# DRINKING WATER CONTAMINATION AND THE INCIDENCE OF LEUKEMIA:

AN ECOLOGIC STUDY

# By

Jerald Fagliano, M.P.H., Michael Berry, M.P.H, Frank Bove, Sc.D., and Thomas Burke, Ph.D., M.P.H.

Division of Occupational and Environmental Health New Jersey Department of Health

.

December 1987

#### ABSTRACT

The New Jersey Department of Health conducted an epidemiologic study of an ecologic design to begin its assessment of the possible relationship between the incidence of leukemias and the occurrence of volatile organic chemical (VOC) contamination of drinking water supplies. A study area was selected comprised of subpopulations differentially exposed to drinking water VOCs (primarily trichloroethylene, tetrachloroethylene, and related solvents) and/or trihalomethanes (THMs). Populations served by community water supplies were classified into exposure categories according to VOC contamination status based on 1984-85 sampling data and historical information. Six years of incidence data (1979-1984) on leukemias were collected from the New Jersey Cancer Registry. For females, the standardized incidence ratio (SIR) in towns in the highest of three exposure categories was elevated with statistical significance (SIR = 1.53, 95% C.I. 1.02-2.21) using 1982 New Jersey rates as the standard. SIRs for females were not statistically elevated in areas classified as uncontaminated or slightly contaminated with VOCs. No association was observed in males in any of the exposure categories. When populations were aggregated according to THM status, no associations were observed. A Poisson regression analysis of the data, using finer exposure strata, revealed the same results. Additionally, the regression demonstrated that the increase in rate was distributed evenly across all age strata. The rate ratio for females at the highest exposure stratum for total non-THM VOCs compared to the least exposed stratum was RR = 1.65. The observed association suggests that drinking water contaminated with VOCs may increase the incidence of leukemia among exposed females, but limitations inherent in the ecologic study design preclude causal inference. Further epidemiologic study of a case-referent design is warranted.

## I. INTRODUCTION

Since the middle 1970s, there has been considerable epidemiologic interest in the relationship between organic contaminants in drinking water and increased cancer incidence in exposed populations. The focus of the several studies has been on chlorinated compounds that can form in relatively high concentrations following chlorination of surface water for disinfection. These compounds, some carcinogenic or mutagenic, include the volatile trihalomethanes (THMs) and many nonvolatile substances. There is mounting evidence that consumption of chlorinated surface water is related to an increased risk of bladder and possibly colorectal cancers. These studies have been thoroughly reviewed (1 - 7).

In recent years, numerous groundwater supplies throughout the nation have been found to contain volatile organic compounds (VOCs) other than THMs. Groundwater was long thought to be relatively protected from such contamination, but national and state surveys have demonstrated the vulnerability of this water resource (8-9). In this decade, efforts have begun in many states to assess the extent and magnitude of VOC contamination of groundwater supplies. As more contamination is found, interest is growing in the potential health risks to exposed populations.

In New Jersey, routine semi-annual testing for 14 VOCs has been required of public community water systems since late 1984 under amendments to the state Safe Drinking Water Act (10). A list of these VOCs can be found in Table 1. While most of the over 600 community systems have been found to be free of the additional contaminants, about 18% of systems contained detectable levels of non-THM VOCs in the period 1984-85 (11).

In Woburn, Massachussetts, an association was found between the pattern of VOC water contamination (trichloroethylene and others) and the incidence of childhood leukemias (12). This association, while controversial, suggests that further epidemiologic analysis of the pattern of non-THM VOC water contamination and the incidence of leukemias is warranted in other geographic areas.

The New Jersey Department of Health (NJDOH), under an agreement with the New Jersey Department of Environmental Protection (NJDEP), decided to perform an epidemiologic study of an ecologic design to begin its examination of the possible relationship between the incidence of leukemia and water contamination with VOCs. Access to water supply monitoring data and the existence of a population-based cancer registry facilitated the performance of this type of study. The NJDOH Cancer Registry has collected information on incident cases in the state since 1979, and has reciprocal reporting agreements with neighboring states (New York and Pennsylvania).

To perform an ecologic study, an appropriate geographic area of the

state must be selected for study. Ideally, an area selected for study should be completely served by community water systems for which monitoring data is available, and should contain subunits that experience a range of contaminant concentrations to ensure exposure differences.

