CANCER MORTALITY IN WOBURN

A Three Decade Study (1949 – 1978)

November 17, 1981

Prepared by

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(modified for the Internet February 1999)

We gratefully acknowledge the assistance of Richard Rowe, M.D., Intern from the Harvard School of Public Health, for collecting the data, of John L. Cutler, M.D., Ph.D., Center for Environmental Health, Centers for Disease Control and the Division of Health Statistics and Research, MDPH in the preparation of this report.

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SUMMARY

As a follow up of a study of childhood leukemia and kidney cancer incidence during 1969 – 1978, mortality from these causes was tabulated for the period 1949 – 1978. There was an increase in childhood leukemia mortality starting in 1959 – 1963. No concentration of childhood leukemia deaths occurred in the Walker Pond neighborhood, where the incident case cluster occurred in the 1970's. During 1949 – 1968, kidney cancer mortality was lower than expected but rose sharply during the 1970's. There was no geographic concentration of deaths.

I. INTRODUCTION

This report is one of several by the Massachusetts Department of Public Health on the possible association between cancer incidence and environmental hazards in Woburn. The initial investigation stemmed from public concern about toxic wastes in town dumpsites and the finding of chlorinated organic compounds in two wells (G and H) in 1979. A local clergyman, alarmed by the number of childhood leukemia cases that he had heard of in Woburn, asked the Massachusetts Department of Public Health to investigate the situation. With help from the Centers for Disease Control (CDC), an investigation was carried out in 1980.[1]

The 1980 study revealed an increased incidence of childhood leukemia in Woburn, with a concentration of cases in eastern Woburn. Also there was an elevated incidence of kidney cancer. An interview study did not demonstrate any differences between leukemia cases and controls. There still remained a question as to what effects the contaminated drinking water may have had on the occurrence of cancer. Since the wells were dug in the mid-1960's, a retrospective mortality study of the 1950's and 1960's was undertaken to study trends of cancer mortality in Woburn before, during, and (eventually) after the use of the two contaminated wells. This report covers the period 1949 – 1978 and concentrates on a review of mortality from childhood leukemia and kidney cancer.

II. METHODS

A systematic search through all death certificates at the Woburn City Hall was carried out for the period 1949 – 1968. Every certificate that mentioned cancer was abstracted for pertinent information (name, address, age, sex, cause of death, year). The data were analyzed according to the site of cancer, year of death, age of death and sex. These observed deaths were compared to the expected number of deaths, by computing standardized mortality ratios (SMRs*). The expected deaths were computed using the mortality rates provided by Dr. Richard Monson at the Harvard School of Public Health who has collected data from death certificates on every cause of death in Massachusetts since 1925 and computed age and sex-specific mortality rates. These were computed for each 5-year interval from 1925 – 1975. These mortality rates have been used in previous publications for Boston and Massachusetts mortality profiles.[2,3]

III. RESULTS

A. LEUKEMIA

During the period 1949 – 1978 there were fourteen deaths due to childhood leukemia (less than 20 years). (TABLE 1) Mortality began to rise in 1959 – 1963.

B. KIDNEY CANCER

Mortality from kidney cancer comprised a total of 27 deaths during the period 1949 – 1968, and 19 deaths occurred during the third decade. Mortality was lower than expected during the first two decades, but not significantly so, and it rose rapidly in the 70's.

IV. RECENT ENVIRONMENTAL DATA

The Environmental Protection Agency (EPA) recently issued a report on the quality of ground water in eastern and northern Woburn.[4] Relevant sections of this document are appended. The study indicates that presence of 42 organic EPA priority pollutants in the groundwater of eastern and northern Woburn. The presence of chemical contaminants in groundwater raises the possibility that drinking water from the aquifer may have been contaminated in the past or may become contaminated in the future. Most of

the *SMR = (NO. DEATHS OBSERVED: NO. DEATH EXPECTED) x 100. The number of expected is computed by applying statewide age-and-sex-specific rates to the Woburn population. Organic substances detected do not produce cancer by ingestion, but some are regarded as potential human carcinogens.

