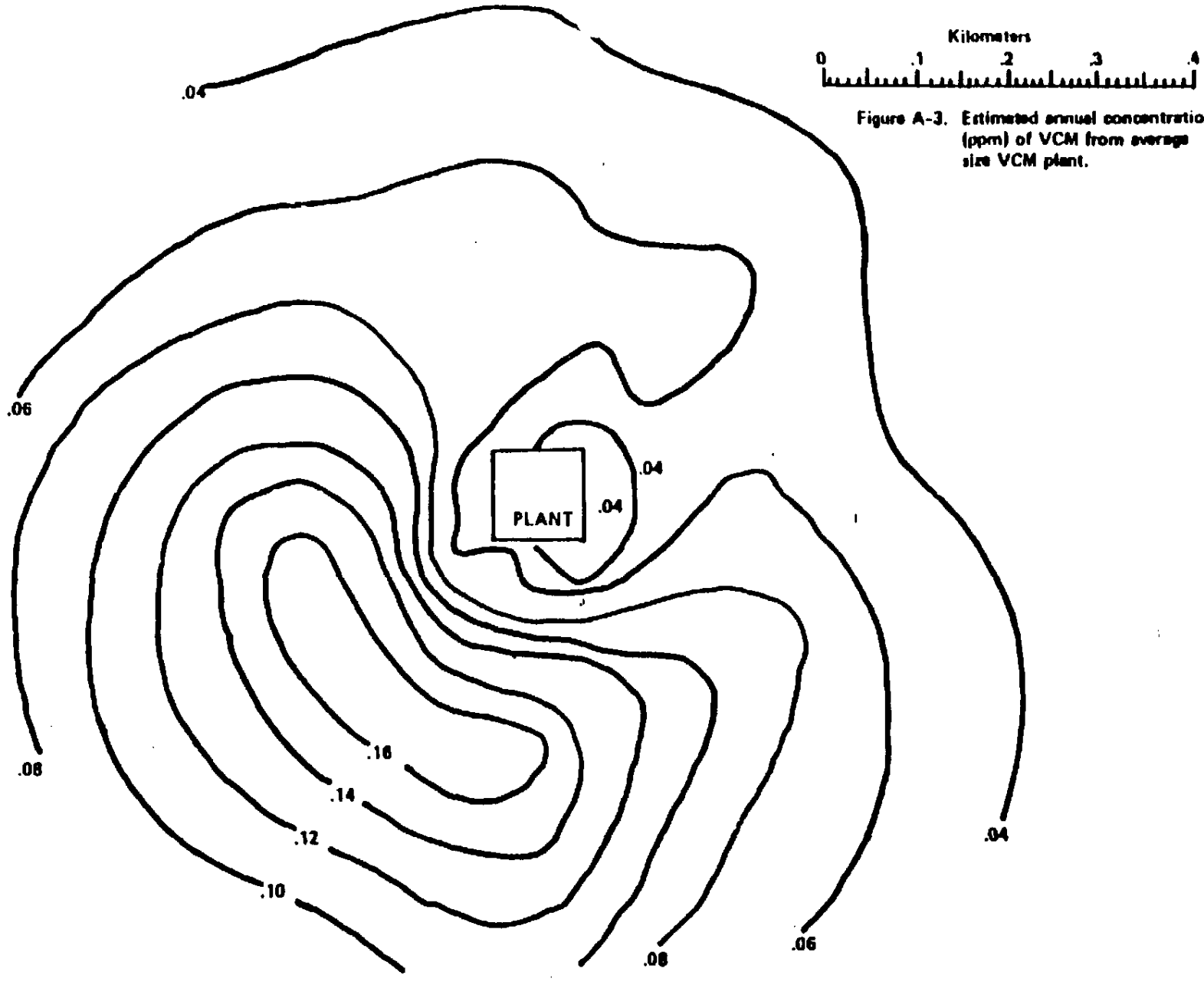


Figure A-3. Estimated annual concentrations (ppm) of VCM from average size VCM plant.

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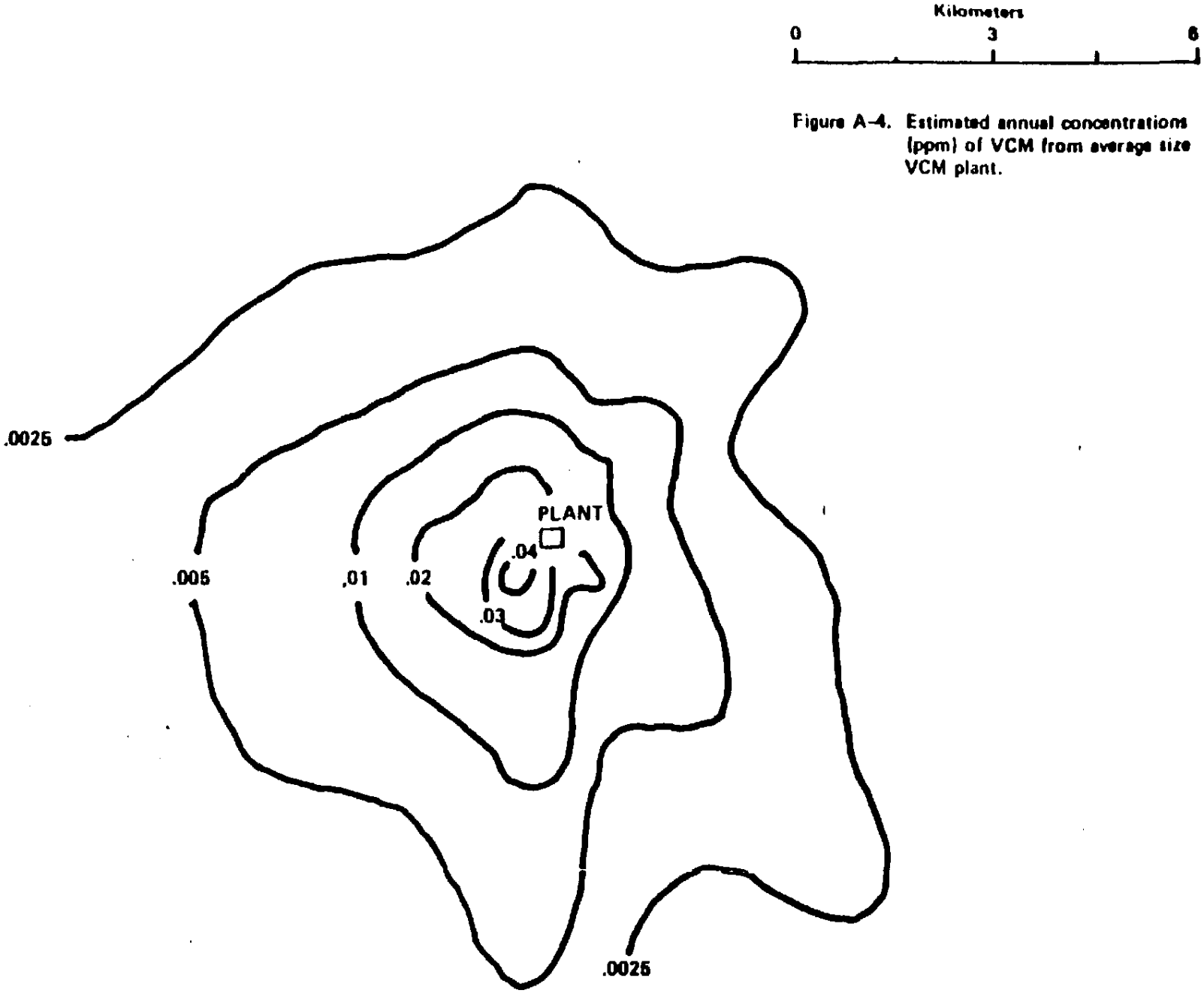


Figure A-4. Estimated annual concentrations (ppm) of VCM from average size VCM plant.

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TABLE A-1 Population Exposed by Location

Location	No. & Size of Plants	Meteorological Conditions	Population vs. Distance (mi) 0-1/2	1/2-1	1-3	3-5
a) PVC Plants						
Carson City, CA	2	A	--	16,541	259,309	405,461
Saugus, CA	1	H	--	10,935	--	12,029
Delaware City, DE	1&1L	A	--	--	6,970	9,218
Pace, FL	1	A	--	--	--	7,071
Henry, IL	1L	A	37	146	1,161	2,013
Illiopolis, IL	1	L	30	117	922	1,597
Calvert City, KY	1	A	102	398	3,134	5,436
Louisville, KY	1L	A		6,315	111,146	146,000
Plaquemine, LA	1	A	177	673	5,328	9,237
Baton Rouge, LA	1	A	--	--	25,615	54,193
Perryville, MD	1L	H	--	2,091	11,091	9,791
Fitchburg, MA	3	A		34,395	67,767	114,489
Springfield, MA	1	A		8,258	40,671	108,966
Midland, MI	1	L	492	1,899	15,045	26,067
Aberdeen, MS	1L	VH	114	442	3,504	6,073
Burlington, NJ	2	L		8,159	122,419	230,361
Pedrickton, NJ	1	A	--	--	9,113	98,992
Passaic, NJ	1	L	22,512	29,458	194,601	269,259
S. Kearny, NJ	1	L		18,656	214,594	696,928
Flemington, NJ	1	A	81	306	2,394	4,153
[illegible]sville, NY	1	L	5,900	20,702	143,760	215,230
Williamsville, NY	1	L	11,522	12,552	51,872	113,422
Ashtabula, OH	1	A	434	1,671	13,239	22,937
Huron, OH	1	L	98	380	2,988	5,175
Avon Lake, OH	1L	L	827	5,511	16,398	8,022
Painesville, OH	1&1L	A	--	--	21,550	16,434
Oklahoma City, OK	1L	L	--	--	25,526	73,581
Pottstown, PA	1L	H	--	2,447	36,121	30,382
Deer Park, TX	1L	H	--	12,820	32,151	86,508
Freeport, TX	1&1L	L	--	--	4,746	15,879
Texas City, TX	2&1L	L	3,440	--	19,460	34,751
Pt. Pleasant, WV	1	H	263	1,007	7,984	13,836
S. Charleston, WV	1	VH		5,698	40,685	53,432
(b) VCM Plants:						
Geismar, LA	3	A	62	233	1,852	3,211
Baton Rouge, LA	1	A	--	--	25,615	54,193
Lake Charles, LA	2	A	--	--	11,601	44,100
Plaquemine, LA	1	A	177	673	5,328	9,237
Norco, LA	2	A	215	823	6,525	11,306
Deer Park, TX	2	H		12,820	32,151	86,508
Freeport, TX	1	L	--	--	4,746	15,879
Calvert City, KY	1L	A	102	398	3,134	5,436
Guayamilla, PR	1	A	202	780	6,173	10,692