## Study Population

The study area was restricted to towns completely within an area bounded by the Lower Passaic River and Saddle River drainage basins, as defined by maps prepared by the New Jersey Geologic Survey (Figure 1). The study area, which covers parts of three counties, conforms to the criteria listed above. It is almost completely served by public water supplies, many of which have tapped VOC contaminated groundwater, while others utilize groundwater or surface water free of detectable levels of non-THM VOCs. It is primarily urban and residential in character so that town coding in the Cancer Registry is likely to be accurate, since mailing address and actual residence are likely to be the same. The particular boundaries of the study area were chosen to minimize the influence of the researcher in the inclusion and exclusion of towns. Towns within the study area were excluded if less than 90% of the population was served by public water systems.

The age-, sex-, and race-specific populations of each municipality were obtained from 1980 U.S. Census summaries.

## Exposure Measurement

Relevant information on public water systems in the area was provided by the NJDEP Division of Water Resources (DWR). DWR personnel compiled all sampling results for each water system serving populations in the study area. The drinking water quality data included the 14 VOCs under the state's monitoring requirements from 1984 and 1985, and THM analyses from 1984 and 1985.

DWR provided present and historical information on extent of distribution systems, well or reservoir use, and patterns of water purchases among systems so that the water supply of each town could be characterized.

For each water system, the mean chemical-specific concentrations of VOCs and THMs in all distribution system samples were calculated by NJDOH for the period 1984-85. The number of distribution system samples for each supply varied from two to 50 in the period, and included required routine samples taken by the supplier, additional samples required of the supplier, and verification samples taken by DWR. Values for each non-THM VOC, total non-THM VOCs, and total THMs were then assigned to each town based on the water supplies that serve that town.

DWR also provided historical information on water quality in each system, including sampling information and general knowledge of the expected duration and magnitude of past contamination.

## Disease Measurement

Incident cases of leukemias for the towns within the study area were

collected from the New Jersey Cancer Registry operated by the Department of Health. Data were collected for the six-year period 1979-84. Information on each case included date of diagnosis, age at diagnosis, town of residence at time of diagnosis, race and sex, and histologic type according to the International Classification of Diseases for Oncology (13).

Data Analysis I -- Standardized Incidence Ratios

Based on the 1984-85 contaminant values assigned to each town, towns were categorized as to contaminant status for the following variables:

- 1) total non-THM VOCs
- 2) trihalomethanes

Categorical levels were defined for each variable, empirically derived from inspection of the values. Towns were grouped into categories for total non-THM VOCs and for THMs. Towns were also classified as to expected historical non-THM VOC contaminant status (approximately late 1970s to 1980) based on judgments of DWR personnel.

For each variable, incidence data for the towns within an exposure category were pooled, and standardized incidence ratios for the combined town groups were calculated. Expected incidence for the grouped areas were derived by applying New Jersey statewide 1982 age-sex-specific rates of total leukemias to the pooled 1980 age-sex-specific populations (14). Standardized incidence ratios (SIRs) were calculated by dividing the observed number of cases by the expected number for each town grouping. The 95% confidence intervals of the SIRs were calculated to assess statistical significance using the estimation method of Byar (15).

#### Data Analysis II -- Regression

For each gender, the number of leukemia cases in each exposure stratum and age-group specific population was fitted to a log-linear regression model assuming a Poisson distribution of the counts. Separate regression models were developed for total non-THM VOC and for two subsets of that variable: trichloroethylene (TCE) and tetrachloroethylene (perchloroethylene, or PCE) exposure. The general method is described by Breslow et al (16) and Clayton (17) and was performed using the GLIM software package (18).

The exposure stratum was defined as the chemical specific value or total non-THM VOC value derived for each town. Populations and cases for towns that were assigned the same value were pooled. Three age groupings (0-19, 20-49, and 50+) were used to adjust for the effect of age.

A hierarchical backward elimination method (19) was used to determine a final models. Predictor variables included in the models were age group and exposure stratum, and interactions were included. Rate ratios were calculated from the parameters generated by the models.

## **III. RESULTS**

Three of the 30 towns originally contained in the study area were excluded since less than 90% of their population is served by public community water supplies (approximately 50%, 4%, and 1%, respectively). While exact figures are unavailable, 95% to 100% of the populations in each of the remaining 27 towns in the study area are served by public community water supplies. These 27 towns contained 688,055 persons (324,049 males and 364,006 females), or 9.3% of the population of New Jersey in 1980.