V. DISCUSSION

Kidney cancer and childhood leukemia are histogenetically quite different. With the possible exception of radiation, there is no suggestion in the medical literature that agents known to be leukemogenic are also kidney carcinogens. It is therefore unlikely that a single extrinsic agent or factor would account for both the elevated mortality from kidney cancer and the elevated incidence of childhood leukemia during the period 1969 – 1978. Therefore, if either disease is environmentally induced, separate factors must be sought. One of the purposes of this study was to test further whether there was an association between the use of water from wells G and H and the occurrence of leukemia. These additional data seem to weaken this association. The number of childhood leukemia deaths began to rise in the 1959 – 1963 period, before the wells were drilled and continued to rise in the period in which the wells were drilled. Given an average latent period of two to five years, childhood leukemia associated with wells G and H should not have started to increase until 1969 – 1973, when in fact, the rate was lower than expected. The very small number of childhood leukemia deaths, 14 over 30 years, precludes any strong statistical conclusion. If some of the childhood leukemia occurring in the 1970's was related to wells G and H, then the incidence should decline during the next few years, since wells G and H were closed in 1979.

The earlier MDPH report reviewed factors that might be related to kidney cancer development. No relationship to any factor was observed, but possibilities discussed included occupation and place of residence 20 years earlier. The present mortality study lacks the data to examine these relationships. What can be emphasized is that any extrinsic factor would have had to exert its effects 15 to 30 years prior to the 1969 – 1978 decade, assuming a 15 to 30 year latent period for induction of kidney cancer. This would tend to exclude Wells G and H, which began pumping in 1964 and 1968 respectively.

VI. CONCLUSIONS

A three-decade mortality study of childhood leukemia and kidney cancer leads to the following conclusions:

1. Childhood leukemia mortality was not elevated during the period 1949 – 1958, mortality began to rise in 1959 – 1963.
2. No unusual geographic distribution of childhood leukemia mortality occurred during the period 1949 – 1968.
3. These data are consistent with the possibility that, excluding a chance occurrence, the elevated incidence of leukemia and the concentration of cases in eastern Woburn during 1969 – 1978 must have been due to some newly introduced factor which became present and active during the late 1950's. If contaminants present in Wells G and H contributed to this increase, then leukemia should decline in the late 1980's.
4. Mortality from kidney cancer was significantly elevated in the 1970's. Assuming a latent period of 15 to 30 years for induction of kidney cancer, environmental factors introduced after about 1950 can be excluded from having any relationship to the cancer development. It is very unlikely, therefore, that wells G and H are implicated.

REFERENCES

1. Parker, G. S. et. al. WOBURN: Cancer incidence and environmental hazards, 1969 – 1978 Massachusetts Department of Public Health publication, January 23, 1981.

2. Monson, R.R.: Causes of Death in Boston, J. Chr. Dis. 33:21-28 (1979).

3. Monson, R.R.: Mortality in Massachusetts General Population, J. Chr. Dis. 33:29-35 (1979)

5. Ecology and Environment, Inc. Interim Report on the Groundwater Quality of East and North Woburn, Massachusetts. EPA FIT Project TDD No. F1-8010-04B. May 6, 1981.

TABLE I CHILDHOOD LEUKEMIA DEATHS

YEARS	OBSERVED	EXPECTED	SMR
1949-53	1	1.3	78
1954-58	0	1.8	0
1959-63	3	2.5	120
1964-68	4	2.4	169
1969-73	1	2.0	50
1974-78	5	1.4	357
TOTAL	**14**	**10**	**140**

TABLE II KIDNEY CANCER DEATHS

YEARS	OBSERVED	EXPECTED	SMR
1949-53	2	2.7	75
1954-58	3	3.6	83
1959-63	1	4.1	24
1964-68	2	4.6	43
1969-73	8	5.2	154
1974-78	11	4.9	224
TOTAL	**27**	**25.1**	**108**

APPENDIX 1

Note:

The following sections were taken from ***Interim Report on the Groundwater Quality of East and North Woburn, Massachusetts.*** **Ecology and Environment, Inc. EPA FIT Project TDD No. F1-8010-04B. May 6, 1981.**

Figures 6A, 8, 17 and 23 were included by Dr. Telles to highlight the aerial distributions of a selection of environmental contaminants. The Interim Report, however, contains 54 aereal distribution maps of organic and inorganic contaminants detected in Woburn groundwater.

