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TO: Director, Centers for Disease Control

FROM: Chronic Diseases Division
 Center for Environmental Health

SUBJECT: Cancer in Woburn, Massachusetts

SUMMARY

In 1980 the Massachusetts Department of Public Health and the Centers of Disease Control investigated possible associations between environmental hazards and the incidence of childhood leukemia plus three types of cancer -- kidney, urinary bladder, and liver -- in Woburn residents for the period 1969 – 78. This investigation confirmed that the increase in incidence of childhood leukemia there, concentrated in eastern Woburn, was statistically significant when compared with nationwide rates. The incidence of kidney cancer was also significantly elevated, but less so. Because of limited environmental data, the investigation did not establish an association between known environmental hazards in Woburn and either of these higher than expected cancer incidences. Rates for liver and bladder cancer were not significantly elevated.

INTRODUCTION

On December 13, 1979, the Chronic Diseases Division (CDD) received two inquiries about possibly increased cancer incidence in Woburn, Massachusetts. The first came from Milton Kotelchuck, Ph.D., Director, Division of Health Statistics and Research, Office of State Health Planning, Massachusetts Department of Public Health (MDPH), who had examined mortality statistics from the town and found increased mortality rates of various cancers. Local concern was that this cancer excess might be related to chemical waste dumps or chemical contamination of drinking water in the area. The second inquiry came from John T. Truman, M.D., pediatric hematologist at the Massachusetts General Hospital in Boston, who informed Clark W. Heath, Jr., M.D., Director, CDD, that since 1972 he had seen as patients six children with acute lymphocytic

leukemia from a six-block area of Woburn. Dr. Truman recommended that an epidemiologic study be made of this apparent case cluster.

Jean French, Ph.D., Staff Assistant to the Director, CDD, consulted with Dr. Kotelchuck about further evaluation of the mortality data. John L. Cutler, M.D., Ph.D., Staff Epidemiologist, CDD, met with Dr. Truman to review the childhood leukemia cases in detail and with Dr. Kotelchuck to plan a more detailed epidemiologic study.

Case Definition

A case was defined as confirmed kidney cancer, liver cancer, urinary bladder cancer, or childhood leukemia in a Woburn resident under 19 years of age, whose cancer was diagnosed between 1969 and 1978 (1969 and 1979 for the leukemia cases).

Case Finding

MDPH's computerized mortality file provided data on all deaths of Woburn residents from kidney cancer, liver cancer, or leukemia from 1969 – 1978. Rev. Young, Dr. Truman, and two other sources – major medical referral centers in Boston – supplied information on incident cases of childhood leukemia. Clinical and pathology records from the referral centers and local hospitals enabled us to verify each diagnosis and determine the date of diagnosis and cell type. Our search for additional cases at a referral center with a computerized tumor registry of cases dating back over 20 years was unrevealing.

After we completed the interviews, a local pathologist notified MDPH that he thought the annual number of urinary bladder cancer specimens had increased in recent years. A review of pathology records at the two local hospitals primarily used by Woburn residents identified 6 additional cases of renal cancer and 29 cases of bladder cancer.

Interviews

MDPH and CDC developed, pretested, and revised the study questionnaire, which included the following topics:

| | |
|---|---|
| <u>Demographic Information</u> Sex Place and date of birth Ethnic background Religious background | <u>Pregnancy History</u> * Father's military history Father's exposures prior to mother's case pregnancy Mother's occupational exposures during pregnancy |
| <u>Disease Process</u> Age and date of diagnosis Age and date of death Time of onset of symptoms Description of course of illness Treatment | <u>Family Background and Medical History</u> <u>Smoking History</u> |
| <u>Past Medical History</u> History and description of specific illnesses Diseases prior to onset Birth defects Medical procedures Immunizations Transfusions Tonsillectomy X-ray | <u>Residential History</u> <u>School History</u> * |
| <u>Immunization History</u> * | <u>Occupational History</u> |
| <u>Pregnancy History</u> Number pregnancies, deliveries, miscarriages Age of mother and father Birth order Mother's history of illness, personal activities, exposures during case pregnancy | <u>Occupational History of Family Members</u> <u>Environmental Exposures</u> Gardening Activities in Woburn lakes/streams Contact with farm animals Eating habits Activities near dump sites/open bodies of water in Woburn Hobbies Exposure to hair spray/hair dye Household pets Church and community activities Travel outside Woburn Military service Exposure to specific chemicals |

* Asked only of leukemia case and control respondents

BACKGROUND

Located 12 miles northwest of Boston, Woburn was a major leather processing and chemical production center in the 19th and early 20th centuries. Today it is both a residential community and an industrial center. The Aberjona River flows in a southerly direction through east Woburn.

Residents of Woburn and neighboring towns have complained about their water and unpleasant atmospheric odors for at least 100 years (1). Since the early 1970's, a large area in northeastern Woburn has been developed as a light industrial park. This area was used primarily for agriculture and cattle grazing until 1853, when a chemical company built a plant there to produce acids and other chemicals for the textile, leather, and paper industries. In 1899 the company acquired an adjacent plant to produce lead arsenite and lead arsenate, and until 1915 it was the leading U.S. producer of arsenical insecticides. The company produced chemicals through the 1920's, and animal glues until 1970 (2).

Although recent concern about the quality of Woburn's environment has focused on the northeastern section, there are many other potential sources of pollution in the town, dating back many years. Leather tanneries were prominent throughout the town in the 19th century. Flower growing has been another major industry, but not in recent years (3). It is unknown whether hazardous residues remain from these or other industries.