FOOTNOTES:

a/ Numbers followed by "L" indicate "Large" plants as defined in the text. Other numbers indicate "Average" size plants.

b/ Ratio of ambient concentration to emissions is characterized as Low (L), Average (A), High (H), or Very High (VH).

c/ Adjusted to show effect of multiple exposures at varying distances from plants. Not to be multiplied by number of plants.

TABLE A-2 Aggregate VC Exposure

	Distance From Plant (mi)				
	0-1/2	1/2-1	1-3	3-5	Total
(a) PVC Plants					
Weighted total population	34,000	228,000	2,013,000	3,439,000	
VC concentration (ppb)	323	57	15	5.7	
Total exposure (million population x ppb)	11.0	13.0	30.2	19.6	73.8
(b) VCM Plants					
Weighted total population	1,200	37,000	169,000	434,000	
VC concentration (ppb)	113	20	5.2	2.0	
Total exposure (million population x ppb)	0.1	0.7	0.9	0.9	2.6
Total for all plants					76.4

Appendix B. Estimates of Effect Rates from Animal Data

In analyzing the effects of vinyl chloride, we are fortunate to have at least semi-quantitative data for both human and animal exposures. Choices had to be made concerning how to use this data. Vinyl chloride is known to cause both cancer and non-cancer effects in humans. With respect to cancer effects, we have animal experiments in which rats were exposed to known concentrations for known periods of time. We also have the human occupational experience, in which increased incidence of liver angiosarcomas and other cancers were observed in exposed workers. Ideally, we should use human data throughout and avoid the problem of extrapolating from an animal model to human beings. Unfortunately, we do not have good data on the level of exposure of workers. In arriving at a specific number to write down and use in the calculations, it would be necessary to guess at these factors. We decided, therefore, to derive a dose-response relationship from the animal data, for cancer effects, but to use the human data to the greatest extent possible to confirm or deny the validity of the relationship. These checks are described in Appendix D. Since both the human and the animal data involve exposure at high levels, the difficulty in extrapolation from high to low exposure levels would be the same in either case.

With respect to effects other than cancer, the animal data is too fragmentary to estimate quantitatively the ratio between cancer and non-cancer effects caused by exposure to vinyl chloride. However, the human data does permit an estimate of this ratio, and this estimate can then be used together with the dose-response estimate for cancer to place some limits on the magnitude of the non-cancer effects.

The Animal Data on Cancer

The basic experiment is Maltoni's experiment BT1. Sprague-Dawley rats were exposed by inhalation to a specified concentration of vinyl chloride for 4 hr/day, 5 days/week for 52 weeks. After the 52 weeks, the animals were not exposed to vinyl chloride but were observed for the remainder of their lifetimes. The results of the experiment were as follows:

VC Concentration (ppm)	Liver Angiosarcomas	All Tumors
10,000	9/61 (15%)	38/61 (62%)
6,000	13/60 (22%)	31/60 (52%)
2,500	13/59 (22%)	32/59 (54%)
500	7/59 (12%)	22/59 (37%)
250	4/59 (7%)	16/59 (27%)
50	1/59 (2%)	10/59 (17%)
0	0/58 (0%)	6/58 (10%)

Two conclusions can be drawn immediately from these results. First, the rate of occurrence of both liver angiosarcomas and total cancers increases much more slowly with dose above 500 ppm than below this concentration. The cause of this phenomenon is not understood, but, whatever it may be, it does not appear to be relevant to extrapolation to lower doses in the parts-per-billion range. Hence, all the curve-fitting reported here is done only for the data for 500 ppm and below.

Second, the control group had no liver angiosarcomas, but about 10% of them did have other types of cancers, as is to be expected. Therefore, in estimating the dose-response relationship for total cancers, it will be necessary to use a method which allows for a positive response at zero dose.

Extrapolation to Low Doses

It is widely understood in the scientific community that no firm logical basis can exist for extrapolating data to levels many orders of magnitude beyond the range of experimental observation. In spite of this, two alternative assumptions are frequently made in the literature. The first of these is that the response is linear in dose, e.g., half as many effects are produced at half the dose. This is also referred to as the "one-hit" model, since it would follow logically from the assumption that each minute increment of exposure to a carcinogen has the same independent probability of causing a cancer regardless of the dose level. This assumption is generally accepted as prudent in radiation carcinogenesis. For carcinogenesis in general, this model is usually considered to provide an upper limit to the level of effects likely at extremely low doses, because the possible existence of detoxification mechanisms might render small doses ineffective in contributing to cancer effects and thus result in a threshold below which no effects would occur. The second assumption frequently made is that the response is linear when the logarithm of the dose is plotted against the proportion of responses expressed in a probit scale (i.e., the value of the argument of the cumulative normalized Gaussian distribution function corresponding to that proportion); for convenience, we refer to this as the "log-probit" model. This model is derived from the assumption that individuals in the population have a statistical distriibution of susceptibility to cancer effects which is normally distributed with log dose.

The linear model is much easier to apply, since the number of effects depend only on the number of individuals exposed and their average dose. The log-probit model, on the other hand, requires data as to the distribution of doses in the population and is therefore more likely to be influenced by anomalies in the methods for estimating population exposure. For this reason and because of the conservativeness of the linear model, the calculations were made using the linear model, but a sensitivity analysis was performed to show how the results would vary were the log-probit model used.

Straight lines were fitted to Maltoni's data using a maximum-likelihood method. Essentially, this method assumes that the probabilities of cancer at the different doses were related by a linear function and determines the values of the slope and intercept that would make the likelihood of the observed results as high as possible. Mathematical details are provided in Appendix C.

For the data on liver angiosarcoma, the line was constrained to go through the origin. (Without this constraint, a slightly negative intercept was obtained.) With the constraint, the following relationship is obtained:

$$P = 2.53 \times 10^{-4} d$$

where P is the probability of a liver angiosarcoma and d is the dose in ppm. For all tumors, the corresponding line is

$$P = 0.123 + 5.26 \times 10^{-4} d$$

Here, the intercept is the best estimate from all data at 500 ppm and below of the cancer rate in the absence of vinyl chloride; the slope is the best estimate of the rate at which tumors are caused by exposure to vinyl chloride. Thus, inn the animals, vinyl chloride appears to cause about one other cancer for each liver angiosarcoma. Estimates of the total number of cancers caused by vinyl chloride are based on the slope of this line, not on the intercept.

The standard deviations of the estimates of the slope were found to be 6.9×10^{-5} for liver angiosarcoma and 1.1×10^{-4} for all tumors. (These estimates are derived from large-sample theory). It cannot be emphasized too much that the standard deviations reflect only that uncertainty in the estimates that is due to limited sample size; they are derived from the linear model and cannot reflect the much greater uncertainty due to our ignorance of whether the linear model is correct. They also do not reflect the effects of the part-time exposure used in the experiments or, most importantly, of the difference between human beings and Sprague-Dawley rats; these are discussed below.

The goodness of fit is shown in the following table, which compares the actual data obtained in the experiments with the values expected from the above equations.

	Liver Angiosarcoma		All Tumors	
Dose (ppm)	Actual	Expected	Actual	Expected
0	0	0.00	6	7.16
50	1	0.75	10	8.84
250	4	3.74	16	15.05
500	7	7.47	22	22.81

In order to test the sensitivity of these projections to the assumed linear form of the dose-response relationship, the data for liver angiosarcoma was also fitted to a log-probit equation. Using an unweighted

least-squares fit to the data for doses of 500 ppm or less, the following equation is obtained:

$$\text{probit} = -3.71 + 0.932 \log \text{dose}$$

where log indicates common (base 10) logarithm and dose is in ppm. The fit is extremely close: the maximum difference between predicted and actual values is 0.02 probits.