Table 2 contains chemical-specific mean values assigned to each town. Appendices I and II contain more detailed information on water system VOC data and supply patterns that were used to derive the values. Total non-THM VOC average concentrations ranged from 72 ppb to below 1 ppb during the 1984-85 sampling period. Tetrachloroethylene (perchloroethylene, or PCE) and trichloroethylene (TCE) were each found at relatively high average concentrations in four town water supplies (three of which contained both). 1,1,1-trichloroethane (TCA) and dichloroethylenes (DCE) were found in relatively high average concentrations in two water systems each, in all cases together with either TCE, PCE, or both. Several additional town water supplies contained evidence of inconsistent or relatively high average concentrations in 19 of the 27 towns (up to 76 ppb). Three towns were served in 1984-85 by water supplies contaminated with relatively high average concentrations of both THMs and other non-THM VOCs. In a qualitative sense, contaminant status in the late 1970's and early 1980's based on historical information provided by DWR appears to be similar to status in 1984-1985.

Over the six-year period 1979-1984, 208 leukemia cases among males and 164 leukemia cases among females were observed in the entire study area. The age distribution of leukemias, for the major histologic types, are summarized in Table 3. The most frequently reported histologic type among younger age groups of both sexes was acute lymphocytic leukemia (ALL). Among older age groups, chronic lymphocytic leukemia (CLL), acute granulocytic leukemia (AGL), and chronic granulocytic leukemia (CGL) were most frequently reported. Few cases of acute monocytic leukemia were reported, and all were among older males. A large proportion (28%) of the reported cases were coded without specifying histology. Table 4 contains a summary of the number of total leukemia cases for each gender and town.

#### SIR Analysis

The categorization of towns for the SIR analysis is found in Table 2. Based on inspection of the average values for each town, three categories of total non-THM status were formed: ++ (72-37 ppb), + (12-5 ppb), or - (3 to less than 1 ppb). Two categories of THM status were formed: + (76-25 ppb) or - (4-1 ppb). Historical status of non-THM VOC contamination was categorized as + (evidence) or - (no evidence).

Of the 27 towns in 1984-85, five were rated ++ for total non-THM VOCs, seven were rated +, and the remainder were rated -. (Figure 2 displays the geographic pattern of this categorization.) For THMs, 19 towns were rated + and eight were rated -. For historical contaminant status, 13 towns received a + rating.

For the entire study area, the observed number of leukemia cases from 1979-84 did not differ from the expected for either males or females. However, among towns classified as ++ for total non-THM VOCs, the incidence of leukemia was significantly elevated among females (Tables 5). The incidence among males in the ++ towns did not differ from the expected (Table 6). Among towns classified as + or as - for total non-THM VOCs, leukemia incidence did not differ from the expected for either males or females.

No statistically significant elevation of leukemia incidence was observed for groups of towns classified as either + or - for THM status among males or females.

The classification of towns according to expected historical contaminant status resulted in similar SIRs as were derived from 1984-1985 data. Again, the incidence of leukemias among females was statistically greater than expected in towns classified as contaminated but not in towns classified as uncontaminated.

#### **Regression Analysis**

The data that were modeled in the regression analyses are found in Table 7. Regressions were performed for both sexes although only results for females are reported in detail here. (As in the SIR analysis, no associations were apparent for males.)

The final models of the regression analysis for females are found in Table 8. The final models do not contain interaction terms between age groups and contaminant status, since these terms did not contribute significant reductions in deviance compared to the simpler model containing contaminant status and age (Table 9). The terms for contaminant status do significantly decrease the deviance compared to the simpler model containing just age terms, and therefore remain in the final models.

These results confirm and extend the findings of the SIR analysis. Among females, the sum concentration of all non-THM VOCs was a statistically significant predictor of leukemia incidence, adjusted for age. Among females, the magnitude of the coefficient for total VOCs was 0.007 (p<0.05). This value is interpreted as the natural logarithm of the increase in the rate ratio per unit increase in exposure. Thus, for the maximum town average total non-THM VOC concentration observed in the study, 72 ug/1, the rate ratio would be exp[(72 ug/1)(.007)] = 1.65.

Modeled separately, the coefficient for TCE concentration, adjusted for

age, was also statistically significant (0.012, p<.05). Also modeled separately, the coefficient for PCE concentration, adjusted for age, was marginally significant (0.035, p=.05). At the maximum town average TCE and PCE concentrations observed in the study, the respective rate ratios would be exp[(46 ug/1)(.012)] = 1.74 and exp[(16 ug/1)(.035)] = 1.75, respectively.

- -

## IV. DISCUSSION

This study suggests an association between the contamination of drinking water supplies with non-trihalomethane volatile organic chemicals and an increased incidence of leukemias among females. But because of limitations of the study design, causal inferences cannot be made with any confidence at this time.