During the summer of 1979, nationwide publicity about the Love Canal toxic waste dump and extensive local publicity about possible environmental hazards in Woburn caused some local residents to wonder if cancer rates there were higher than average. A resident wrote to CDC asking if cancer rates were elevated in Woburn. Available data were insufficient to provide a definitive answer. In October a local clergyman, Rev. Bruce Young, reported to the Mayor of Woburn and to the press that he had identified 10 cases of childhood leukemia in one area of the town during the preceding 15 years. Further, the Boston pediatric hematologist mentioned previously, Dr. Truman, reported to CDC that he had seen six cases of acute lymphocytic leukemia in a 6-block area of Woburn since 1972. The National Institute for Occupational Safety and Health (NIOSH) learned of three living persons with kidney cancer among former workers of a pet food plant, now closed, in Woburn – an unusually high number among a workforce thought to be fewer than a thousand when the plant was in full operation.

Over the period 1969 – 78, there were statistically significantly more deaths than expected from all causes in Woburn. Although 2,738 deaths were expected, based on the statewide experience, 2,944 deaths actually occurred, an 8% excess $P < 0.01$, chi-square. Deaths were elevated by about the same amount for both sexes. Cancer mortality was significantly elevated in Woburn (13%) for the 1969 – 1978 period ($P < 0.01$, chi-square), with male and female patients experiencing a comparable excess of cancer deaths. In addition, for that period significantly more deaths than expected were reported from cancers of the liver, female organs other than the cervix uteri, and kidney.

METHODS

Statistical Methods

Various statistical distributions are used to determine the probability that a given observation could have occurred by chance. The distribution used in this report, unless otherwise noted, is the Poisson distribution. Two other common distributions, chi-square and binomial, are used where appropriate. The term “significant” is used here exclusively in the sense of statistical significance. Trained interviewers from CDC and NIOSH conducted the interviews the week of June 23, 1980. Interviews were conducted with one or both parents of the 12 persons with childhood leukemia. For each leukemia patient, two age- and sex-matched controls were interviewed: one control lived near the patient; the other, much farther away. An employee in the office of the Woburn Superintendent of Schools selected a list of potential controls from enrollment lists. We interviewed all 3 persons known to have kidney cancer, relatives of 17 deceased kidney cancer patients, and relatives of 5 deceased liver cancer patients. MDPH and CDC analyzed responses to all items on the study questionnaire, but only those which resulted in some specific association or which were related to the specific types of cancer in the study are discussed here. In the absence of cancer incidence data for Massachusetts, we used the Third National Cancer Survey (TNCS) age- and sex-specific incidence data to calculate the expected number of cases for each disease (4). Demographic data for Woburn were obtained from the 1970 U.S. census and MDPH’s official population estimates for 1975 (5). For geographical and residential analyses, we used Woburn census tract population data from the 1950, 1960, and 1970 U.S. censuses.

RESULTS

A. Leukemia

The most important findings concerning childhood leukemia in Woburn were that its incidence was significantly elevated, especially for male subjects, and that cases were concentrated in the eastern part of town.

Twelve cases of childhood leukemia occurred in Woburn from 1969 – 79. Nine (75%) of the children had acute lymphocytic leukemia, and all cases were diagnosed before the patients reached 15 years of age. Childhood leukemia incidence in Woburn in this period was significantly higher than expected: 12 observed, 5.3 expected ($p = 0.0058$) (Table 1). Boys had an elevated rate ($p = 0.0038$), and girls – overall - did not, although the girls' cases were all diagnosed when they were between ages 10 and 14, a significant elevation for that age group ($p=0.008$). The ratio of boys to girls was high, but not significantly so ($p > 0.1$, binomial distribution).

Table 1: Comparison of Observed Number with Expected [←] Number of Childhood Leukemia Cases, by Sex and Age Group, Woburn, 1969 – 1979

| Sex/Age Group | Woburn Population 1970 | Observed Number of Cases | Expected Number of Cases | Ratio Observed/Expected | Poisson Probability | |
|---------------|------------------------|--------------------------|--------------------------|-------------------------|---------------------|-------|
| Boys | < 1-4 | 1,784 | 4 | 1.4 | 2.8 | .054 |
| | 5-9 | 2,057 | 3 | 0.9 | 3.3 | .063 |
| | 10-14 | 2,128 | 2 | 0.7 | 2.8 | .156 |
| | All ages (< 1-14) | 5,969 | 9 | 3.1 | 2.9 | .0038 |
| Girls | < 1-4 | 1,714 | 0 | 1.3 | - | |
| | 5-9 | 1,982 | 0 | 0.5 | - | |
| | 10-14 | 2,083 | 3 | 0.4 | 7.5 | .008 |
| | All ages (< 1-14) | 5,779 | 3 | 2.2 | 1.4 | .38 |
| Both sexes | < 1-4 | 3,498 | 4 | 2.7 | 1.5 | |
| | 5-9 | 4,039 | 3 | 1.4 | 2.1 | |
| | 10-14 | 4,211 | 5 | 1.1 | 4.5 | |
| | All ages (< 1-14) | 11,748 | 12 | 5.3 | 2.3 | .0058 |

* Expected on the basis of Third National Cancer Survey, 1969 – 1971, whites, all areas combined

The dates of diagnoses were distributed uniformly over the 11-year period. Although 4 of the patients were born in the 6-month period between December 1963 and May 1964, this also was not significant; there was a 10% probability that at least 4 of the 12 patients would be born in some 6-month period (6).