Both the linear and log-probit fitted curves are plotted in Fig. B-1 on a log-probit scale. It can be seen from this graph that, in the range of concentrations of interest (say, 5 to 300 ppb), the log-probit projection yields risks of one to three orders of magnitude lower than the linear projection. Since the result of a complete log-probit analysis of human effects due to exposure to vinyl chloride in the ambient air near plants would tend to be dominated by the high concentrations close to the plants (unlike the linear analysis), the log-probit analysis would yield final results one to two orders of magnitude lower than the linear model.

Extrapolation to Human Exposure

The equations derived above can be used to project what the response of rats would be in similar experiments at much lower doses. To apply these results to human community exposures, it is necessary to modify the results to reflect 24-hour exposure and, most importantly, the difference between humans and rats. The latter is particularly problematical. The remainder of this Appendix will be based on animal data. Human data will be discussed in Appendix D.

In the experiments, the animals were exposed to vinyl chloride for 4 hr/day, 5 days/week. In the community, the exposure would be continual for 24 hr/day, 7 days/week. This effect can be corrected for by dividing the continuous dose received by people by (7x24)/(5x4) = 3.4 and using the same linear dose-response function to calculate the risk. This is mathematically equivalent to multiplying the slope by the same amount.

Adjusting for the difference between humans and rats is much more difficult. As in the extrapolation to low doses, very little evidence is available. Furthermore, controlled experiments involving human carcinogenesis are unlikely to be carried out. In spite of the lack of evidence, the consensus of the scientific community appears to be that, if an assumption must be made, it should be assumed that the same number of cancers would be induced in humans over their lifetimes as are induced in rats over their lifetimes. With this assumption, the one-year exposure of the experimental is expected to cause the same probability of cancer (i.e., the same incidence rate) as about 30 years of exposure to humans at the same concentration. Thus, to determine the annual rates of cancer induction in humans,

the rates derived above should be divided by 30.

Making these adjustments and also converting the units to cases per million population per ppb per year, we obtain a rate of 0.071 liver angiosarcomas and 0.147 total cancers (in excess of background rates) per million population per ppb vinyl chloride per year. The standard deviations of the estimates, which, again, reflect only the statistical uncertainty due to limited sample sizes, are 0.019 and 0.031, respectively.

Incidence of Non-cancer Effects

Liver damage has been a frequent observation in animals exposed to vinyl chloride. The lowest dose in chronic experiments (times longer than 3 months) at which non-cancerous liver effects are observed is 100 ppm (Torkelson, et al., 1961). The effect observed was liver enlargement which cannot be called damage since the microscopic structure was normal, but it is an indication that the liver is affected in some way by vinyl chloride. Of all reports on animals the lowest dose causing liver cellular degeneration is 500 ppm (7 hrs/day, 5 days/week, for 4-5 months). A Russian report of bone resorption, cardiovascular impairment and neurological changes in the hypothalmus of rats and rabbits at concentrations of 12-16 ppm for 6 months (Basalaev, 1972) has also appeared. It is unfortunate that we currently have no published reports of pre-cancerous liver damage occurring in the same experimental group of animals in which cancer later develops. Therefore there is no way to estimate the incidence ratio of non-cancer to liver angiosarcoma effects in animals.

The human data on non-cancer effects is discussed in Appendix D.

Overall Effect Rates

In Appendix A, it was estimated that the 4.6 million people living within 5 mi of PVC or VCM plants would be exposed, on the average, to 17 ppb of vinyl chloride. Combining this information with the dose-response estimates derived above, we can calculate that, for example, vinyl chloride emisssions in the absence of control would cause 4.6 x 17 x 0.071 = 5.5 cases of liver angiosarcoma per year in the U.S., using the linear model. Similar calculations for all cancer yield the following results:

Type of Effect	Cases Caused per Year
Cancer	
Liver angiosarcoma	5 1/2
All cancer	11

It should be realized that cancers caused by vinyl chloride have about a 20-year latent period. Thus, we would not expect to observe these numbers of cancers occurring now; rather, these are the numbers

that are estimated to be initiated now but which will produce symptoms perhaps 20 years from now if emissions remain unchanged from current uncontrolled levels. Similarly, we would expect that cancers caused by vinyl chloride that are showing symptoms now would have been caused by emissions over the past 20 years, during which total vinyl chloride production increased from about 10% of its current leveel.

Error Analysis

The following discussion attempts to estimate the errors involved in this analysis other than the errors introduced by extrapolation from high to low doses and from animals to man, which are discussed in Appendix D. Quantitative estimates are provided below for the following sources of error:

(1) Errors in the slopes of the dose-response lines due to the limited sample sizes in the experiments.

(2) Errors in estimating the ambient concentrations around plants.

(3) Errors resulting from not considering effects on the population living further than 5 mi from plants.

(4) Errors resulting from not considering the decomposition of vinyl chloride in the atmosphere.

The standard deviations of the estimates for the slopes of the dose-response lines were calculated from maximum-likelihood large-sample theory, as described in Appendix C. As mentioned above, the results are 27% and 21% of the estimated slopes for liver angiosarcoma and all cancer, respectively.

Ideally, an estimate of the errors in the diffusion modeling could be derived from a comparison with actual monitoring data. Unfortunately, such a comparison is not available. In its absence, we use the difference between the two independent estimates as a measure of the degree of uncertainty in the estimates. As was noted in Appendix A, the two sets of results were generally within 25% of each other.

We can estimate the magnitude of the errors due to the 5-mile cut-off and vinyl chloride decomposition as follows. Suppose that, say, 10 million people live between 5 and 10 mi from the plants where they would be exposed to an average concentration of VC of about 2.4 ppb, based solely on the diffusion modeling. (The figure of 10 million people is chosen to show the sensitivity of the calculation to the 5-mile cut-off. The 5-10 mile annulus contains 3 times the area of the 5-mile circle, but would probably contain less than 3 times the population, since in many cases the bulk of the urban area would be within the 5 mi distance.) This exposure would cause 3.5 cases of cancer of all types per year and corresponding numbers of other health effects, according to the linear model. However, vinyl chloride is believed to decompose in the

atmosphere with a half-life estimated at about 6 hours. Assuming an overall average wind speed of 10 mi/hr, the average concentration in the 5 to 10 mile annulus would be reduced by decomposition by about 8%, so about 3.2 cases of cancer would be occurring there. At the same time, decomposition would reduce the average concentration within 5 miles as well, by about 3.4%. The overall effect of these compensating errors would be to increase the estimate by about 25%.

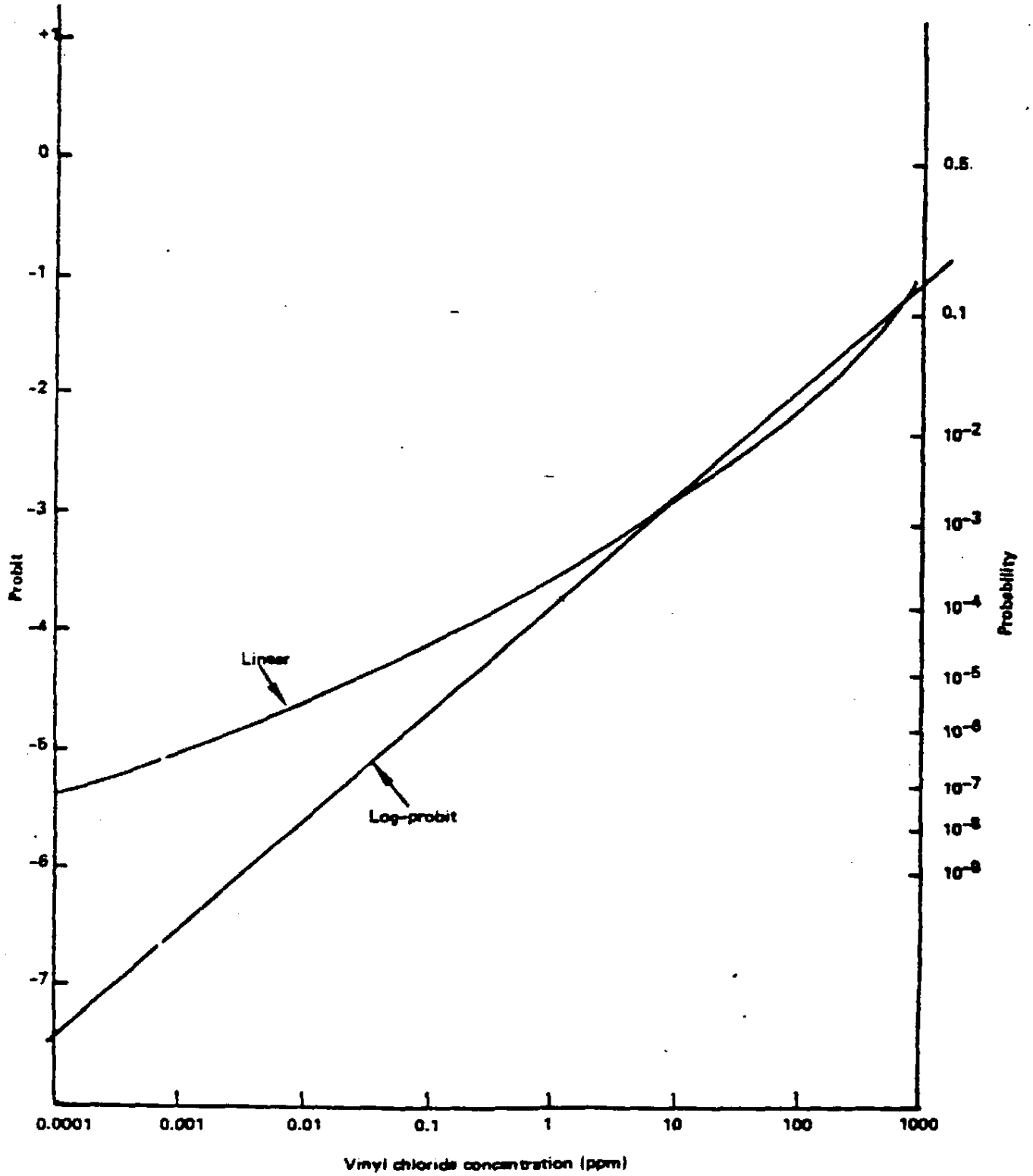
Assuming that the first two sources of error are independent, the estimates can be combined using a Pythagorean formula. Thus, the combined error estimate would be the square root of

$$(.21)^2 + (.25)^2$$

or about .3% of the estimated 11 cases of cancer per year predicted by the linear model. This is about 3.6 cases per year. Since the second two sources of error listed above are estimated to lead to an underestimate by about 2.8 cases per year, a total error estimate might be plus about 6 or minus about 1 cases out of 11 per year.