The study used an ecologic design, a method that has both advantages and disadvantages. Ecologic studies are practical in that they can be performed relatively quickly, utilizing existing data collected for reasons not necessarily related to the particular study. But because data is analyzed at the group level rather than the individual level, the testing of etiologic hypotheses has limitations. As Morgenstern (20) has pointed out, ecologic studies are susceptible to bias in the estimation of effect if there are factors operating at the individual level that are not accounted for in an aggregate exposure assessment. Additionally, at the group level, the degree of correlation among various predictor variables (multicolinearity) is likely to increase, so that the independent effects of predictors may be difficult to isolate. To minimize potential biases and multicolinearity in ecologic studies (and thereby increase confidence in the interpretation of an association), Morgenstern suggests making the grouping by exposure strata as small as possible and using regression to estimate effect.

Misclassification of exposure may exist at both the population and at the individual level. While it appears that sample data of community water systems in 1984 and 1985 are an adequate reflection of conditions that existed in years prior, few measurements exist to confirm this assumption, and substantial reliance on the subjective memory and experience of DWR personnel was required. While a qualitative assessment of historical contamination may be considered reliable, the actual degree of contamination cannot be known.

Historical information is important to more adequately assess the level of exposure at the time period relevant to the progression of leukemia. In this study, exposure information (1984-85) was collected at the end of the case collection period (1979-84). This apparent flaw in logic is mitigated by the information provided by DWR that indicated the probable constancy of contaminant status, at least in a qualitative sense, back in time. Additionally, the relatively short latency of leukemias, compared to other cancers, would tend to minimize the problems associated with using exposure information gathered after the development of disease.

Within a given population classified according to exposure, there may be significant variability of actual exposure to water-borne contaminants experienced by individuals. Many factors can contribute to this variability, including personal tap-water drinking habits, residential history and migration patterns, location of workplace or schools compared to residence, and other water uses that liberate volatile compounds into the indoor air. Also, exposure to the same VOCs may occur through occupation and the use of solvents and cleaning agents at home.

Confounding variables may exist. Exposures to other chemicals in the workplace, or other factors that may be causally related to the development of leukemias, may be correlated with the pattern of water contamination in the study area, so that the association observed between water contamination and female leukemias may in reality reflect another causal relationship.

The biological plausibilty of the observed association is an interesting question that bears on the interpretation of study results. At least two of the common contaminants, trichloroethylene (TCE) and tetrachloroethylene (PCE) have been shown to be carcinogenic in experimental animal studies. TCE administered by gavage for two years to B6C3F1 mice resulted in a dose-related increase in hepatocellular adenomas or carcinomas (21-22), and may have been related to increased renal tubular cell neoplasms in male Osborne-Mendel rats (23).

PCE administered by inhalation for two years to B6C3F1 mice produced a dose-related increase in hepatocellular adenoma and carcinoma, and may have been related to mononuclear cell leukemia and renal tubular cell neoplasms in F344/N rats (24). PCE administered by gavage to B6C3F1 mice for two years also resulted in increased hepatocellular carcinoma (25). The evidence from these and other animal studies suggest that TCE and PCE have the potential to be carcinogenic to humans.

Observations in humans exposed to TCE and PCE or related solvents have not demonstrated a consistent association with leukemias. Studies of the mortality experience of dry cleaning workers have variously reported statistically elevated deaths from cancer of the bladder (26), lung and kidney (27), cervix uteri, genitals, and kidney (28), and all sites, cervix uteri, lung, and skin (29). Mortality from leukemia was elevated (but not statistically significant) in one of the studies (29). In a case-referent study of childhood leukemia, fathers' exposure to chlorinated solvents was associated with excess leukemia in the children. The authors hypothesized that children may be exposed to the substances because their parents may carry home the solvents on contaminated clothing, skin, and in exhaled air (30).

Though this NJDOH study was undertaken to explore the same association that was observed in the Woburn study, there are some differences that should be discussed. The association observed in Woburn, Massachussetts was between exposure to drinking water contaminated with VOCs and the incidence of childhood leukemia, primarily of the acute lymphocytic type, and was prompted by an observed cluster of cases. The New Jersey study chose to consider all histologic types of leukemia and all ages in this ecologic study.

The concentrations of VOCs in the two contaminated wells in Woburn were reported as 267 ppb of TCE, 21 ppb of PCE, and 12 ppb of chloroform (a THM). Subsequent sampling also revealed the presence of trichlorotrifluoroethane

at 22 ppb and dichloroethylene at 28 ppb (12). The actual concentrations of contaminants in the Woburn distribution system were not reported, so comparisons cannot be made to the distribution system levels in the New Jersey towns reported here.