At the time of diagnosis the leukemia cases were concentrated in the eastern part of Woburn, just north of Walker Pond. Figure 1 displays Woburn's six census tracts and the location of cases in each tract. Six patients lived within or on the border of census tract 3334, in an area approximately ½ mile in radius. There was a significant concentration of cases in this census tract; the probability that 6 or more of the 12 cases would occur in this area, which contains only 17% of the town's population in the 1- to 14-year age group, is less than 0.01 (binomial distribution). The six cases in this census tract were 7.5 times ($p = 0.00018$) higher than expected (Table 2). Childhood leukemia incidence for the rest of Woburn was not significantly elevated ($p = 0.297$).

Figure 1: Residences of Childhood Leukemia Patients at Time of Diagnosis, Woburn, Massachusetts, 1969 – 1979

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Table 2: Comparison of Observed Number with Expected^{*} Number of Childhood Leukemia Cases , by Category, Woburn, 1969 – 79

| Both Sexes Ages < 1-14 | Woburn Population 1970 | Observed Number of Cases | Expected Number of Cases | Ratio Observed to Expected | Poisson Probability |
|-----------------------------------|------------------------------|--------------------------------|--------------------------------|-------------------------------|------------------------|
| Census tract 3334 | 1,707 | 6 | 0.8 | 7.5 | 0.0002 |
| All Other Woburn Census tracts | 10,041 | 6 | 4.5 | 1.3 | 0.30 |
| Boys Ages < 1-14 | | | | | |
| Census tract 3334 | 884 | 5 | 0.4 | 12.5 | 6×10^{-5} |
| All other Woburn Census tracts | 5,085 | 4 | 2.6 | 1.5 | 0.24 |

* Expected on the basis of Third National Cancer Survey, 1969 – 71, whites, all areas combined

Residence and the Environment

East Woburn residents complained more often about the quality of the water, citing its bad odor, taste, and color, as well as how it corroded plumbing fixtures and dishwashers, and stained washed clothing. They also complained more about airborne odors.

School Histories

Children in the case and control groups who were of elementary school age attended schools near their homes. All five school-aged children with leukemia who lived near Walker Pond attended the same elementary school. In four instances, however, the leukemia had been diagnosed before the child entered school.

Family Medical History

There was little difference between family histories of children in the case and control groups. No one in the case group had a family history of leukemia.

Pregnancies and Miscarriages

The miscarriage rates for mothers in the case and control groups were similar. The two groups did not differ regarding mother's age at delivery, birth order of child, or mother's use of tobacco, alcohol, and drugs. A few mothers in each group had minor illnesses during pregnancy; none reported influenza. Two mothers in the case group, but none in the control group, received dental X-rays during pregnancy, and no member of either group was exposed to carcinogens in the workplace.

Church Membership

The majority of families in both groups were Roman Catholic. Seven Roman Catholic case families attended five different churches.

Other Findings

There were no significant differences between case and control groups with respect to medical histories, parents' occupations, or environmental exposures. No illnesses of pets were reported.

B. Kidney Cancer

The most important findings concerning kidney cancer were that the incidence was significantly elevated in male residents, and there was a concentration of cases just east and northeast of Horn Pond.

Thirty Woburn residents had diagnosed cases of cancer of the kidney or cancer of the ureter from 1969 – 78. The overall incidence was elevated in both sexes but significantly so only in male patients (Table 3) ($p = 0.021$). Thirteen cases were diagnosed from 1969 – 73 and 17 cases from 1974 – 78.

Table 3: Comparison of Observed Number with Expected* Number of Kidney Cancer Patients, by Sex, Woburn, 1969 – 1978

| Sex | Woburn Population 1970 | Number of Cases Observed | Number of Cases Expected | Observed/ Expected | Poisson Probability |
|-----------------|------------------------------|--------------------------------|--------------------------------|-----------------------|------------------------|
| Male Patients | 17,939 | 20 | 12.0 | 1.7 | .021 |
| Female Patients | 19,128 | 10 | 7.4 | 1.4 | .22 |
| Total | 37,067 | 30 | 19.4 | 1.5 | |

* Expected on the basis of Third National cancer Survey, 1969 – 1971, whites, all areas combined

Table 4 presents the age distributions by sex of the patients according to their age at diagnosis, and it compares them with the corresponding distributions of the TNCS. The number of Woburn men whose cases were diagnosed when they were aged 50 – 54 was significantly higher than expected (7 vs. 1.3, $p = 0.00043$). No significant elevations were found among individual age groups for female patients. The ratio of male to female patients was slightly higher than that found in the TNCS (1.67 vs. 1.35).

Table 4: Observed and Expected * Age and Sex Distribution for Kidney Cancer Patients

