Childhood Leukemia in Woburn, Massachusetts

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Synopsis

Possible associations between environmental hazards and the occurrence of childhood leukemia were investigated in Woburn, MA, for the period 1969–79. Residents of Woburn were concerned over what they perceived to be a large number of childhood leukemia cases; at the same time there was extensive publicity about uncontrolled hazardous waste sites in Woburn, which resulted in its being placed on the Superfund list. Many believed that the elevated rate of childhood leukemia was related to these sites or to two city water wells that had been closed in 1979 when they were found to be contaminated by organic chemicals.

An occurrence was defined as childhood leukemia when it was diagnosed in a Woburn resident less than 20 years old between 1969 and 1979 and confirmed by review of hospital and pathology records. This investigation confirmed an increase in incidence which was distributed uniformly over the 11-year period. Six of the persons with leukemia were located close to each other in one census tract, 7.5 times the expected number. Parents of the children and of two matched control groups were interviewed about medical history, mother's pregnancy history, school history, and environmental exposures. There were no significant differences between the leukemia victims and persons in the control groups. No leukemia sufferer had contact with a hazardous waste site. While the contaminants of Wells G and H, which had been closed, are not known leukemogens, it is not possible to rule out exposure to this water as a factor, particularly in the eastern Woburn residents.

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.

Since the early 1970s, 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 1920s and animal glue until 1970 (*1*).

Although recent concern about the quality of Woburn's environment has focused on its northeastern section, other potential sources of pollution date back many years in the town. Leather tanneries were an important industry throughout

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Methods

An occurrence was defined as confirmed childhood leukemia, diagnosed between 1969 and 1979 and confirmed by review of hospital and pathology records, in a Woburn resident under 20 years of age.

The computerized mortality file of the Massachusetts Department of Public Health (MDPH) provided data on all leukemia deaths of Woburn residents from 1969 through 1978. Major medical referral centers in Boston supplied information about living and deceased persons with childhood leukemia. Because of Woburn's proximity to Boston, it is unlikely that cases were referred to other centers. Clinical and pathology records from the referral centers and local hospitals enabled us to verify each diagnosis and to determine the date of diagnosis and the cell type.

We used a pretested questionnaire to interview the parents of 12 children about past medical history, mother's pregnancy history, school history, and environmental exposures. For each leukemia patient, we interviewed the parents of two exact age- and sex-matched well controls—one who lived near the patient; the other, in the distal half of the city. The two control groups were selected so that geographic factors could be elucidated. They were analyzed separately. An employee in the office of the Woburn Superintendent of Schools selected a list of potential controls from elementary school enrollment lists. We analyzed responses to all items on the study questionnaire, but we discuss in this paper only those which suggested specific associations. In the absence of cancer incidence data for Massachusetts, we used the Third National Cancer Survey race-, age-, and sex-specific incidence data to calculate the expected number of leukemia cases (*2*). Demographic data for Woburn were obtained from the 1970 U.S. census and MDPH's official population estimate for 1975.

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 exclusively in the sense of statistical significance ($P < .05$).

Results

There were 12 cases of childhood leukemia in Woburn, 1969-79. Nine (75 percent) of the children had acute lymphocytic leukemia, and all 12 were less than 15 years of age at diagnosis. Childhood leukemia incidence in Woburn in this period was significantly higher than expected: 12 cases observed, 5.2 expected ($P = 0.007$) table 1. Boys had an elevated rate ($P = 0.0038$), and girls did not, although 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 ($P = 0.081$, binomial distribution). The dates of diagnosis were distributed uniformly over the 11-year period ($P = 0.398$ for five cases diagnosed before May 1974, binomial distribution) table 2. However, four of the patients were born between December 1963 and May 1964 which was statistically ($P = 0.037$, scan statistic) significant (*3*).

When analyzed by residence, the leukemia children at the time of diagnosis were concentrated in the eastern part of Woburn, near Walker Pond. The map shows Woburn's six census tracts and the location of cases in each tract. Six patients lived in or on the border of census tract 3334 within a radius of approximately 1/2 mile. 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 percent of the town's population in the infant- to 14-year age group, is less than 0.01 (binomial distribution). The six cases in this census tract were 7½ times ($P=0.0002$) higher than expected. Childhood leukemia incidence for the rest of Woburn was not significantly elevated ($P=0.30$).