(Note under the log-probit model, the effect of the 5-mi cut-off would be negligible.)

Figure B-1. Comparison of linear and log-probit projections for liver angio sarcoma (Maltoni experiment BT 1)



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Appendix C. Mathematical Treatment of Dose-Response Data

The dose-response curves given in Appendix B were derived from the animal experiments using an extension of the standard maximum-likelihood (ML) method of statistical estimation. The results at different doses are combined and the line chosen is the one for which the likelihood of the observed results is the greatest. A discussion of the standard ML method may be found in any elementary text on mathematical statistics.

Equations for ML Estimators

Assume that M experiments (including the controls, if any) are performed. In the i-th experiment, n_i animals are exposed to a dose d_i and x_i responses are observed. We assume that the response of each animal is independent of the others, so each experiment is a set of Bernouilli trials in which the probability of response p_i is a function of d_i with unknown parameters. In particular, we consider two functional forms here:

$$p_i = \beta d_i \tag{1}$$

$$p_i = \alpha + \beta d_i \tag{2}$$

The method is readily adapted to other functional forms.

Eq. (1) will be called the "no zero response" case and is appropriate when there is no measurable response at zero dose. Eq. (2) will be called the "positive zero response" case and is appropriate when a measurable response occurs at zero dose. (The mathematics allows $\alpha < 0$, of course, which would imply at threshold below which no response would occur. Inferring the existence of a threshold is fraught with difficulty.)

For the positive-zero-response case, the joint probability of the results obtained is

$$L(\alpha, \beta) = \prod \binom{n_i}{x_i} (\alpha + \beta d_i)^{x_i} (1 - \alpha - \beta d_i)^{n_i - x_i} \tag{3}$$

(all sums and products are assumed to be over the range $i = 1, \ldots, M$ unless otherwise indicated). The values of α and β which maximize L (unless they fall on the boundary $\alpha + \beta d_i = 1$ for some i) satisfy the equations

$$\frac{\partial}{\partial \alpha} \log L = 0 \tag{4}$$

$$\frac{\partial}{\partial \beta} \log L = 0 \tag{5}$$

After simplification, Eqs. (4) and (5) reduce to

$$\sum \frac{x_i - (\alpha + \beta d_i) n_i}{(\alpha + \beta d_i)(1 - \alpha - \beta d_i)} = 0 \qquad (6)$$

$$\sum \frac{x_i d_i - (\alpha + \beta d_i) n_i d_i}{(\alpha + \beta d_i)(1 - \alpha - \beta d_i)} = 0 \qquad (7)$$

There are a set of two simultaneous 2M-th order algebraic equations for which no closed-form solution is possible. However, a numerical solution can be obtained by, for example, Newton's method for two variables which is described below.

For the no-zero-response case, Eq. (5) applies and is equivalent to Eq. (7) with $\alpha = 0$. This simplifies to

$$\sum \frac{x_i - n_i d_i \beta}{1 - \beta d_i} = 0 \qquad (8)$$

This is, again, a 2M-th order algebraic equation which can be solved numerically by the standard Newton's method, described below.

Asymptotic Variances

With the ML method, asymptotic variances of the ML estimators are readily obtained. (See S. S. Wilks, Mathematical Statistics (Wiley, 1962), pp. 379-81.) These are given by

$$\frac{1}{\sigma_\alpha^2} = E\left\{\left[\frac{\partial}{\partial \alpha} \log L\right]^2\right\} \qquad (9)$$

$$\frac{1}{\sigma_\beta^2} = E\left\{\left[\frac{\partial}{\partial \beta} \log L\right]^2\right\} \qquad (10)$$

for the positive-zero-response case. Eqs. (9) and (10) reduce to

$$\sum \frac{n_i}{(\alpha + \beta d_i)(1 - \alpha - \beta d_i)} \qquad (11)$$

$$\sum \frac{n_i d_i^2}{(\alpha + \beta d_i)(1 - \alpha - \beta d_i)} \qquad (12)$$

Again, for the no-zero-response case, the corresponding result is obtained by setting $\alpha = 0$ in Eq. (12), which gives

$$\sum_{i=1}^{k} \frac{n_i d_i^2}{p_i(1-p_i)} \tag{13}$$

Eqs. (12) and (13) were used in Appendix B to calculate the standard deviations of the slopes of the dose-response lines.

Newton's Method for One Variable

We wish to solve the equation $f(x) = 0$, where f has a continuous first derivative. Starting with an initial estimate x_0, we approximate the curve $y = f(x)$ by its tangent at $x = x_0$; the point where the tangent intersects the x - axis is an improved estimate of the root.

The equation of the tangent is

$$y - f(x_0) = f'(x_0)(x - x_0) \tag{14}$$

Setting $y = 0$ in Eq. (14) yields

$$x = x_0 - \frac{f(x_0)}{f'(x_0)} \tag{15}$$

Eq. (15) can be iterated until sufficient accuracy is obtained.

Newton's Method for Two Variables

Here, we wish to solve the equations

$$\begin{aligned} f(x,y) &= 0 \\ g(x,y) &= 0 \end{aligned} \tag{16}$$

and have an initial estimate x_0, y_0. The plane tangent to $f(x,y) - z = 0$ at $(x_0, y_0, f(x_0, y_0))$ is

$$(f_x, f_y, -1) \cdot (x - x_0, y - y_0, z - f) = 0 \tag{17}$$

or

$$(x - x_0)f_x + (y - y_0)f_y - (z - f) = 0 \tag{18}$$

where the subscripts indicate differentiation and all functions are evaluated at (x_0, y_0). Similarly, the plane tangent to $g(x,y) - z = 0$ at $(x_0, y_0, g(x_0, y_0))$ is

$$(x - x_0)g_x + (y - y_0)g_y - (z - f) = 0 \tag{19}$$

The planes given by Eqs. (18) and (19) intersect in a line, which in turn intersects z = 0 in a point which is an improved estimate of the root. This point can be obtained by solving Eqs (18) and (19) simultaneously with z = 0. This gives

$$x - x_0 = -\frac{f_0 - g f_y}{f_x g_y - f_y g_x} \tag{20}$$

$$y - y_0 = -\frac{f_x g - g_x f}{f_x g_y - f_y g_x} \tag{21}$$

Eqs. (20) and (21) can be iterated until sufficient accuracy is obtained.

APPENDIX D: USE OF HUMAN DATA TO ESTIMATE RISKS

In Appendix B the linear model describing the dose-response curve for the incidence of liver angiosarcoma in rats was modified by two factors in order to estimate the number of cases of human angiosarcoma in human populations. These two (multiplicative) factors were 8.4, to convert the 4 hrs/days, 5 days/week rat exposures to continuous exposures, and 1/30 = 0.033, to convert the annual rates in rats to in humans. In this appendix the available human data on liver angiosarcoma were examined to validate these factors, revised estimates of the number of human cases per year of exposure to ambient vinyl chloride concentrations around plants are made, and the number of people incurring effects other than liver angiosarcoma is estimated. The emphasis is placed on the quantitative comparison of data in rats and humans.

Interspecies Comparison of Life Expectancy and Angiosarcoma Latent Times

The most obvious difference between rats and people is their relative size and attendant metabolic rate and life expectancy. Both life expectancy and the time required for tumors to develop are independent manifestations of the basic differences in metabolic rates.

According to the 1970 U.S. census data, the life expectancy of one-year old infants in the U.S. is 75.6 years for females and 68.3 years for males. This is to be compared to about 100 weeks or 1.93 years for rats. The ratio of life expectancies is therefore about 1.9/72 = 0.026.

The time required for tumor development in people can be estimated from the case histories of 14 confirmed cases of liver angiosarcoma, all of whom are highly-exposed workers in the vinyl chloride industry (reference 2). The average time between first exposure to vinyl chloride and diagnosis is 19.6 years, and the average duration of exposure is 17.2 years. The distribution of exposure durations preceding diagnosis among these cases is approximately log normal, i.e. it is a normal distribution when plotted against the logarithm of latent time. The median time is 17 years, and the variance is 0.15 units of log (time) per one standard deviation. By contrast, Maltoni found that the average time in rats between onset of exposure and appearance of tumors was 80 weeks (after a 52-week exposure), regardless of dose between 10,000 ppm and 250 ppm. The one liver angiosarcoma which appeared in the 50 ppm group occurred 135 weeks after onset of exposure, but this one case is regarded as insufficient information to disprove the conclusion that the latent time in rats is independent of dose. The approximate ratio of times required for tumor development in humans and rats is 1.54/19.6 = 0.078.