The appropriateness of analyzing total leukemias in this study is also an important question of biological plausibilty. Leukemias are, at least clinically, a set of different diseases. The most common histologic types of leukemias, observed both nationally and in this study, are: acute lymphocytic (ALL), chronic lymphocytic (CLL), acute granulocytic (AGL), and chronic granulocytic (CGL). ALL is the most common form in childhood, while the others are most common in adulthood. Little is known of the etiology of these four types, and the several other types, of leukemia. Known or suspected risk factors include certain genetic traits (particularly for CGL and CLL), ionizing radiation (apparently most types but CLL), infectious agents, and chemicals such as benzene (primarily AML) (31).

Combining histologic types together in this ecologic analysis may be improper if the hypothesized etiologic agent, contaminated drinking water, is specific to a particular histologic type. On the other hand, if the hypothesized agent affects the incidence of all or many types, combining types may be appropriate.

In the analyses reported here, it was not possible to analyze incidence by specific histologic type because 28% of all leukemia cases were coded to a non-specific histology. Some unknown number of the non-specific cases may have fit into a specific category, so that case ascertainment for specific histologies may be incomplete.

It is interesting that an association was observed in females but not in males. This inconsistency with respect to gender cannot be explained given the data and level of analysis used in this study. Speculation is possible but is probably not warranted without further study.

The results of this study suggest that a relationship may exist between leukemia incidence and VOC contamination of drinking water, at least among females. By itself, however, this ecologic study cannot lead to a causal inference. But since the observed association may reflect an underlying causal relationship, the potential public health importance warrants further epidemiologic study. If repeating this type of study in another area of the state (or in another state) yields a similar result, the evidence would lend support to a causal inference. An epidemiologic study design that is better able to test an etiologic hypothesis is also indicated. In particular, a case-referent study would allow an analysis of several potential risk factors that could not be assessed in this ecologic study.

## ACKNOWLEDGMENTS

We thank Sandy Krietzman and other staff of the NJDEP DWR for collecting sampling data and other information on the drinking water systems of the study area, and for reviewing our exposure assessment for accuracy. We also thank Steve Shiboski of NJDOH for providing advice on data analysis. TABLE 1. Volatile organic compounds for which semi-annual monitoring is required of public community water systems in New Jersey.

methylene chloride carbon tetrachloride 1,2-dichloroethane 1,1,1-trichloroethane vinyl chloride 1,1-dichloroethylene trans-1,2-dichloroethylene trichloroethylene tetrachloroethylene benzene xylenes chlorobenzene dichlorobenzenes trichlorobenzenes

TABLE 2. Average contaminant concentration, 1984-85, by town and contaminant, with classification of towns according to contaminant status for SIR analysis. Results in ppb ('0' - less than 1 ppb).

TOWN	HIST- ORICAL	<u>TOTAL</u> <u>NON-THM VOC</u>	<u>TCE</u>	PCE	<u>TCA</u>	DCE	<u>THM</u>	
1	+	3 -	0	1	2	0	4	-
2	-	1 -	0	0	ō	0	48	+
3	+	12 +	3	3	3	3	67	+
4	+	40 ++	23	9	2	ī	25	+
5	+	5 +	0	3	ī	ō	3	-
6	-	47 ++	0	16	28	Ō	4	-
7	+	67 ++	46	5	0	11	57	+
8	+	5 +	0	3	1	0	3	-
9	+	5 +	0	3	1	0	3	-
10	-	2 -	1	1	0	0	70	+
11	-	2 -	1	1	0	0	70	+
12	+	7 +	1	5	0	0	3	-
13	+	72 ++	14	12	0	44	41	+
14	+	5 +	0	3	1	0	3	-
15	-	0 -	0	0	0	0	76	+
16	-	0 -	0	0	0	0	76	+
17	-	1 -	0	0	0	1	39	+
18	+	9 +	1	3	0	0	48	+
19	-	1 -	0	0	0	0	62	+
20	+	37 ++	17	12	6	1	1	-
21	+	1 -	0	0	0	0	69	+
22	-	1 -	0	0	0	1	39	+
23	-	3 -	0	0	0	1	39	+
24	-	1 -	0	0	0	0	48	+
25	-	1 -	0	0	0	0	48	+
26	-	<b>1</b> -	0	0	0	0	48	+
27	-	1 -	0	0	0	0	48	+

## Categorization for SIR analysis:

Historical:	+	evidence of historical contamination
	-	no evidence of historical contamination*
Total non-THM VOCs:	++	72, 67, 47, 40, 37
	+	12, 9, 7, 5
	-	3, 2, 1, 0
THM:	+	76, 70, 69, 67, 62, 57, 48, 41, 39, 25
	-	4, 3, 1

TCE = trichloroethylene, PCE = tetrachloroethylene (perchloroethylene), TCA = 1,1,1-trichloroethane, DCE = dichloroethylenes, THM = trihalomethanes

Note: Some towns utilizing surface water sources during the drought of the early 1980s were given a - rating though they may have experienced some VOC contamination for a brief period of time.