SECTION I – INTRODUCTION

1.1 PURPOSE

To gather groundwater quality data for East and North Woburn, Massachusetts to be used in conjunction with reports on the geology (TDD # F1-8010-02A) and hydrology (TDD #F1-8010-03A) of the area. This data will be used to determine and recommend appropriate remedial actions to be performed using funds provided by the Hazardous Waste Containment Act of 1980.

1.2 OBJECTIVE

1.21- To perform sampling and priority pollutant analysis of thirty-two (32) wells in East and North Woburn.

1.22- To construct areal contamination distribution maps consistent with the groundwater and subsurface geologic data gathered under TDD's #F1-8010-02A and F1-8010-03A.

1.23- To provide human health effects data regarding all detected contaminants.

SECTION 4 – ANALYTICAL RESULTS

The results of the priority pollutant analyses indicate the presence of 42 organic priority pollutants in the groundwater of North and East Woburn. In addition, twenty-two metals were quantified. Following is

a list of all organics and inorganics identified, the maximum concentration detected within the study area for each, and the number of wells in which each was detected.

Acid Compounds	Number of Wells	Maximum Concentration (in ppb)
p-Chloro-n-cresol	1	35
Phenol	5	266
4-Nitrophenol	1	2831

Base/Neutral Compounds	Number of Wells	Maximum Concentration
Fluoranthene	2	21
Naphthalene	1	36
N-Nitrosodiphenylamine	2	275,731
Bis(2-Ethylhexyl) phthalate	11	62,524
Butyl benzyl phthalate	2	38
Di-n-butyl phthalate	5	4,226
Di-n-octyl phthalate	7	<10
Diethyl phthalate	6	<10
Anthracene	1	<10
Phenanthrene	1	<10
Pyrene	1	<10
Nitrobenzene	1	2,669

Volatile Compounds	Number of Wells	Maximum Concentration
Benzene	14	76
Carbon tetrachloride	1	11
Chlorobenzene	1	<10
1,2-Dichloroethane	2	82
1,1,1-Trichloroethane	12	611
1,1-Dichloroethane	8	25
Chloroform	6	<10
1,1-Dichloroethylene	4	20
1,2-trans-dichloroethylene	16	210
Ethylbenzene	3	<10
Methylene chloride	15	23
Trichlorofluoromethane	3	775
Tetrachloroethylene	9	89
Toluene	15	106
Trichloroethylene	27	2,290

Pesticides	Number of Wells	Maximum Concentration (in ppb)
Aldrin	2	<5
Dieldrin	1	<5
4,4'-DDT	5	<5
4,4'-DDE	4	23
4,4'-DDD	2	<5
α – endosulfan	2	<5
Heptachlor	2	<5
Heptachlor epoxide	4	<5
α – BHC	5	5
β – BHC	7	<5
χ – BHC	3	<5
δ – BHC	9	<5

Metals	Number of Wells	Maximum Concentration (in ppb)
Aluminum	17	362,000
Chromium	11	2,070
Barium	29	2,160
Beryllium	1	2
Cadmium	4	40
Cobalt	9	250
Copper	17	47,800
Iron	30	637,000
Lead	10	4,550
Nickel	9	360
Manganese	29	17,200
Zinc	27	17,400
Boron	24	3,170
Vanadium	11	640
Calcium	30	513,000
Magnesium	30	95,000
Sodium	30	295,000
Arsenic	8	7,000
Antimony	1	90
Selenium	1	87
Thallium	3	50
Mercury	8	49
Tin	8	300

SECTION 4- ANALYTICAL RESULTS (CONT.)

Figures 3 to 57 are areal distribution maps of all detected organic and inorganic contaminants with the exception of anthracene, phenathrene, pyrene and chlorobenzene. Each of these four compounds was detected in only one well in quantities of less than 10 ppb as noted below.