This estimate contains considerable error. One error is that the

observed average exposure duration preceding diagnosis in the workers is an underestimate of the true biological latency. There are two reasons for this statement:

a. The number of people in the industry has increased steadily since 1940. Therefore the current work force contains an over representation of people with short exposure times, and the cases which have appeared are systematically biased toward short exposure times.

b. Even if the number of exposed people had not increased in the past, the disease could still develop in those people in the future, which would result in a longer average exposure duration before diagnosis.

A more fundamental difficulty in attempting a comparison of exposure times for rats and humans is the uncertainty whether the disease is caused by a gradual build-up of some type of tissue irritation which requires constant intake of vinyl chloride or whether it is caused by an event occurring randomly in the exposure history and progressing to angiosarcoma independent of subsequent exposures. Intuitively, we think the latter mechanism may be happening in animals because of the independence of latency time with respect to dose. However, in people, there is evidence of latency time variation with dosage (see reference 7). If there are indeed different mechanisms in rats and people the ratio of exposure durations computed above has no valid interpretation.

Latent time considerations would dictate that the annual incidence of human cases should be 0.078 times the annual incidence of rat tumors. The model in Appendix B assumed a factor 0.033, so that exposure duration considerations would dictate that the rates calculated from the model should be multiplied by 2.4.

One intuitive measure of the sensitivity of a species is to compare for similar doses, the time it takes to get cancer, expressed as a fraction of total lifespan. For rats this is 80/100 = 0.8 of a lifetime irrespective of dose and for humans it is 19.6/72 = 0.27 of a lifetime. By this measure, humans are over three times more sensitive than rats.

Comparison of Angiosarcoma Incidence

The estimation of liver angiosarcoma incidence in workers is possible from several epidemiology studies, but unfortunately the only study in which it was possible to define the vinyl chloride exposure (reference 4) had too small a sample size to detect liver angiosarcoma cases.

In this plant, measurements of vinyl chloride sampled in the breathing zone of the workers were begun in 1950, and continued until the

present. It was found that in an old section of the plant, exposure in the wet end operations averaged 120-385 ppm, over an 8 hour/day 5 day/week time period, with documented excursions up to 4000 ppm. The measured excursions coincided with reports of workers feeling dizzy on the job. In 1959, following reports of toxicological effects of vinyl chloride, the operations were improved, a continuous monitor was installed and subsequent exposures for the same job were reduced to 20-80 ppm with excursions of 500 ppm. In a newer section of this plant the highest exposure job was 135-825 ppm before 1959 and 30-240 ppm thereafter.

Since this is one of the largest companies and one of those most concerned with worker exposure, it is likely that these are lower than the industry average, so that one might assume from this that the cases of angiosarcoma occurring among workers in the last 5-10 years were the result of past chronic exposures of 200-500 ppm.

In Table 1 the incidence of angiosarcoma from several industrial studies is summarized. The Tabershaw-Gaffey study (reference 5) of records of 8384 men in the entire vinyl chloride industry classified 1817 of them in the high exposure, high duration category. Since the study encompassed 33 plants, it was not possible to define objectively what "high" exposure was, but instead each plant classified cases high or low considering only its own experience. The term "high duration" here means more than 60 months on the job. They found two angiosarcomas in the 1817 people, but from detailed studies by other investigators an additional four were found to be misclassified as other diseases, so that the incidence of angiosarcoma among the high exposure, high duration workers was found to be 6/1817 = 3.3 x 10-3. The average exposure duration for this group was 15.9 years.

Nicholson et. al. (reference 6) studied 255 workers employed in one plant for more than 5 years. Of these, 151 worked in direct PVC production and 3 cases of liver angiosarcoma were found in this group for an incidence of 20 X 10-3. They were exposed for 14, 17, and 23 years respectively. The exposure duration for the entire 255 workers ranged from 5 to 28 years, and most of them were exposed for more than 23 years.

In the Heath and Falk study (reference 7) of angiosarcoma cases in the Louisville plant, 7 cases were seen among 270 people working directly in polymerization activities, for an incidence of 26 X 10-3. The mean duration of exposure (employment) was 17 years in the seven cases. The authors stated that these 270 workers represent about 20% of the plant employees.

The study of J. K. Wagoner (reference 8) of 745 male workers in two plants, 31 cancer deaths, 6 of them liver cancer were reported. This population of workers had been engaged in the polymerization of vinyl chloride for at least 15 years. The type of liver cancer was not specified, so that this study can yield only an upper limit of the liver

angiosarcoma incidence; it is less that 6/745 = 8 X 10-3. Since no breakdown of job categories was made in this study it is likely than many of the 745 people did not have the highest exposure tasks.

In comparing the incidence in table 1, it is significant that studies with the larger number of people have the lowest incidence and the studies with the most complete definition of worker exposure have the highest rate. It can be concluded that the incidence among just the most highly exposed workers is about 0.02.

The incidence numbers in Table 1 represent the number of cases diagnosed before 1974 divided by population exposed before 1974. These numbers, however, do not really represent the true incidence of the disease because all members of the population exposed before 1974 have not had time to develop angiosarcoma because of its 17-year average exposure duration. At least two alternative approaches are available to adjust for latency time effects. The first approach described in this paragraph assumes a fixed exposure duration and considers only the population exposed earlier than this time. The second approach described in the next section considers the entire exposure duration distribution. In the first approach the incidence is expressed in terms of the population exposed before 1974-17 = 1957, which is only a fraction of the population exposed before 1974. To calculate this fraction, information concerning the number of people entering the work force as a function of time from reference 5, table 2 has been used. It is found that the ratio of people whose exposure began before 1974 to the number whose exposure began before 1957 is 3.8. Therefore the population in which cases of angiosarcoma have appeared is 1/3.8 as much as the population in table 1, and the incidence among workers is 3.8 times that given in table 1, or 0.02 X 3.8 = 0.076. This means that sometime in their lives, over 7.5% of all highly-exposed workers are expected to get liver angiosarcoma.

The average exposure time for high duration workers reported in the industry-wide Tabershaw-Gaffey report (reference 5) is 16 years. Therefore, the incidence rate, defined as the number of cases per person-year of exposure, is 0.076/16 = 0.00475 per person-year.

Alternative Approach to Calculating Incidence Rate

The difficulty with the previous approach is that a fixed exposure duration was assumed whereas the actual distribution of exposure durations is approximately normally distributed in log (duration) for the 14 reported occupationally-exposed cases. For each year, t, of exposure, n(t) person-years of exposure have occurred, and the number of cases, c(t), that have occurred before 1974 due to the exposure at time t is

$$c(t) = n(t)\ p\ L(74-t)$$

where p is the probability per person-year of exposure that a person

will get angiosarcoma sometime in his life after being exposed for one year, and L(74-t) is the probability that the exposure occurred a long enough time ago to appear by 1974 if he was exposed in year t. The function L (x) is the cumulative distribution of latency times and is equal to the probability that the latent time is less than or equal to X years. The total number of cases is the sum of c(t) over all times from the beginning of exposure to times so recent that L (74-t) is vanishingly small. For convenience we shall write n(t) as the product of the total number of person-years of exposure, N, and a distribution function, q(t) which is the fraction of the N person-years that occurred in year t. With these quantities defined, the probability p can be calculated from

$$p = \frac{\sum c(t)}{N \sum q(t) \times L(74-t)} \qquad (1)$$

For this calculation the function q(t) was derived from table 2 of the Tabershaw study (reference 5), where the distribution of man-years of employment is presented as a function of the year in which exposure started. It is assumed that the distribution of man-years is the same for highly-exposed employees as for all 7128 workers in his study. The distribution of exposure durations for the 14 known occupational cases of liver angiosarcoma was taken from reference 2 and used for L(74-t). With these functions, the cumulative product in the denominator of equation (1) is 0.378. This is the fraction of cases which have been initiated by exposure through 1974 which have been diagnosed.

Therefore the overall effect of this more exact treatment, where the exposure duration distribution is explicitly considered, is to multiply the calculated probability by 1/0.378 = 2.65. This turns out to be not much different than the earlier approach, where the factor was 3.8. We consider the alternative approach to be conceptually more firmly based. Therefore we obtain 0.02/(17 X 0.378) = 0.0031 as the probability per year of exposure that a highly-exposed worker will get angiosarcoma sometime in his life. The errors in this calculation are of two types: (a) uncertainties in the numerical estimates of parameters; and (b) conceptual errors in the assumptions of the model. The first type can be quantified in the following way. The chronic dose experienced by highly-exposed workers is estimated to be somewhere between 200 ppm and 500 ppm. The value of 350 ppm was chosen as the mid-point of that range, so the variation could be $\pm$ 150 ppm, or $\pm$ 43%. Although this error does not strictly enter into the calculation of the 0.0031 value, it does contribute to the error when this is compared with the animal data, as discussed on page D-6. The incidence of angiosarcoma in highly-exposed workers was 0.02 (table 1) and by intuition the author would guess that it should be within the range of 0.01 or 0.03, for a variation of 50%. The number of hours of daily exposure in the workers is almost certainly not as much as 8, but may well be over 6 hours, so one may say it is 7 $\pm$ 1 or 7 $\pm$ 14%. If we assume that these errors are random, the error in the product of these factors is

$$\sqrt{(0.43)^2 + (0.50)^2 + (0.14)^2} = 0.67$$

The conceptual errors in the assumptions of the model are not directly quantifiable. One error is that we assumed no breaks in exposure once employment began. People who left the job and returned later have contributed their man-hours of exposure more recently than we assumed, and would therefore not contribute as much to the occurrence of cases observed today. This has caused us to over-estimate the incidence. A second error is that, as discussed on page D-2, the distribution of latent times is biased toward shorter exposure intervals than is likely to be the actual case. The error acts in the same direction as the first, and would cause a further over-estimate of the incidence.