East Woburn residents (both case families and control families) complained more often than other residents about the quality of the water, citing its bad odor, taste, and color; how it corroded plumbing fixtures and dishwashers; and how it stained laundered clothing.

Children in the case and control groups who were old enough attended elementary schools near their homes. Five attended one elementary school. In four instances, however, the leukemia had been diagnosed before the child entered school.

There were no significant differences between the control groups, and they were pooled for comparisons with the study group. Family histories of children with leukemia did not differ from control children. None of the leukemic children had a family history of leukemia.

The frequency of miscarriages for mothers in the case and control groups was not significantly different statistically. The two groups did not differ regarding mother's drug usage. A few mothers in each group had had minor illnesses during pregnancy; none reported influenza. Two mothers in the case group, but none in the control groups, received dental X-rays during pregnancy, and no parent of either group was exposed to carcinogens in the workplace.

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

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

Environmental Data

At the start of this investigation, hazardous waste sites in Woburn received the most attention. In July 1979, Environmental Protection Agency (EPA) field investigators discovered an abandoned lagoon, up to 5 feet deep, covering 0.8 of an acre, contamined with lead and arsenic, the latter in concentrations as high as 1,050 parts per million (*1*). 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. Contaminants in this pond have not entered the drinking water. Besides these chemicals, pits of buried animal hides and slaughterhouse wastes were discovered to be responsible for much of the foul odor residents frequently complained about.

Table 1. Comparison of observed number with expected number of childhood leukemia cases by sex and age group, Woburn, MA, 1969–79

Sex and age group (years)	Woburn's 1970 population	Number of cases		Ratio of observed to expected	Poisson probability
		Observed	Expected [1]		
Boys:					
Under 1–14...	1,784	4	1.4	2.9	.054
5–9.....	2,057	3	0.9	3.3	.063
10–14.....	2,128	2	0.7	2.9	.156
Girls:					
Under 1–14...	1,714	0	1.3	...	...
5–9.....	1,982	0	0.5	...	...
10–14.....	2,083	3	0.4	7.5	.008
Total:					
Under 1–14...	11,748	12	5.2	2.3	.007

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

Table 2. Cases of childhood leukemia by date of diagnosis, Woburn, MA, January 1969 to August 1979

Sex	Date of diagnosis
F.....	[1] Mar. 15, 1969
M.....	[1] Nov. 18, 1969
M.....	[1] July 12, 1971
M.....	[1,2] Jan. 31, 1972
M.....	[1,2] June 27, 1973
M.....	July 23, 1974
M.....	Feb. 20, 1975
F.....	Dec. 8, 1975
F.....	Aug. 9, 1976
M.....	Oct. 13, 1976
M.....	Mar. 31, 1978
M.....	[1] Aug. 20, 1979

[1] Case near Walker Pond.
[2] Case in child less than 4 years old.

Woburn's water supply has been tested several times for contamination. Wells G and H, near Walker Pond, proved to contain chloroform, trichloroethylene, and tetrachloroethylene. All other Woburn wells have consistently met the interim Federal drinking water standards (*1*). Well G began to pump on October 1, 1964, was on line until early 1967, and from then until 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, when organic contaminants were discovered, both G and H wells were perma-