| Age at Diagnosis | <u>Male Patients</u> | | | | <u>Female Patients</u> | | | |
|----------------------------|----------------------|----------|-------|----------|------------------------|-----------|------|----------|
| | Woburn | | TNCS | | Woburn | | TNCS | |
| | Obs. | Com % | Exp. | Cum % | Obs. | Cum. % | Exp. | Cum % |
| 1-19 | 0 | 0 | 0.52 | 4.3 | 0 | 0 | .47 | 6.4 |
| 20-24 | 0 | 0 | 0.02 | 4.5 | 1 | 10 | .05 | 7.0 |
| 25-29 | 0 | 0 | 0.05 | 4.9 | 0 | 10 | .04 | 7.6 |
| 30-34 | 0 | 0 | 0.09 | 5.7 | 0 | 10 | .05 | 8.2 |
| 35-39 | 0 | 0 | 0.25 | 7.7 | 0 | 10 | .17 | 10.5 |
| 40-44 | 0 | 0 | 0.48 | 11.7 | 0 | 10 | .34 | 15.1 |
| 45-49 | 2 | 10 | 1.20 | 21.7 | 0 | 10 | .43 | 20.9 |
| 50-54 | 7 | 45 | 1.33 | 32.8 | 1 | 20 | .67 | 30.0 |
| 55-59 | 2 | 55 | 1.35 | 44.0 | 1 | 30 | .83 | 41.2 |
| 60-64 | 2 | 65 | 1.73 | 58.41 | 3 | 60 | .82 | 52.3 |
| 65-69 | 4 | 85 | 1.59 | 71.7 | 2 | 80 | .87 | 64.0 |
| 70-74 | 0 | 85 | 1.22 | 81.8 | 1 | 90 | .98 | 77.3 |
| 75-79 | 3 | 100 | 1.16 | 91.5 | 1 | 100 | .69 | 86.6 |
| 80-84 | 0 | 100 | 0.59 | 96.4 | 0 | 100 | .60 | 94.7 |
| 85+ | 0 | 100 | 0.43 | 100.0 | 0 | 100 | .39 | 100.0 |
| | | | | | | | | |
| Total | 20 | | 12.01 | | 10 | | 7.40 | |
| | | | | | | | | |
| Median age At diagnosis | | | 58.5 | | | | 62.5 | |

* Expected on the basis of Third National Cancer Survey, 1969 – 1971, whites, all areas combined

During the period 1969 through 1978, 24 cases of renal cell carcinoma, 5 cases of transitional cell carcinoma of the renal pelvis, and 1 case of transitional cell carcinoma of the ureter were diagnosed and ascertained by the procedures described above. For purposes of analysis these three categories of kidney cancer have been combined, as was done in the TNCS. Expected incidence has been computed from the combined incidence reported from the TNCS. The National Cancer Institute's Cancer Patient Survival Report (7) lists the distribution of kidney cancer by cell type in whites to be: carcinoma NOS (not otherwise specified), 5%; transitional cell, 19%; hypernephroma, 56%; adenocarcinoma NOS, 9% Wilms', 6%; all other, 4%. If we assume carcinoma NOS, hypernephroma, and adenocarcinoma NOS to be equivalent to renal cell carcinoma, then compared with the expected number calculated from the TNCS the ratio of renal

cell to transitional cell carcinoma is 3.7:1, and for the Woburn cases it is 4.8:1 – a small and insignificant difference.

Residence

No concentration of cases was apparent when patients' residences at time of diagnosis were mapped. Residences were mapped at 5-year intervals from 15 to 30 years before diagnosis, revealing a concentration of cases in the area just east and northeast of Horn Pond at 20 years before diagnosis (Fig.2). Of the 17 patients known to live in Woburn then, 7 lived in census tract 3333, near Horn Pond, where about 15% of the total Woburn population lived between 1950 and 1960. The 17 renal cancer cases were distributed according to the population distribution, and the probability of finding 7 or more cases in census tract 3333 is low ($p = 0.016$). Thus, an unusually large number of persons affected lived there.

Figure 2: Residences of Kidney Cancer Patients 20 Years before Diagnosis, Woburn, MA
1969-78

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Occupational Histories

Complete occupational histories were obtained for 20 of the patients by interview. Partial occupational histories were obtained from medical charts for the others.

Table 5 lists those patients with possible occupational exposure to known carcinogens or lead. Ten of the men for whom we had complete histories might have been exposed to known carcinogens, and all of them could possibly have been exposed to lead. Of the nine men with incomplete histories, two may have been exposed to known carcinogens, and four to lead. Women had fewer potential exposures in their workplaces. These frequencies are difficult to evaluate in the absence of occupational data from an appropriate comparison group or general information on the distribution of occupations in Woburn. Persons potentially exposed had been employed in these occupations about 20 years before diagnosis, an important period considering the suspected latent period of renal cancer. We mapped residences of patients unlikely to have had occupational exposure, but there was no apparent pattern.

Table 5: Occupations[←] of Kidney Cancer Patients with Potential Exposure to Lead and Known Carcinogens

| <u>Sex</u> | <u>Occupations with Potential Exposure to Lead</u> | <u>Occupations with Potential Exposure to Carcinogens</u> |
|------------|--|---|
| M | Bricklayer | Shipyard worker |
| | Electronics worker | |
| M | Printer | |
| M | Postal carrier | Furniture worker |
| | Foundry worker | |
| M | Leather worker | Leather worker |
| M | Motor vehicle inspector | Maintenance worker |
| | Leather worker | Leather worker |
| M | Leather worker | Leather worker |
| M | Gas station attendant | Welder |
| | Welder | |
| M | Leather worker | Maintenance worker |
| M | Gas station attendant | |
| M | Fishery (canning?) | Carpenter |
| | | Maintenance worker |
| M | Welder | Welder |
| M | Farm work/greenskeeper | |
| M | Farm work | |
| M | Leather worker | Leather worker |
| M | Painter | Painter |
| F | | Paper box manufacturing |
| F | | Glue machine operator |
| F | Farm work | |

Environmental Exposures

There were fewer complaints about environmental problems around the kidney cancer patients' homes than from the parents of the leukemia patients and controls. Roughly a third complained about the poor quality of the drinking water or about mosquito spraying in the neighborhood or a nearby industry of factory, in most cases a leather or tanning factory. The majority of families in the survey consumed home-grown vegetables and had a household pet. None had eaten fish caught in Woburn, nor had anyone frequented the dumpsites.