A third error is that the true biological latent time is not directly observable, since the process leading to irreversable disease may have been initiated some unknown time after the first year of exposure. By using the duration of total exposure, we tend to over-estimate the true biological latent time. This error operates in a direction opposite to the preceding two errors. The net result of these of these three errors is unknown in direction or size.

The overall conclusion is that the incidence rate is 0.0031 per person-year of exposure with a quantifiable error due to uncertainties in estimation of parameters of 67% and an additional unknown conceptual error in the precise assumptions.

It is important to point out that the incidence rates calculated in this Appendix are probabilities based on the duration of exposure to vinyl chloride, rather than on the number of cases which happen to be diagnosed in a population each year. In descriptive epidemiology the latter concept is called the incidence rate. The epidemiological incidence in the general United States population is discussed in Appendix E.

Comparison With Animal Data

At this point we are in a position to compare the human annual incidence rate (0.0031) with that predicted from the animal model in Appendix B. For continuous exposure to 350 ppm, Appendix B would predict an incidence rate of $(0.071 \times 10^{6}) \times 3.5 \times 10^{5}) = 0.0249$ per person-year of exposure. If this is adjusted to occupational exposure conditions by dividing by $(24 \times 7)/(7 \times 5) = 4.8$, the incidence rate for highly-exposed occupational groups is predicted to be 0.0052 per person-year of exposure. This is a factor of 1.68 higher than the human rate. Therefore all of the estimates in Appendix B should be multiplied by $1/1.68 = 0.60$ to be consistent with the human data. When the error estimates are explicitly taken into account, we find for the animal model, that the range encompassed by plus or minus one standard error is $(0.0052 \pm 27\%) = 0.0038$ to 0.0066 and for the human data it is $(0.0031 \pm 67\%) = 0.0010$ to 0.0052. Therefore, within the accuracy of our procedures, the two approaches for calculating the incidence are in agreement.

The rationale for comparing human incidence data with the rat experiments is considered more firm than the alternative approach of comparing latency times, as discussed on page D-1, because of the conceptual difficulties already pointed out.

The other effects of vinyl chloride besides liver angiosarcoma are evaluated in the remainder of this appendix.

Ratio of all cancers to liver angiosarcoma

The study of Nicholson, et al. (reference 6) showed that out of 255 workers 24 deaths were recorded, 9 of them were due to cancer, 3 of which were liver angiosarcoma. Unfortunately this study cannot be used to estimate the number of non-angiosarcoma cancers caused by vinyl chloride because if the 3 angiosarcomas were not in the population, there would be 6 cancers out of 21 deaths, which is not different than one would expect in the general population. Reference 15, p. 2 indicates that of all deaths in the general population, 1/5 of them are due to cancer.

In the Tabershaw study of mortality in the VC-PVC industry (reference 5) the cancer mortality of the high exposure group is only slightly higher than the overall cancer mortality for the entire group. Although this slight excess could be due to vinyl chloride exposure, it is not enough to enable one to estimate how many cancers of all sites are induced relative to liver angiosarcoma.

Studies by Ott, et al. (reference 4) and Wagoner (reference 8) have shown statistically significant excesses in mortality from many primary cancer sites, including brain, respiratory system, and lymphomas. However, angiosarcomas were not observed, so that the ratios of total/angiosarcoma incidence cannot be estimated from these studies.

In a proportional mortality study of 161 workers in the Louisville plant, Monson, et. al. (reference 16) found a larger rate of cancers than expected for U. S. white males at the following sites: digestive tract (excluding liver), liver and biliary tract, lung and brain. They found 41 deaths due to all cancers (compared with 27.9 expected) for an excess of 13 cases. In the same population there were 5 deaths due to liver angiosarcoma (compared with a vanishingly small number expected). Therefore the total number of cancer deaths is 13/5 = 2.6 times the number of liver angiosarcoma deaths. One cannot be absolutely sure that all of the cancers in this population are due to exposure to vinyl chloride, but in view of the other studies cited above, it can be said that vinyl chloride is a strong risk factor in the development of other cancers. In the rat experiments of Maltoni, a total of 76 animals had Zymbal gland carcinomas and liver angiosarcoma combined, compared with 47 with liver angiosarcoma, for a ratio of total cancer/liver angiosarcoma of 1.62. When all tumors were considered in the Appendix B, the ratio was 2.0. Therefore, both the human and animal data show that vinyl chloride induces about twice as many total number of cancers as liver angiosarcomas alone.

Freouency of Non-Cancer Effects Relative to Liver Angiosarcoma

In a literature review of 12,724 vinyl chloride workers in the United States and Europe (reference 12) Marsteller, et al. found 118 cases of acroosteolysis (a gradual erosion of bone at the fingertips), 97 people with symptoms of Raynaud's syndrome (cold hands and feet), 40 people with skin legions, and 73 with liver disturbances. We might assume that, of these 328 cases of reported symptoms, 20% of the people had 2 symptoms each and the other 80% had one each, so that the total number of people reporting symptoms in 328/1.2 = 273 people. This incidence of overt symptoms (273/12,724 = 21 X 10^{-3}) is to be compared with the incidence of angiosarcoma in a similar large worker population such as is seen in reference 5, Table 1, which is 3.3 X 10^{-3}. The ratio is 7 people with symptoms for every liver angiosarcoma case.

In detailed clinical observations of 50 highly exposed people currently working in plants, Marsteller (reference 12) finds that only one had a history of liver disease and 8 had experienced Raynaud's syndrome. Despite this lack of overt symptoms, he found that 38 of the 50 had pathologically high BSP retention times (a measure of serious cellular liver damage), 31 had marked liver enlargement, which could be palpated, and 42 had abnormally low blood platelet counts. Therefore for every person with clinical symptoms of vinyl chloride exposure there is expected to be 38/9 = 4.2 people with liver malfunction serious enough to cause abnormal BSP retention times. This brings the ratio of serious liver malfunction to angiosarcoma to 4.2 X 7 = 30.

The frequencies of abnormal BSP measurements and low platelet counts seen by Veltman (reference 13) in 70 vinyl chloride workers are almost identical to those found by Marsteller, but Veltman found only half the frequency of Reynaud's syndrome.

In a liver function screening program among 1183 workers at Louisville, Creech and Makk (reference 14) used a large battery of blood chemistry tests of liver function followed by more elaborate diagnosis of people who were abnormal in the initial screen. In this series of tests, 59 people were found to have liver function abnormalities; 17 of them major abnormalities which required biopsies and other elaborate tests. The plant physicians considered the 59 cases abnormal enough to justify moving the people to work areas where there were no hepatic toxins. Of the 17 serious cases, 2 turned out to be liver angiosarcoma. Therefore from this study, the ratio of definitive liver toxicity to liver angiosarcoma cases is 59/2 = 30, a ratio which agrees with the literature survey of Marsteller, et al. (reference 12).

TABLE 1 - LIVER ANGIOSARCOMA INCIDENCE AMONG HIGHLY EXPOSED WORKERS

Reference	Number of Angiosarcoma Cases	Number of People Surveyed	Incidence ($X\ 10^{-3}$)
5	6	1,817	3.3
6	3	151	20
7	7	270	26
8	< 6	745	< 8

RSV 0013871

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Appendix E

Is Residence Near Vinyl Chloride Plants a Risk Factor In Frequency Of Deaths Due To Liver Angiosarcoma?

I. Introduction

Abundant evidence exists that vinyl chloride is one of the chief causative factors for the high frequency of liver angiosarcoma among highly-exposed workers in the vinyl chloride-polyvinyl chloride industry. In proposing to regulate air emissions around these plants on the basis of health hazards to people living near factories, it is important to look for evidence that this disease occurs more frequently among people living near plants than those living at random places in the United States. Accordingly an investigation was made of the place of residence, relative to the vinyl chloride and polyvinyl chloride plants which are covered by the proposed regulation, of the people who thus far are known to have contacted liver angiosarcoma.

II. Sources of Data

A. Plants and Locations

The list of plants and their addresses which was used in the investigation is shown in Table I. It was compiled from the APHA report (reference 1) and the EPA Air Programs Office document (reference 2). This list includes some plants that are no longer in operation and at least one plant which is reported (by APHA) to involve no current work with vinyl chloride. Some of the plants make co-polymers with vinyl chloride and other monomers. The list is believed to include all plants in the contiguous states which make vinyl chloride monomer or polyvinyl chloride resin. The fact that it contains extra plants does not detract from this study; in fact it might be desirable to expand the investigation to all plants known to fabricate plastics if it were not for difficulty that such an expansion would excessively broaden the list of chemicals which could be implicated as causal agents.