TABLE 4. Number of leukemia cases 1979-1984 and 1980 populations by town and gender.

•

.

TOW	<u>N</u>	MA	LE	FEMALE		
		CASES	<u>POP</u>	<u>CASES</u>	<u>POP</u>	
1	ALLENDALE	2	2852	2	3049	
2	ELMWOOD PARK	5	8709	5	9668	
3	FAIR LAWN	13	15546	7	16683	
4	GARFIELD	8	12699	9	14104	
5	GLEN ROCK	1	5533	1	5964	
6	HO-HO-KUS	0	1973	0	2156	
7	LODI	5	11346	8	12610	
8	MIDLAND PARK	2	3553	1	3828	
9	RIDGEWOOD	5	11900	6	13308	
10	ROCHELLE PARK	3	2616	1	2987	
11	SADDLE BROOK	4	6769	3	7315	
12	WALDWICK	5	5314	5	5488	
13	WALLINGTON	1	5125	4	5616	
14	WYCKOFF	5	7522	2	7978	
15	BELLEVILLE	8	16440	6	18927	
16	BLOOMFIELD	21	22038	13	25754	
17	GLEN RIDGE	3	3749	3	4106	
18	MONTCLAIR	7	17583	14	20738	
19	NUTLEY	15	13768	7	15230	
20	HAWTHORNE	11	8539	7	9661	
21	HALEDON	4	2992	2	3615	
22	TOTOWA	4	5207	3	6241	
23	WEST PATERSON	4	5506	1	5787	
24	PATERSON	40	64581	25	73389	
25	CLIFTON	20	34867	19	39521	
26	PROSPECT PARK	2	2446	1	2696	
27	PASSAIC	10	24876	9	27587	
TOT	AL	208	324049	164	364006	

.

.

TABLE 5.	Standardized towns classi Number of ca	l incidence fied accor ses: 1979-	ratios (SIR ding to vari 84.	s) among f ous exposu	emales for groups of re categories.
EXPOSURE (	CATEGORY	NUMBER OF OBSERVED	CASES EXPECTED	SIR (95%)	CONFIDENCE INTERVAL)
ALL TOWNS		164	146.8	1.12	(0.95-1.30)
HISTORICAL	2 - NON-THM V	OCs			
+		68	50.8	1 34 *	(1 04-1 70)
-		96	96.0	1.00	(0.81-1.22)
TOTAL NON-	THM VOCs - 1	984-85			
++		28	18.3	1.53 *	(1, 02 - 2, 21)
+		36	30.3	1.19	(0.83 - 1.65)
-		100	98.2	1.02	(0.83-1.24)
TRIHALOMET	HANES - 1985				
+		140	126 8	1 10	(0 03-1 30)
-		24	20.0	1 20	(0.73 - 1.30)
		27	20.1	1.20	(0.77-1.70)

.

\* statistically elevated (p < 0.05)

.

TABLE 0. Stand towns Numbe	s classified acco or of cases: 1979	ording to va -84.	rious exposu	ire categorie	es.
EXPOSURE CATEGO	NUMBER O DRY OBSERVED	F CASES EXPECTED	SIR (95%	CONFIDENCE	(NTERVAL)
ALL TOWNS	208	195.7	1.06	(0.92-1.21)	)
HISTORICAL - NO	N-THM VOCs				
+ -	69 139	68.4 127.4	1.01 1.09	(0.78-1.28) (0.92-1.29)	)
TOTAL NON-THM V	/OCs - 1984-85				
++ + -	25 38 145	25.0 40.7 130.0	1.00 0.93 1.12	(0.65-1.47) (0.66-1.28) (0.94-1.31)	
TRIHALOMETHANES	3 - 1984-85				
+ -	177 31	168.5 27.3	1.05 1.14	(0.90-1.22) (0.77-1.61)	