Compound	Well
Anthracene	GZ-2
Phenanthrene	GZ-2
Pyrene	GZ-2
Chlorobenzene	439

The areal distribution maps represent only the known extent of contamination. There are many areas with insufficient data because accessible wells were not present. The proposed monitoring wells will provide data to fill the gaps in the existing data.

Analytical data tabulated by sampling location are presented in Appendix A.

ATTENTION:

Figure 6A: The Areal Distribution of Nitrobenzene- East and North Woburn, MA
Figure 8: The Areal Distribution of Benzene- East and North Woburn, MA
Figure 17: The Areal Distribution of Ethylbenzene- East and North Woburn, MA
Figure 23: The Areal Distribution of Volatile Organics- East and North Woburn, MA

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For more information, contact the Massachusetts Department of Public Health, Bureau of Environmental Health Assessment at (617) 624-5757.

Thank you.

5.4 Potential Human Health Effects of Chemicals Identified in Study Area Groundwater, Woburn, Massachusetts

5.41 Chlorinated Organic Chemicals

The chlorinated volatile organics are widely used as solvents. They all have excellent solvent ability and are generally not flammable, two properties which contribute to their wide acceptance in industry. One property which the chlorinated volatile organics have in common is their ability to produce anesthesia and central nervous system depression after inhalation of large quantities. Very little human data exist concerning effects of chronic ingestion of small quantities of chlorinated solvents.

The following chlorinated volatile organics were detected in the groundwater of the Woburn study area:

1,1,1-Trichloroethane – also called methyl chloroform, is one of the most widely used industrial solvents and metal degreasers. 1,1,1-Trichloroethane has been regarded as one of the "safest" industrial solvents. Most of the human toxicity data on 1,1,1-trichloroethane come from studies of industrial inhalation exposures. Levels of 1,1,1-trichloroethane ranging from 1000 ppm to 2000 ppm in air have been reported to cause uncoordination and "drunken" behavior. At slightly lower air exposure levels, human subjects report drowsiness. The toxicological significance of long-term ingestion of 1,1,1-trichloroethane is unknown. Data obtained from animal studies indicate that the liver is affected at near-lethal doses, in the range of 700 – 1000 mg/kg.

1,2-trans-Dichloroethylene – also known as acetylene dichloride, is used as a general purpose solvent and in the manufacture of perfumes, lacquers, thermoplastics and pharmaceuticals. There is no evidence that low concentrations of 1,2-trans-dichloroethylene constitute a human health hazard.

1,1-Dichloroethylene – or vinylidine chloride, is used in the synthesis of saran and other copolymers. It is also used in the manufacture of some adhesives. Unlike its isomer 1,2-trans-dichloroethylene, vinylidine chloride is known to be toxic by ingestion in animals. Liver damage is the most common

effect. Although there are no human data, 1,1-dichloroethylene should be treated as a potential human liver toxin (hepatotoxin). There is also evidence that lifetime ingestion of 1,1-dichloroethylene causes tumors in laboratory animals, but there are no data linking it to cancer in people.

1,1-Dichloroethane – also known as ethylidene chloride, is used as an extraction solvent and in the synthesis of pharmaceuticals. The toxicological properties of 1,1-dichloroethane have not been studied extensively. It has been shown to cause liver injury when fed to laboratory animals over their lifetime in high doses.

1,2-Dichloroethane – or ethylene dichloride, is used in chemical syntheses, as a solvent and degreaser, and in paint, finish and varnish remover. It has been used as an additive in leaded gasoline. 1,2-Dichloroethane vapor is highly toxic. Damage to both the respiratory tract and the cornea of the eye may result from prolonged exposure to 1,2-dichloroethane vapor. There is evidence that long-term ingestion of 1,2-dichloroethane may result in liver and kidney damage. This substance has been demonstrated to cause tumors in rats and mice exposed over their lifetimes. There are no data linking 1,2-dichloroethane with human cancer, but on the strength of the animal data, it should be considered a potential human carcinogen.