* National Institute for Occupational Safety and Health (NIOSH), CDC. To the Work-Relatedness of Disease, Revised Edition. Kusnetz S, Ed, 1979.

Other Factors

Smoking, past medical, and family medical histories revealed nothing remarkable.

C. Liver Cancer

Only 7 of the 10 reported fatal cases of liver cancer were confirmed to be primary hepatomas; thus, the incidence was not elevated (7 observed vs. 6.4 expected). Cases did not cluster by date of diagnosis or by patient's residence at diagnosis.

D. Bladder Cancer

The close functional relationship between the kidney and the bladder, and the suggestion by a local pathologist that bladder cancer may be elevated in Woburn, prompted an additional investigation of bladder cancer mortality and incidence.

Bladder cancer mortality was not elevated in Woburn in the 10-year period 1969 – 73 -- 13 cases observed, 14.5 cases expected -- nor for either of the two 5-year periods. Male subjects had fewer deaths than expected – 5 deaths observed and 9.6 deaths expected ($p=0.093$) – and female subjects more deaths than expected – 8 observed, 4.9 expected ($p = 0.13$). We identified 29 cases of bladder cancer diagnosed from 1969 through 1978 (Table 6), compared with 45.9 cases expected ($p = 0.0073$). There were no male patients aged 75 or older; 14.4 were expected ($p = 1 \times 10^{-6}$). Cases did not cluster by patients' residences or dates of diagnosis.

Table 6: Comparison of Observed with Expected * Number of Urinary Bladder Cancers, by Sex, Woburn, 1969 – 1978.

| Sex | Woburn Population 1970 | Number of Cases Observed | Number of Cases Expected | Observed/ Expected | Poisson Probability |
|-----------------|------------------------------|--------------------------------|--------------------------------|-----------------------|------------------------|
| Male Patients | 17,939 | 20 | 33.3 | 0.6 | .0087 |
| Female Patients | 19,128 | 9 | 12.6 | 0.71 | .195 |
| Total | 37,067 | 29 | 45.9 | 0.63 | .0073 |

Expected on the basis of Third National Cancer Survey, 1969 – 71, whites, all areas combined

ENVIRONMENTAL DATA

At the start of this investigation, much attention was focused on hazardous waste sites in Woburn. In July 1979, Environmental Protection Agency (EPA) field investigators discovered an abandoned lagoon, covering 0.8 of an acre and up to 5 feet deep, contaminated with lead and arsenic, the latter in concentrations as high as 1,050 parts per million (2). Although we do not know for certain, the arsenic was probably deposited between 1899 and 1915. Engineers demonstrated that arsenic had leached into a nearby pond. Other wastes in nearby areas are shown in Table 7. Besides these chemicals, pits of buried animal hides and slaughterhouse wastes were discovered during site preparation operations for the industrial park, and these are believed to be responsible for much of the foul odor residents frequently complained about.

Table 7: Types and Concentrations of Metals Found in Industri-Plex Site¹

| Metal | <u>Samples (ppm)</u> | | | | Normal Soil Contrations ² |
|-----------|----------------------|---------|---------|--------|--------------------------------------|
| | A | B | C | D | |
| Iron | 257,000 | 42,900 | 418,600 | 5,800 | 15,000 |
| Manganese | 15,800 | 169 | 119 | 17 | 285 |
| Copper | 84 | 32 | 1,690 | 38 | 14 |
| Lead | 287 | 99 | 229 | 811 | 14 |
| Zinc | 227 | 129 | 1,990 | 57 | 36 |
| Chromium | 386 | 33 | 7 | 36 | 36 |
| Cadmium | 0 | 11 | 7 | 0 | - |
| Nickel | 339 | 21 | 14 | 14 | 13 |
| Tin | 920 | 1,990 | 1,590 | 255 | - |
| Calcium | 19,800 | 199,800 | 299 | 2,070 | 3,200 |
| Magnesium | 1,380 | 2,490 | 100 | 151 | - |
| Sodium | 25,700 | 14,980 | 428 | 255 | 2,600 |
| Aluminum | 85,000 | 55,900 | 830 | 26,400 | 33,000 |
| Arsenic | 110 | 200 | 1,050 | 880 | 6 |

¹ Soil samples taken 9/24/79 at 128 Commerce Way, Woburn.

² Department of Environmental Quality Engineering. Special Analysis—Samples of Waste Deposits—D’Annolfo’s Commerce Way, Woburn. Contained in memo from Robert Cleary, DEQE, to Richard Chalpin, DEQE, dated October 24, 1979.

Woburn's water supply has been tested several times for contamination. Wells G and H (Fig. 3) proved to contain pollutants. All other Woburn wells have consistently met the interim Federal drinking water standards (2). Well G began to pump in 1964, was on line until early 1967, and from then to May 1979 was on and off, depending on Woburn's water needs. Well H, which started pumping in July 1967, was shut down from December 1967 until August 1974, and then used intermittently as needed. In May 1979 both G and H were permanently shut down when organic contaminants were discovered (Table 8). Although these wells were originally planned as supplemental sources of Woburn's water needs, a considerable volume of water was pumped from them when they were on line. Some of the water from G and H reached most of Woburn, but most went to eastern Woburn (Fig. 3). In 1979 wells G and H had no detectable chromium and lead, and very low levels of arsenic (Table 8), in conformance with interim Federal drinking water standards.