•

TABLE 6 Standardized incidence ratios (SIRs) among males for grouns of

-

•

		AGE	GROUP			
EXPOSURE STRATUM	0-19	9	20-0	49	50+	
0 (ppb)	10,022	(1)	18,026	(2)	16,663	(16)
1	51,640	(10)	71,633	(11)	58,780	(53)
2	2,429	(0)	4,096	(0)	3,777	(4)
3	2,422	(1)	3,927	(0)	2,487	(2)
5	9,083	(0)	11,943	(2)	10,051	(8)
7	1,646	(0)	2,387	(2)	1,464	(3)
9	4,635	(0)	8,329	(2)	7,774	(12)
12	3,634	(0)	6,207	(1)	6,842	(6)
37	2,168	(0)	3,698	(1)	3,795	(6)
40	3,901	(2)	5,610	(0)	5,403	(7)
47	595	(0)	830	(0)	731	(0)
67	2,934	(1)	5,624	(2)	4,052	(5)
72	1,120	(1)	2,461	(1)	2,035	(2)
TCE						
EXPOSURE STRATUM	0-19	)	20-4	49	50+	
0 (ppb)	73,762	(12)	106,359	(15)	88,682	(79)
1	8,710	(0)	14,803	(4)	13,015	(19)
3	3,634	(0)	6,207	(1)	6,842	(6)
14	1,120	(1)	2,461	(1)	2,035	(2)
17	2,168	(0)	3,698	(1)	3,795	(6)
23	3,091	(2)	5,610	(0)	5,403	(7)
46	2,934	(1)	5,624	(2)	4,052	(5)
PCE						
EXPOSURE STRATUM	0-19	)	20-4	49	50+	
0 (ppb)	63,101	(11)	92,350	(13)	77,070	(70)
1	3,412	(1)	5,332	(0)	4,607	(5)
3	17,352	(0)	26,479	(5)	24,667	(26)
5	4,580	(1)	8,002	(4)	5,516	(8)
9	3,091	(2)	5,610	(0)	5,403	(7)
12	3,288	(1)	6,159	(2)	5,830	(8)
16	595	(0)	830	(0)	731	(0)

Note: Female population is sum for all towns that fall in the same average exposure stratum and is from the 1980 census. The number of cases divided by the population is <u>not</u> the age-specific rate since the cases were collected over a six-year time interval.

TABLE 7. Female population and number of cases (in parentheses)per age-dose group in Poisson regression analyses.

TABLE 8. Poisson regression analysis: Final models for females.

EXPOSURE: TOTAL NON-THM VOCS

<u>Model</u>	<u>Coeffic</u>	<u>ient (Standar</u>	<u>d</u> <u>Error</u> )	<b>£</b> *
Intercept	-1.85	(0.25)	<0	.0001
AGE(20-49)	-0.022	(0.32)	0	.47
AGE(50+)	1.78	(0.27)	<0	.0001
TOTAL VOCS	0.0072	(0.0039)	0	.032

# EXPOSURE: TCE CONCENTRATION

<u>Model</u>	<u>Coeffic</u>	<u>ient (Standard</u>	<u>Error</u> )	₽*
Intercept	-1.83	(0.25)		<0.0001
AGE(20-49)	-0.021	(0.32)		0.47
AGE(50+)	1.78	(0.27)		<0.0001
TCE	0.0121	(0.0067)		0.035

# EXPOSURE: PCE CONCENTRATION

<u>Model</u>	<u>Coeffic</u>	<u>ient (Standard</u>	<u>Error</u> ) <u>p</u> *
Intercept	-1.85	(0.25)	<0.0001
AGE(20-49)	-0.019	(0.32)	0.48
AGE(50+)	1.78	(0.27)	<0.0001
PCE	0.035	(0.021)	0.048

\* The p value is an approximation based on the one-tailed z distribution, which closely approximates the t distribution with 35 degrees of freedom.

TABLE 9. Poisson regression analysis: ANOVA tables for females.

# EXPOSURE: TOTAL NON-THM VOCS

<u>Model</u>	<u>Degrees</u> of <u>Freedom</u>	Deviance	<u>Change</u>
AGE	36	32.47	
VOCS	35	29.42	3.05/1
AGE*VOCS	33	28.10	1.32/2

# EXPOSURE: TCE CONCENTRATION

<u>Model</u>	<u>Degrees</u> of Freedom	<u>Deviance</u>	<u>Change</u>
AGE	18	19.60	
TCE	17	16.73	2.87/1
AGE*TCE	15	15.91	0.82/2

# EXPOSURE: PCE CONCENTRATION

.

<u>Model</u>	<u>Degrees</u> of <u>Freedom</u>	Deviance	<u>Change</u>
AGE	18	22.03	
PCE	17	19.45	2.58/1
AGE*PCE	15	19.23	0.22/2

•



FIGURE 1. The study area: all towns completely within the boundaries of the Lower Passaic and the Upper Saddle River drainage basins.