Methylene chloride – or dichloromethane, is used as a paint remover, solvent, degreaser, and propellant for aerosols. Methylene chloride is highly volatile, even at low temperatures. The vapor is extremely hazardous to the eyes. When inhaled, methylene chloride is metabolized to carbon monoxide by humans, and carboxyhemoglobinemia, a condition in which the blood hemoglobin will not carry sufficient oxygen, results. It is unknown if this effect will also result from ingestion of methylene chloride. Very little is known regarding chronic toxicity of ingested methylene chloride in people.

Chloroform – or trichloromethane, is used in the manufacture of fluorocarbon propellants and refrigerants, fluorocarbon plastics, and some insecticides. It is also used as a solvent. Chloroform is no longer used as an anesthetic, and the Food and Drug Administration (FDA) has banned its use in throat

lozenges and other pharmaceuticals. Chloroform is a neurological and liver toxin in man, and lifetime exposure has been shown to cause cancer in several species of laboratory animals. Although there is no direct evidence linking chloroform with human cancer, it is considered a potential human carcinogen.

Chloroform is a common contaminant in natural waters in the United States. There is some evidence that the use of chlorination as a disinfectant in wastewater treatment plants may result in the formation of chloroform and other chlorinated organics. Recently, the United States EPA established a drinking water Maximum Contaminant Level of 100 ppb for chloroform. This level constitutes an enforceable standard in public drinking water for suppliers serving over 75,000 persons.

Carbon tetrachloride – is used in the manufacture of chlorofluorocarbon refrigerants and propellants, in agriculture as a soil fumigant, as a metal degreaser and as a source of chlorine in many commercial organic synthetic processes. Carbon tetrachloride causes liver, kidney, and lung injury in humans. Chronic exposure to carbon tetrachloride may cause cirrhosis of the liver. Chronic exposure to carbon tetrachloride causes tumors in laboratory animals and it should be treated as a potential human carcinogen. Because of its extreme liver toxicity, household use of carbon tetrachloride has been banned by the FDA. It is no longer used as a dry cleaning solvent or as a fire extinguishing agent.

Trichloroethylene – commonly called TCE, is used as a metal degreaser, an extraction solvent, a dry cleaning agent, and in chemical synthesis. Trichloroethylene vapor is a recognized hazard in industry, and neurological symptoms have been reported. It is suspected of causing liver injury after chronic vapor exposure. Very little is known regarding effects of chronic ingestion of low levels of trichloroethylene. In lifetime feeding studies, trichloroethylene caused tumors in mice, but not in rats. There is no evidence linking trichloroethylene with human cancer.

Tetrachloroethylene – also called perchloroethylene or PCE, is the most commonly used commercial dry cleaning solvent. It is also used as a vapor degreasing solvent, a drying agent, a heat transfer medium, and an intermediate in the synthesis of fluorocarbons. Tetrachloroethylene has generally been

used safely in the dry cleaning and other industries. It is recognized as a vapor hazard that causes neurological symptoms resembling intoxication. There are no data that describe chronic effects of low level ingestion. Tetrachloroethylene has produced tumors in laboratory animals, but there is no evidence which suggests that it causes cancer in man.

5.4.2. CHLOROFLUOROCARBONS

The chlorofluorocarbons are short-chain aliphatic hydrocarbons in which one or more chlorine and fluorine atoms replace the hydrogens. The chlorofluorocarbons are chemically inert, non-flammable, and heat stable to approximately 600 ºF. They are highly volatile. Many exist in the vapor state at ambient temperatures. They are denser than water and have low surface tensions. They have low water solubilities, but are slightly soluble in organic solvents, oils and lipophilic material. The major industrial uses of chlorofluorocarbons are in aerosol propellants and in refrigerants. Chlorofluorocarbons are used as fire extinguishing agents, lubricants, hydraulic fluids, and dielectric fluids. They are also used in the manufacture of plastics and in air conditioners.

Trichlorotrifluoroethane – or 1,1,2-trichloro- 1,2,2-trifluoroethane has been patented as Freon 113 (DuPont). It is not considered highly toxic. Trichlorofluoroethane is a common refrigerant, air conditioner fluid, and dry cleaning solvent.