Figure 3. Location of Wells and Area Serviced by wells G and H, Woburn, Massachusetts 1979

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Table 8: Contaminants in Wells G & H, Woburn, Massachusetts, 1979

| <u>Organics</u> Department of Environmental Quality Engineering ¹ | <u>Wells G</u> | | | <u>Wells H</u> | | |
|--|-------------------|-------------------------|-------------|-----------------|-------------|-------------|
| | <u>5/14</u> | <u>7/24</u> | <u>9/24</u> | <u>5/14</u> | <u>7/24</u> | <u>9/24</u> |
| Chloroform | 11.8 ² | - | 1.1 | ND ³ | - | ND |
| Trichloroethylene | 267.4 | - | 117.6 | 183.6 | - | 63.0 |
| Tetrachloroethylene | 20.8 | - | 18.3 | 13.4 | - | 9.0 |
| 1,1,1-tetrachloroethane | 0.6 | - | ND | ND | - | 2.1 |
| Dibromochloromethane | 2.0 | - | ND | ND | - | ND |
| <u>Environmental Protection Agency⁴</u> | | | | | | |
| Trichlorotrifluoroethane (Freon) | | 22 ppb | | | 23 ppb | |
| Dichloroethylene | | 28 ppb | | | ND | |
| Dichlorotrifluoroethane | | 5 ppb | | | ND | |
| <u>Metals</u> | | | | | | |
| Arsenic | | 0.0020 ppm ⁵ | | | 0.0015 ppm | |
| Chromium | | ND | | | ND | |
| Lead | | ND | | | ND | |

1. Special Analysis – Woburn. Water samples taken from wells G and H by McCall, DEQE (5/14/79), and Erickson, DEQE (9/24/79)
2. Micrograms per liter or parts per billion (ppb)
3. ND – None Detectable
4. Analysis for Volatile Organics, on Woburn wells G and H. Results contained in memorandum from R. Siscanaw, EPA, to E. Taylor, EPA
5. Milligrams per liter or parts per million (ppm)

We have no information indicating types and levels of contaminants, if any, in wells G and H before May 1979. Recent static testing of well G for the 129 chemicals on EPA's priority pollutant list revealed no new contaminants but did confirm the presence at similar concentrations

of the organic chemicals found previously. Extensive testing of all other Woburn wells showed the water meets State and Federal drinking water standards.

Air Quality Data

Data on air quality are limited. A study in 1977 measured hydrogen sulfide and sulfur dioxide concentrations at various locations within and near the Industri-Plex site in northeast Woburn and in nearby Reading. Areas downwind of the construction site experienced hydrogen sulfide concentrations 10 to 100 times the odor threshold, levels which may induce headaches, bronchitis, nervous system disorders, and eye irritation in susceptible individuals (8).

DISCUSSION

A. Leukemia

During the years 1969 – 79, the incidence of childhood leukemia in Woburn was statistically elevated. For male subjects, the incidence in eastern Woburn was 12.5 times that expected from TNCS incidence data. The male-to-female ratio was three, slightly more than double the 1.4 ratio of TNCS whites aged < 1 – 14. The age distribution among female subjects was unusual in that all three patients were between ages 10 and 14 at diagnosis. Excess acute lymphocytic leukemia has occurred among cohorts of children born after influenza epidemics (9). Four leukemia patients were born in a 6-month period, but the mothers of these children did not report having had preceding influenza.

Several other investigators of possible leukemia clusters have found that cases have occurred predominantly in Catholic families and members of the same parish or church (10). We found no such association.

The case-control study failed to identify any factor that significantly distinguished the case from the control group. This is not altogether surprising, for with few exceptions, investigations of leukemia clusters have failed to demonstrate significant associations or even promising leads as to possible environmental causes (10,11). Statistical tests in such small populations are of little value.

Approximately 3 miles north of the concentration of leukemia cases is the Industri-Plex site, in which, as described above, hazardous waste dumps containing such toxic heavy metals as lead, arsenic, and chromium recently have been uncovered. The Aberjona River flows through

this site, and its watershed flows south through the area where the leukemia patients reside. The wastes have been there for a number of years, but these heavy metals are insoluble in water and are therefore not easily transported. In fact, wells G and H, located about ½ mile north of the area where the leukemia cases were concentrated, had no detectable lead or chromium, and arsenic only at levels considerably below the official drinking water standard. Chromium and arsenic are known carcinogens, but they are not leukemogenic (12, 13). Although lead affects the kidneys and central nervous system as well as other organs, it does not cause leukemia (14). Further, the leukemia patients and their immediate families had little, if any, physical contact with the site. Four of the six cases in the area of concentration were diagnosed before the patients reached age 7, and none of the children had played near or on the dump site.

Possibly of more relevance to the leukemia concentration is the contamination of wells G and H. Well G was on line for sometime before most of the leukemia cases were diagnosed. As far as we have been able to determine, environmental data before 1979 do not exist to indicate what, if any, contaminants were in wells G and H. Residents of eastern Woburn have complained of poor water quality since the 1960s. These complaints, however, cannot be correlated with the presence of specific contaminants. None of the chemicals found in wells G and H are known to cause leukemia, although chloroform (15), trichloroethylene (16), and tetrachloroethylene (17) have caused tumors in experimental (laboratory animal) investigations. Even if a suspected leukemogen such as benzene (18, 19) were found now in the wells, it would still be necessary to establish that it had been present and that the patients had been exposed to it sometime before diagnosis. The lack of environmental data for earlier periods is a major obstacle in establishing a link between specific environmental contaminants and the occurrence of leukemia in Woburn. Although the contaminants found in wells G and H are not known to cause leukemia, the fact that organic contaminants were found in the water supply must be emphasized. The source of the present contaminants is unknown.