In several cases street addresses of the plants are not available. In these cases it was assumed that the plant was somewhere within the city limits of the postal address (for large cities) or was exactly at the center of town (for small towns or suburbs with no definite boundaries).

B. Liver Angiosarcoma Cases

In September 1974, Dr. Henry Falk of the Cancer and Birth Defects Section, Center for Disease Control (CDC) initiated an extensive case-finding national survey of all liver angiosarcomas which have been

diagnosed between 1964 and 1974. He obtained the information from three sources:

(1) A systematic examination of death certificates filed at the National Center for Health Statistics, Research Triangle Park, N. C. Copies of death certificates were obtained for those deaths where the diagnosis was similar to, or could easily be confused with, liver angiosarcoma. Death certificates always give date and place of death, age, sex and race, date (and frequently place) of birth, residence and occupation at time of death, occasionally the time lived at the residence, the immediate and underlaying cause of death and a statement of whether an autopsy was performed.

(2) Case records on file with the Armed Forces Institute of Pathology. These included cases diagnosed at military hospitals. Usually residence and occupational information were not provided, and for the most part only age, sex, hospital location, autopsy number and a minimum amount of clinical information were available from this source.

(3) Response to requests for information originally sent out by Dr. Falk to state public health departments and hospital pathologists throughout the nation. Typically a letter from a state official would list cases on record and Dr. Falk would send letters to the pathologists identified by the state requesting further information and permission to contact physicians and patients. This process invariably results in complete clinical histories of the patients, (dates of hospital admission, description of methods of diagnosis, detailed surgical and autopsy reports) but rarely includes more than a bare minimum of personal information such as residence and occupation.

Currently Dr. John Herbert at CDC is continuing the investigation by interviewing next of kin and physicians in order to get residence, occupational and medical histories, family members with cancer, possible exposure to other chemicals through home hobbies, drinking and smoking histories and any other personal information which could indicate causal connection with the disease. As of early October, Dr. Herbert has followed up about 20 cases in this way.

In order to confirm the initial diagnosis of liver angiosarcoma, Dr. Louis B. Thomas, Director, Pathology Laboratory, National Cancer Institute has agreed to examine the sections of tissue which CDC obtained from the hospitals. This confirmation is regarded to be absolutely essential, especially when attempting to draw conclusions from a small number of cases. The information currently on hand is far from complete. As of early October 1975, a large backlog of slides was awaiting confirmation at the NCI and over 90% of the cases have not yet been followed up by CDC.

III. Procedures of this Study

With the cooperation of Dr. Herbert, the author, assisted by Dr. Kenneth Cantor, went to CDC and extracted the available information on liver angiosarcoma cases. The form shown on Attachment A was filled out to the extent possible for each case in the files. To maintain confidentiality of personal information, the names of the deceased were not copied from CDC records. Instead the cases are identified in our files by state and initial letters of the name.

A total of 286 cases were identified. Out of this total the cases with information on place of residence at time of death were selected; this group was further examined and cases with known or suspected exposure to arsenicals or thorotrast and cases of known occupational exposure to plastics fabrication and VC-PVC favrications were eliminated. A subsample of the entire group of 286 cases was analyzed for age and sex distribution and tested on a state-wide basis for clustering to a greater degree than expected on the basis of population concentration.

Following the case selection procedure, the place of residence of all selected cases was identified using maps and atlases of various scales.

The initial survey of residences was done by locating cities and towns in an encyclopaedia atlas (reference 3). This was found to be adequate to locate residences to within 10 miles from plant locations. In cases that were suspected to be within 10 miles of the plant, indexed street maps were consulted at the Geography and Map Division, Library of Congress, Alexandria, Virginia.

The plant locations had been identified on a series of U.S. Geological Survey topological maps (scale of 1: 250,000, or 10 statute miles = 6.4 cm) by Teknekron, Inc. under contract with EPA's Office of Planning and Evaluation. These plant locations were verified by detailed street maps at the Library of Congress, for those plants where residence of cases was less than 10 miles from the plants.

IV. Results of the Survey:

Of the 286 case records obtained from CDC, all cases from the states alphabetically from A through M (a total of 166 cases where age and sex is known) were analyzed for age distribution and sex ratio and a test of gross clustering was performed. Such analysis for the entire population of 286 cases will be performed at a later time. It was found that of the 166 cases, 119 were males and 47 females, for a sex ratio of 2.5 males/1 female. The age distribution for females is approximately constant for all ages. It is more variable for males, but there are not obvious bimodalities. When the male/female sex ratio is examined at a function of age, it is found that for all cases less than age 44, the ratio is about 1.0 (27 males/20 females), but for ages above 45 it is larger than a factor of three.

An X^2 test was performed to test the hypothesis that the number of cases in each state is proportional to the 1970 population of the state. If the data should disprove the hypothesis, we would have evidence of large-scale (state-wide) clustering of cases which cannot be explained by population concentrations. The results were that (for the states A through M) X^2 was no larger than chance variation would predict, so that there is no evidence of clustering on a state-wide scale.

Of the 286 case records obtained from CDC, 176 cases remained after elimination of those with no residence information and possible thoratrast, arsenical, and occupational VC exposures. The rejected cases included 12 possible thoratrast exposures, two possible arsenical exposures, one person exposed occupationally to plastics, and six cases from Alaska and Hawaii.

After locating the residence cities and towns of the 176 cases using an United States Atlas, there were only a handful of cases close enough to the plants to justify searching the detailed maps at the Library of Congress. Cases of distances larger than 20 miles from the plant were only rarely recorded, and to date a complete tabulation of number of people versus distance in the range of 5 to 20 miles of all plants has not been made. However, for distances less than five miles, there were only six cases, as shown in Table II.

Case number 1 of Louisville, Kentucky appears on this list through a clerical error because by mistake his work address was traced and found to be less than 5 miles from the plant, whereas the home address is the subject of this study. This accident does cause one to speculate whether a systematic investigation of employees working near vinyl chloride plants would reveal some larger than expected rates of angiosarcoma. It also emphasizes the importance of locating place of occupation in the case of follow-up.

Cases 2 and 4 must be eliminated as candidates for vinyl chloride induced liver angiosarcoma caused by living close to factories. Case 2 moved from Egypt to Jersey City just two years before his death. Cases 4 and 5 turned out to be other types of cancer, according to NCI pathologists.

It is still possible that Cases 1, 3, 5 and 6 can be attributed to residence near vinyl chloride plants, but we will not know until NCI confirms or denies the diagnosis, and until there is more complete follow-up of the residence history of Case 6.

Case 6 is a special situation because he was an infant who died at 5 months of age from a liver cancer which is relatively common among infants. This case must be eliminated from the formal study because it is not a liver angiosarcoma. It could have been caused by vinyl chloride, but we have no knowledge of any facts about the parents except residence at the time of their son's death. Since transplacental carcinogenesis has been observed in animal studies, it is possible that Case 5 is a case of cancer caused by residence close to a vinyl chloride plant.

V. Discussion of Results

Liver angiosarcoma is an extremely rare disease among the United States population. According to data in the Third National Cancer Survey (reference 4) the incidence rate averaged over the nation is 0.0128 per 100,000 population per year. At this rate, the expected yearly incidence in the United States population (203 million people) is 26 per year, and the expected incidence among the 4,580,000 people living within 5 miles of the plants listed in Table I is 0.59 cases per year. Therefore for the 10 year collection of cases in the CDC files we would expect 5.9 cases to occur within 5 miles of all plants if the presence of the vinyl chloride plant contributed no risk factor pre-disposing people to the disease.

The number of cases in our study, 286, is close to the expected 10 year total of 260, and the 3 possible angiosarcoma cases within five miles of vinyl chloride plants would probably match well with the 5.9 cases expected if we could get residence information and trace addresses for the cases in which we have no information. Since we could only trace 176 of the 286 cases, the approximate number of cases close to the plant would total 3 X (286/176) = 4.9 if all were traced. For these reasons our files probably contain most of the cases actually recorded in these ten years.

The high male/female sex ratio and the striking manner in which it increases above age 45 are indications that this is an occupationally-exposed population, and there may be other factors in addition to vinyl chloride.

This survey has produced no evidence that living around vinyl chloride plants is a risk factor in the occurence of liver angiosarcoma. This conclusion is far different than saying that living around plants is not a risk factor for several reasons: 1) this type of survey of a disease with a latent time from first exposure to diagnosis of 17 years reflects exposures that started at some time before 1957, when the quantities of vinyl chloride produced were much smaller than the current production levels. 2) This survey did not include the place of occupation of the currently-suspected collection of liver angiosarcoma cases. Therefore it underestimates the risk of being near a vinyl chloride plant. 3) This survey might not have detected all existing liver angiosarcoma cases, although the number we have is consistent with the national statistics. The circumstantial evidence for this is that there was not substantial overlap between the three sources of case information. If the data sources had been complete, the information collected by CDC from the National Center for Health Statistics would contain all the cases

reported by both the Armed Forces Institute of Pathology and the state health departments. 4) In over 90% of the cases traced in this survey, the only information available was the residence at the time of death, and in some cases, the residence of the spouse or parent only. This information is only a crude indication of where the individuals spent most of their lives. 5) In contrast with our expectation when the survey was started, there is a significant rate of changes in diagnosis after the slides were confirmed by the National Cancer Institute. The 286 cases currently on file cannot be regarded as definitely-established liver angiosarcomas.