Dibromochloromethane – is used in organic synthesis. Its human toxicity has not been studied extensively. Dibromochloromethane vapor is an irritant with narcotic (sleep-inducing) properties.

Trichlorofluoromethane – Freon 11 (DuPont), is used in refrigerators, air-conditioners and fire extinguishers. It also has industrial uses as a solvent and chemical intermediate. Trichlorofluoromethane vapor is a mild irritant. Very few other toxic effects have been reported. There are no data describing its long-term human toxicity.

5.4.3 VOLATILE AROMATICS

Four volatile aromatic compounds have been identified in groundwater samples from the study area. Their properties are dissimilar, so they will be discussed separately.

5.4.6 METALS

Calcium (Ca) – is an alkaline earth element that does not occur free in nature. Calcium compounds are extremely abundant in nature, however, and are widely used in industry, agriculture and medicine. Calcium is an essential human nutrient, and calcium deficiency states are well known in medicine. Calcium is present in foods and natural waters and is considered non-toxic.

Sodium (Na) – is also an alkaline earth element. Like calcium, sodium does not occur in nature in the free state, but sodium salts are ubiquitous in soil, sediments and water. Sodium is an essential element in the human diet. There is evidence linking excess sodium intake with increased incidence of hypertension (high blood pressure). Both federal and Massachusetts guidelines recommend sodium levels of no higher than 20 ppm (20000 ppb) in public drinking water.

There is no evidence that sodium causes high blood pressure. Sodium affects the kidney's ability to regulate water and electrolyte balance in the body. These renal and electrolytic disturbances are more pronounced in people with high blood pressure or who have a tendency to develop high blood pressure. The presence of high levels of sodium in food or water will worsen the tendency to hypertension in people with kidney or heart disease, or who are prone to these diseases.

5.5 SUMMARY

Several organic and inorganic chemicals included on the EPA list of 129 Priority Pollutants have been identified in Woburn groundwater. The presence of chemical contaminants in groundwater raises the possibility that drinking water from the aquifer may have been contaminated in the past or may become contaminated in the future.

Prediction of potential human health effects that may result from contamination of the drinking water supply is difficult because very little information describing human health effects of chronic ingestion of low level chemical contaminants exists. The best source of data on toxicity of environmental contaminants comes from animal experimentation in which laboratory animals are fed specific chemicals daily over their lifetimes. Information obtained from these studies is very useful in determining which organ systems are particularly vulnerable to the chemical insult. These experiments are not always accurate in predicting how man will respond to a particular chemical, and how much of the chemical is required before a human will get sick. Often, the levels of chemicals used in animal experiments may be unrealistic in terms of human experience. High levels of chemicals are used in animal studies because, with high exposure levels, scientists are more likely to see toxic effects in experiments in which a relatively small number of animals are used. Animal studies in which large doses of chemicals are used also present a problem in defining which substances are carcinogens, or tumor-inducing agents. Many substances appear to be able to induce tumors in animals when given in large doses over the animals' lifetimes.

Most of these substances have been used by people in industry over several years and do not appear to be associated with human cancer. Many scientists believe that tumors are the result of a toxic response that occurs when a "threshold dose" of the toxic chemical is exceeded. This theory assumes that cancer is similar to other toxic responses in this respect: there is a dose below which toxicity is not seen and above which adverse health effects appear. There is, however, another theory on the induction of cancer that contradicts the "threshold" model. The "one-hit" model assumes that if a chemical can cause cancer at a high exposure level, then it can also cause cancer at a low exposure level. The only difference is that it becomes more <u>likely</u> statistically that a high dose will cause cancer. There are several other theories that attempt to explain possible causes and origins of cancers; none of the theories can completely explain or disclaim suspected associations between environmental chemicals and human cancer. Most scientists who study cancer agree that most human cancers result from a combination of factors including environment, lifestyle, heredity and nutrition. Although several chemical and physical

agents have been identified as human carcinogens, among them vinyl chloride, asbestos and ionizing radiation, most chemical substances which have caused tumors in laboratory animals can only be labeled "suspected carcinogens" in man. Until more data on actual human exposure to chemicals are obtained, the association between these "suspected carcinogens" and cancer remains tentative.