B. Kidney Cancer

The incidence of kidney cancer was higher than expected in both sexes, but significantly so only in male subjects, particularly those younger than 55 (9 observed, 2.5 expected, $p = 0.001$). Male patients were younger at diagnosis than female patients, and younger than both male and female patients in TNCS. In a Connecticut study of renal cancer, a birth cohort effect was found

for men only with age-specific incidence rates increasing as the year of birth became more recent (20). The observed age distribution in Woburn suggests that such may have also been the case here, although demonstration of such an effect would require data from earlier periods. No clear pattern emerged from the interviews of patients or their next of kin.

In this investigation many usual occupations involved some potential lead exposure – interesting because lead workers have been found to have impaired renal function (21), and because lead ingestion produces kidney cancer in experimental animals (22). Interest in kidney cancer in Woburn arose partly from a report that a higher than expected number of kidney cancer cases had occurred among former workers of a pet food processing plant near a dump site. No one in our case group had worked in this plant.

Seven kidney cancer patients had lived 20 years before the cancer was diagnosed in the area east and northeast of Horn Pond – a significantly greater proportion than expected from the distribution of the city’s population. No environmental or historical data exist to explain what hazards, if any, were associated with this area.

C. Liver Cancer

The higher than expected mortality rate for liver cancer turned out to be an artifact, because several deaths from other causes had been erroneously coded in the medical records as primary liver cancer.

D. Urinary Bladder Cancer

We examined bladder cancer incidence because a local pathologist reported a recent increase and because the urinary bladder is related functionally to the kidney and ureter. The incidence has increased from 10 cases diagnosed in 1969 – 73 to 19 cases in 1974 – 78. Despite this increase, the observed incidence of bladder cancer was lower than expected from the TNCS incidence data. Further, the age-specific rates for persons aged 45 – 74 were very close to those observed in eastern Massachusetts by Cole and his associates (23). The observed number of cases was lower than expected in Woburn because no cases of bladder cancer were observed in men older than 74, the group in which high rates are usually found. Although in one study (24) Cole estimated the risk of bladder cancer in male leather workers to be over double that expected, and

five of the bladder cancer patients worked in the leather industry, we lack the necessary information to determine if this is higher than expected.

E. Environmental Data

Even though a great deal of environmental testing has been performed in Woburn over the past 2 years, two facts point out the limited usefulness of this information in assessing the health effects of environmental hazards in Woburn:

1. Until recently, most attention and environmental sampling has focused on the Industri-Plex area of Woburn where major dump sites of toxic waste products have been uncovered. Only recently has much attention been paid to the area south of Route 128 and, in particular, the area where leukemia cases were concentrated; even less environmental data are available for other parts of Woburn (for example, the area around Horn Pond).
2. The collected environmental data reflect current types and levels of contaminants. The critical exposure period for the cancers studied, however, is likely to be a number of years before diagnosis. In the case of kidney cancer, environmental exposures 15 – 30 years before onset are likely to be most relevant in assessing the possible association between environmental factors and disease. Determination of exposures in the 1960s and early 1970s would be most helpful in the investigation of childhood leukemia.

CONCLUSIONS AND RECOMMENDATIONS

This investigation has established that from 1969 through 1979 the overall incidence of childhood leukemia was significantly elevated in Woburn, particularly in eastern Woburn. The incidence of kidney cancer was also significantly elevated in that period.

Information gathered thus far fails to establish any association between environmental hazards and the increased incidence of childhood leukemia and renal cancer in Woburn. The hypothesis that the increase in leukemia incidence was associated with environmental hazards, and specifically with the contamination of drinking water supplies, is neither supported nor refuted by the study findings. Interviews with parents of leukemia patients, two groups of matched controls,

and family members of renal cancer patients revealed no associations between any environmental factors and disease. In the future, investigators of this possible association should:

- Attempt to ascertain childhood leukemia mortality and incidence in Woburn before 1969, that is, in the 1950s and 1960s. If the incidence of childhood leukemia became elevated in eastern Woburn only after wells G and H came on line, then this would support a relationship between childhood leukemia and contaminated drinking water.
- Determine the incidence of other lymphatic cancers in Woburn to see if they follow a pattern like that of childhood leukemia over the same period.
- Determine the incidence of other types of cancer in Woburn before 1969.
- Continue to attempt to identify the present source of organic contaminants in wells G and H. This information may help identify past practices of waste disposal and sources of contaminants.

Use of trade names is for identifying only and does not constitute endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Please note that the following figures from this report could not be made available via the Internet:

- 1.) Figure 1: Residences of Childhood Leukemia Patients at Time of Diagnosis, Woburn, MA, 1969 - 1979
- 2.) Figure 2: Residences of Kidney Cancer Patients 20 Years before Diagnosis, Woburn, MA, 1969-78
- 3.) Figure 3: Location of Wells and Area Serviced by Wells G&H, Woburn, MA 1979

If you are interested in obtaining copies of these figures please contact us at 617/624-5757. Thank you for your cooperation.