In view of these limitations of the survey, positive statements cannot be made about residence near plants being or not being a risk factor. The only interpretation of these results that is justified is that a search was made of the residence information which exists and this search revealed no evidence that residence near plants is a risk factor. In view of the long 17-year latent time it is expected that any survey of current cases would underestimate the actual risk.

Table I: List of VC-PVC Plants and their Locations

Company	Type VC/PVC)	Location
1. Air Products and Chemicals, Inc.	PVC	Pace, Florida
2. Air Products and Chemicals, Inc.	PVC	Highway 95 Calvert City, Ky.
3. Allied Chemical Corp.		near River Mile 2355 Gulf State Road Baton Rouge, La.
4. Allied Chemical Corp.	VC	Geismar, La.
5. American Chemical Corp.	VC, PVC	Long Beach, Calif.*
6. Atlantic Tubing & Rubber	PVC	Cranston, R.I.*
7. BAST Wyandotte		500 Central Ave. S. Kearney, N. J.
8. Borden, Inc.		Geismar, La.
9. Borden, Inc.		Bainbridge, N.Y.*
0. Borden, Inc.		Compton, Calif.*
11. Borden, Inc.		Demopolis, Ala.*
12. Borden, Inc.	PVC	Illiopolis, Ill.
13. Borden, Inc.	PVC	Leominster, Mass.*
14. Borden, Inc.		near 59th & Interspace St. Oklahoma City, Okla.
15. Continental Oil Co.		Lake Charles, La.
16. Continental Oil Co.	VC	Old Spanish Trail Westlake, La.
17. Continental Oil Co.	PVC	Aberdeen, Miss.
18. Diamond Shamrock Corp.	PVC	River Road Delaware City, Dela.
19. Diamond Shamrock Corp.	VC, PVC	La Porte Highway Deer Park, Texas

Company	Type (VC/PVC)	Location
20. Diamond Shamrock Corp.		Plaquemine, La.*
21. Dow Chemical Co.	VC	Freeport, Tex.
22. Dow Chemical Co.	VC	Oyster Creek, Tex.*
23. Dow Chemical Co.	VC	Plaquemine, La.
24. Dow Chemical Co.	PVC	Midland, Mich.
25. Ethyl Corp.	VC, PVC	Gulf State Road Baton Rouge, La.
26. Ethyl Corp.	VC	Deer Park, Tex.
27. Firestone Tire & Rubber Co.	PVC	Firestone Blvd. Pottstown, Pa.
28. Firestone Tire & Rubber Co.	PVC	Perryville, Md.
29. Foster Grant Co.		389 N. Main Street Leominster, Mass.
30. General Tire Co.	PVC	Ashtabula, Ohio
31. B. F. Goodrich Co.	PVC	Walker & Moore Road Avon Lake, Ohio
32. B. F. Goodrich Co.	PVC	2104 E. 23rd Street Carson City, Calif.
33. B. F. Goodrich Co.	PVC	41st Street & Bells Lane Louisville, Ky.
34. B. F. Goodrich Co.		188 Presidio Place Williamsville, N. Y.
35. B. F. Goodrich Co.	VC	near River Mile 17 Tennessee River Calvert City, Ky.
36. B. F. Goodrich Co.	VC	Henry, Ill.
37. B. F. Goodrich Co.	PVC	Pedrickstown, N. J.
38. Goodyear Tire & Rubber Co.	PVC	Plaquemine, La.

Company	Type (VC/PVC)	Location
39. Goodyear Tire & Rubber Co.		5408 Baker Avenue Niagara Falls, N. Y.*
40. W. R. Grace & Co.		Owensboro, Ky.*
41. W. R. Grace & Co.		South Acton, Mass.*
42. Great American Chemical Corp.	PVC	Fitchburg, Mass.*
43. Keysor-Century Corp.	PVC	2600 Springbrook Avenue Saugus, Calif.
44. Monochem, Inc.	VC	Geismer, La.*
45. Monsanto Co.	PVC	730 Worchester Street Indian Orchard, Mass.
46. Monsanto Co.		Texas City, Tex.
47. Morton Norwich Co.		Ringwood, Ill.*
48. National Starch & Chemical Corp.	PVC	Meredosia, Ill.*
49. Occidental Petroleum Corp.	PVC	River Road Burlington, N. Y.
50. Occidental Petrolium Corp. (Hooker Chemical)	PVC	New South Road Hicksville, N. Y.
51. Olin Corporation	PVC	238 S. Main St. Assonet, Mass.*
52. Pantasote Co.	PVC	26 Jefferson Street Passiac, N. J.
53. Pantasote Co.	PVC	Point Pleasant, W. Va.*
54. Pittsburgh Plate Glass Industries, Inc.	VC	Columbia Southern Road Lake Charles, La.
55. Robintech, Inc.	PVC	786 Hardy Road Painsville, Ohio
56. Shell Oil Co.	VC	State Highway 225 Deer Park, Tex.
57. Shell Oil Co.	VC	Norco, La.*

Company	Type (VC/PVC)	Location
58. Schintech	PVC	Freeport, Tex.*
59. SCM Corp. (Huron Plastics)		Huron, Ohio
60. Stauffer Chemical Co.		2112 E. 223rd Street Carson City, Calif.
61. Stauffer Chemical Co.	PVC	Delaware City, Del.*
62. Stauffer Chemical Co.		School House Road Burlington, N. J.
63. Tenneco Chemicals, Inc.	PVC	Beverly Road Burlington, N. J.
64. Tenneco Chemicals, Inc.	PVC	Flemington, N. J.*
65. Tenneco Chemicals, Inc.	VC	Painesville, Ohio
66. Tenneco Chemicals, Inc.	VC	4403 La Porte Road Pasadena, Tex.
67. Union Carbide Co.	PVC	4337 McCorkle, South Charleston, W. Va.
68. Union Carbide Co.	PVC	Highway 146 & Texas Avenue Texas City, Texas
69. Union Carbide Co.		Taft, La.
70. Union Carbide Co.	Reported Closed	Niagara Falls, N. Y.*
71. Uniroyal Inc.	PVC	720 Fairport Nursery Road Painesville, Ohio
72. Vulcan Materials Co.		Geismar, La.
73. Vulcan Materials Co.	VC	Deer Park, Texas*

*Location not included in APHA estimate of total population.

ANGIOSARCOMA WORKSHEET

CDC #: _____ Name: _____ Sex: _____

Death Certificate: Place of Death: _____ Date of Death: _____

Biopsy Performed: Yes ☐ No ☐ Date of Biopsy: _____

Autopsy Performed: Yes ☐ No ☐ Date of Autopsy: _____

Specimens Available: Yes ☐ No ☐

Diagnosis: _____
(Immediate Cause)

_____
(Underlying Disease)

Date of Birth: _____ If not known: Age (at biopsy): _____

Age (at autopsy): _____

Age at Death: _____

Date of Diagnosis: _____ Age at Diagnosis: _____

Residence at Time of Death: _____

Occupation at Time of Death: _____

Family: Married: Yes ☐ No ☐

Children: Number: _____ Ages at Death: _____

Pathology Reviews:

Specimens Received: Yes ☐ No ☐ Unavailable ☐

Specimens Reviewed: Yes ☐ No ☐

NIH Report: _____

AFIP Report: _____

CDS Report: _____

Followed up by CDC: Yes ☐ No ☐

CDC #: _____ Other Patient I.D.: _____

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Occupational History

	Name of Employer	Address	From	To	Job Title	Job Description	VC	As	Other Chemicals
.									
1.									
1.									
1.									
1.									

Residence History

	City, State	Address	From	To	Distance from VC Plant	Distance from Other Industry
1.						
2.						
3.						
4.						
5.						

Members of family exposed via occupation or residence? Yes [ ] No [ ] If yes, use additional sheet.

Table II Case Summary

Case Identification	Distance from Plant	Time in Area	Age at Death	NCI Confirmation
1. JLK, Louisville Kentucky	Home: 6.7 mi. SSW of B. F. Goodrich Work: 4.3 mi. ESE of B. F. Goodrich	32-1/2 years	33	No
2. IN, Jersey City, New Jersey	Home: 2.0-2.4 mi. SE of BAST Wyandotte	2 years	41	No
3. HCM, Buffalo	Home: Age 0-20: Distance 7-9.2 mi SW of B.F. Goodrich 20-40: 2.6-4.4 mi. WSW of B.F. Goodrich 40-58: 8-9.5 mi. SSW of B.F. Goodrich 58-68: 3.8-5.2 mi. SW of B.F. Goodrich	68 years	68	No
4. CF, Niagara Falls	Home: 0.25 to 1.1 mi. SSE of Goodyear	30 years	63	Yes, not angiosarcoma
5. GB, Niagara Falls	Home: 0.25 to 1.0 mi. NNW of Goodyear	5 months	5 mo.	Yes, not angiosarcoma
6. RJ, Niagara Falls	Home: 2.1-2-2.3 mi. W of Goodyear	?	41	No

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Appendix E References

1. Landau, E., "Population Residing Near Plants Producing Polyvinyl Chloride," American Public Health Association, Contract report, EPA/OTS (August 1975).

2. EPA, OAQPS, Emission Standards and Engineering Division, "Standard Support-Environmental Impact Statement, Vol. II (draft)." April 1975.

3. Encyclopaedia Britannica, "Britannica Junior, Vol. 15" (1959).

4. National Cancer Institute Monograph 41, "Third National Cancer Survey: Incidence Data," March 1975.