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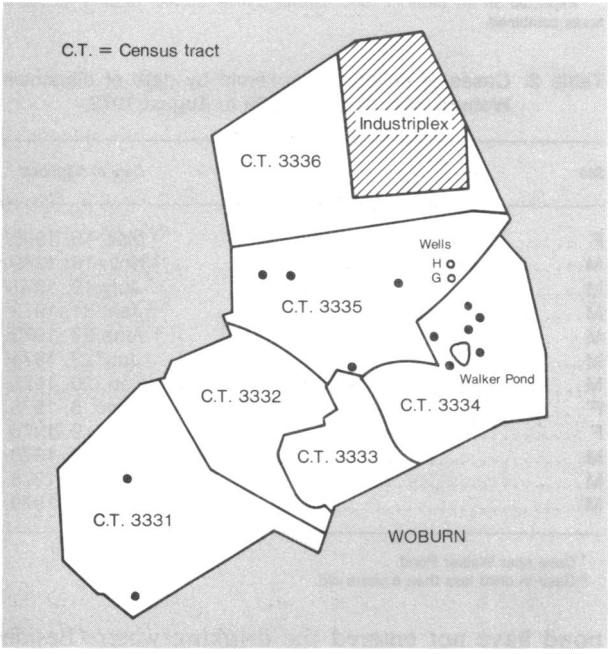
Residences of childhood leukemia patients at time of diagnosis
Woburn, Massachusetts 1969–79



nently shut down. Water from wells G and H was principally distributed to eastern Woburn. 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 pollutants 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 that the water met State and Federal drinking water standards. There are no private drinking water wells in Woburn. Walker Pond has never been a source of drinking water, and its principal recreational use is ice skating.

Data on air quality are limited. A study in 1977 measured hydrogen sulfide and sulfur dioxide concentrations at various locations within and near the industrial 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.

Discussion

Excess cases of acute lymphocytic leukemia occurred among cohorts of children born in a 6-month period, but the mothers of these children did not report having had influenza any time during pregnancy (*4*). Several other investigators of possible leukemia clusters have found that cases have occurred predominantly in Catholic families and in members of the same parish or church (*5*). We found no such association.

In this case-control study, we identified no factor that significantly distinguished the cases from the controls. This is not altogether surprising, because—with few exceptions—investigations of leukemia clusters have failed to demonstrate significant associations or even promising leads as to possible environmental causes (*5*, *6*). Statistical tests in such small populations have little statistical power.

The contamination of wells G and H is possibly relevant to the leukemia cluster near Walker Pond. Well G was on line for some time before most of the leukemia cases were diagnosed. Although none of the chemicals found in wells G and H are known to cause leukemia, chloroform (*7*), trichloroethylene (*8*), and tetrachloroethylene (*9*) have caused tumors of other sorts in experimental (laboratory animal) investigations. If a suspected leukemogen such as benzene (*10*) 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.

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Risk of Acute Respiratory Disease among Pregnant Women During Influenza A Epidemics

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The paper was presented at the 110th annual meeting of the American Public Health Association in Montreal, Canada, November 14-18, 1982. It was based on a study supported by contract number 200-79-0965 with the Centers for Disease Control.

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Synopsis

The medical literature contains little information on the occurrence of excess morbidity among pregnant women during recent influenza epidemics.

Rates of medical visits for acute respiratory disease (ARD) among pregnant and nonpregnant members of a large prepaid practice population were examined. Use of medical services for ARD was ascertained for approximately 1,000 pregnant women and 3,000 nonpregnant women during each of four epidemic periods (1975, 1976, 1978, 1979) and a nonepidemic period (1977). Comparing the combined epidemic periods with the nonepidemic period, there were significant excesses of 23.7 (standard error (SE) = 8.1) ARD contacts per 1,000 attributable to epidemic influenza for pregnant women and 10.2 (SE = 3.4) for nonpregnant women. ARD hospitalization rates among pregnant women were low (2 per 1,000), and there were no maternal deaths.

The significant ARD excess among pregnant women was concentrated in the 1978 period with reappearance of the A/Russia H1N1 subtype in the community and was confined to those under age 25 who would not have been previously exposed to this subtype (94.4 (SE = 28.5)). These findings indicate that recent influenza epidemics caused only modest excess ARD morbidity among pregnant women, and significant excess occurred only in association with antigenic shift. These findings support current national policy recommendations with respect to influenza vaccination of pregnant women.

CONVINCING EVIDENCE EXISTS THAT PREGNANT women experienced abnormally high mortality during the 1918-19 Spanish influenza pandemic (*1-4*). Subsequent epidemics have not generally been associated with excess maternal mortality, although several investigators have suggested that significant