REFERENCES

1. Massachusetts State Board of Health. Seventh annual report, 1876;242.
2. Memoranda and affidavits of the Environmental Protection Agency and the Massachusetts Department of Environmental Quality Engineering, reported in: plan for investigation of hazardous waste problems: Woburn, Massachusetts area. New York: Fred C. Hart Associates, Inc. 1980.
3. Woburn Atlas 1876 and 1926. City Engineer's Office, Woburn, MA.
4. Cutler SJ, Young JL, Jr. Third national cancer survey: incidence data. NCI Monograph 41. DHEW publication no. (NIH) 75-787.
5. Office of State Health Planning. Massachusetts city/town population estimates, 1975.
6. Wallenstein S. A test for detection of clustering over time. *Am J Epidemiol*, 1980;111:367-72.
7. Axtell LM, Aisre AJ, Myers MH, eds. Cancer patient survival report number 5, DHEW publication no. (NIH) 77-992.
8. Bolt, Beranek and Newman, Inc. Evaluation of hydrogen sulfide concentrations at residential and commercial sites surrounding a Woburn construction area, August 1977.
9. Austin DF, Karp S, Divorsky R, Henderson BD. Excess leukemia in cohorts of children born following influenza epidemics. *Am J Epidemiol* 1975;101:77-83.
10. Heath CW. The epidemiology of leukemia. In: D Schottenfeld, ed. *Cancer Epidemiology and Prevention*. Springfield IL: Charles C. Thomas, 1974:318-50.
11. Smith PG. Current assessment of "case clustering" of lymphomas and leukemias. *Cancer* 1978;42:1026-34.
12. Davies M. Lung cancer mortality of workers making chrome pigments. *Lancet* 1978;1:384.
13. Fraumeni JF, Jr. Respiratory carcinogenesis: an epidemiological appraisal. *JNCI* 1975;55:1039.
14. Cramer K, Goyer RA, Jagenburg R, Wilson MH. Renal ultrastructure, renal function, and parameters of lead toxicity in workers with different periods of lead exposure. *Br J Ind Med* 1974;31:113-27.
15. Page NP, Saffiotti U. Report on carcinogenesis bioassay of chloroform. Bethesda, MD: US NCI 1976.

16. National Cancer Institute. Carcinogenesis bioassay of trichloroethylene. CAS No. 79-01-6, NCI-CG-TR-2, 1976.
17. National Cancer Institute. Bioassay of tetrachloroethylene for possible carcinogenesis. DHEW Publication No. (NIH) 77-813, October 1977.
18. Vigliani EC, Forni A. Benzene and leukemia. Environ Res 1976;11:122.
19. U.S. Dept. of Commerce, NITS. Assessment of industrial hazardous waste practices: leather tanning and finishing industry 1976.
20. Finger-Kantor AL, Meigs JW, Heston JF, Flannery JT. Epidemiology of renal cell carcinoma in Connecticut, 1935-1973. JNCI 1976;57:495-500.
21. Baker EL, Landrigan PJ, Barbour AG, Cox DH, Folland DS, Ligo RN, Throckmorton J. Occupational lead poisoning in the United States: clinical and biochemical findings related to blood lead levels. Br J Ind Med 1979;36:314-22.
22. Van Esch GJ, Kroes R. The induction of renal tumors by feeding basic lead acetate to mice and hamsters. Br J Cancer 1969;23:765-71.
23. Cole P, Monson RR, Haning H, Friedell GH. Smoking and cancer of the lower urinary tract. N Engl J Med 1971;284:129-34.
24. Cole P, Hoover, R Friedell GH. Occupation and cancer of the lower urinary tract. Cancer 1972;29:1250-60.

John L. Cutler, M.D., Ph.D.
 Staff Epidemiologist
 Cancer Branch
 Chronic Disease Division
 Center for Environmental Health

Glyn G. Caldwell, M.D.
 Chief, Cancer Branch
 Chronic Diseases Division
 Center for Environmental Health

Joan French, Ph.D.
 Chronic Diseases Division
 Center for Environmental Health

Clark W. Heath, Jr., M.D.
 Director
 Chronic Diseases Division
 Center for Environmental Health

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William R. Adams, Jr., Environmental Protection Agency, Region I, John F. Kennedy Federal Building Room 2203, Boston, MA 02203

Wes Straub, Regional Consultant, NIOSH, Region I, Government Center, John F. Kennedy Federal Building, Boston, MA 02203

Donald E. MacKenzie, Regional Administrator, U.S. Department of Labor, OSHA, 16-18 North Street, 1 Dock Square, Boston, MA 02109

Milton Kotelchuck, Ph.D., Division of Health Statistics & Research, Massachusetts Department of Public Health, 600 Washington Street, Boston, MA 02111

Sharon Rosen, Ph.D, Massachusetts Department of Public Health, 600 Washington Street, Boston, MA 02111

Jerry Parker, Massachusetts Department of Public Health, 600 Washington Street, Room 770, Boston, MA 02111

George W. Evans, M.D., Winchester Hospital, Winchester, MA 01890

James E. Grassi, M.D., Choate Memorial Hospital, Woburn, MA 01801

John T. Truman, M.D., Massachusetts General Hospital, Boston, MA 02114

Robina Folland, Director, Office of Cancer Management, Tufts New England Medical Center, 171 Harrison Avenue, Boston, MA 02111

Roberto Mercado, M.D., Winchester Hospital, Winchester, MA 01890

Medical Record Room, Choate Memorial Hospital, 21 Warren Avenue, Woburn, MA 01801

Medical Record Room, Lawrence Memorial Hospital, 170 Governors Avenue, Medford, MA 02155

Medical Record Room, New England Rehabilitation Hospital, Rehabilitation Way, Woburn, MA 01801

Medical Record Room, Santa Maria Hospital, 799 Concord Avenue, Cambridge, MA 02140

Medical Record Room, Winchester Hospital, 41 Highland Avenue, Winchester, MA 01890

Medical Record Room, U.S. Government Veterans Administration Hospital, 150 S. Huntington Avenue, Boston, MA 02130

Medical Record Room, U.S. Government Veterans Administration Hospital, 1400 VFW Parkway, W. Roxbury, MA 02132