Created on NJDEP GIS

1/89 KLK NJDOH DOEH EHS



FIGURE 2. Pattern of total non-THM VOC contamination in the study area as categorized for the SIR analysis.

Created on NJDEP GIS

1/89 KLK NJDOH DOEH EHS

#### REFERENCES

1) Cantor K.P. and L.J. McCabe, 1978. The epidemiologic approach to the evaluation of organics in drinking water. In <u>Water Chlorination</u>: <u>Environmental and Health Effects</u>, <u>V.2.</u>, R.L. Jolley et al, eds. Ann Arbor Science Publishers, Ann Arbor.

2) Wilkins J.R. III, N.A. Reiches, and C.W. Kruse, 1979. Organic chemical contaminants in drinking water and cancer. <u>Am J Epidemiol</u> 110(4):420-448.

3) Crump K.S. and H.A. Guess, 1982. Drinking water and cancer: review of recent epidemiological findings and assessment of risks. <u>Ann Rev Public Health</u> 3:339-357.

4) Shy C.M. and R.J. Struba, 1982. Air and water pollution. In <u>Cancer</u> <u>Epidemiology</u> and <u>Prevention</u>, D. Schottenfeld and J.F. Fraumeni, eds. W.B. Saunders Company, Philadelphia.

5) Cantor K.P., 1982. Epidemiological evidence of carcinogenicity of chlorinated organics in drinking water. <u>Env Health Perspec</u> 46:187-195.

6) Craun G.F., 1985. Epidemiologic studies of organic micropollutants in drinking water. <u>Sci Total Environ</u> 47:461-472.

7) National Academy of Sciences, 1987. <u>Drinking Water and Health, Vol. 7</u>. National Academy Press, Washington, DC.

8) Pye V.I., R. Patrick, and J. Quarles, 1983. <u>Groundwater Contamination</u> <u>in the United States</u>. University of Pennsylvania Press, Philadelphia.

9) Tucker R.K., 1981. <u>Groundwater Quality in New Jersey</u>: <u>An Investigation of Toxic Contaminants</u>. Office of Cancer and Toxic Substances Research, New Jersey Department of Environmental Protection, Trenton.

10) New Jersey P.L.1983, c.443.

11) NJDEP, 1987. <u>Results of Testing for Hazardous Contaminants in Public</u> <u>Water Supplies Under Assembly Bill 280</u>. (Final Report for 1985 Periodic testing Intervals) Office of Science and Research, and Bureau of Safe Drinking Water, New Jersey Department of Environmental Protection, Trenton.

12) Lagakos S.W., B.J. Wessen, and M. Zelen, 1986. An analysis of contaminated well water and health effects in Woburn, Massachussetts. J Am <u>Stat Assn</u> 81(395), Applications: 583-596.

References, page 2.

13) WHO, 1976. <u>ICD-O</u>: <u>International</u> <u>Classification</u> <u>of</u> <u>Diseases</u> <u>for</u> <u>Oncology</u>. World Health Organization, Geneva.

14) NJDOH, 1985. <u>Cancer Incidence in New Jersey</u>: <u>1981-1982</u>. Cancer Registry Program, New Jersey Department of Health, Trenton.

15) Breslow N.E. and N.E. Day, 1985. The standardized mortality ratio. In <u>Biostatistics</u>: <u>Statistics in Biomedical</u>, <u>Public Health</u>, <u>and Environmental</u> <u>Sciences</u>, P.K. Sen, ed. Elsevier Science Publishers D.V., North Holland.

16) Breslow N.E., J.H. Lubin, P. Marek, and B. Langholz, 1983. Multiplicative models and cohort analysis. <u>J Am Stat Assn</u> 78(381), Applications:1-12.

17) Clayton D.G., 1983. The analysis of prospective studies of disease aetiology. <u>Communications in Statistics</u> 11:2129-2155.

18) Payne, C.D., 1985. <u>The Generalised Linear Interactive Modeling (GLIM)</u> <u>System Manual (Release 3.77)</u>. Numerical Algorithms Group, Oxford.

19) Greenberg R.S. and D.G. Kleinbaum, 1985. Mathematical modeling strategies for the analysis of epidemiologic research. <u>Ann Rev Public Health</u> 6:223-245.

20) Morgenstern H., 1982. Uses of ecologic analysis in epidemiologic research. <u>Am J Public Health</u> 72:1336-1343.

21) National Cancer Institute, 1976. <u>Carcinogenesis Bioassay of</u> <u>Trichloroethylene</u>. Technical Report 2, Bethesda MD.

22) National Toxicology Program, 1984. <u>Toxicology and Carcinogenesis</u> <u>Studies of Trichloroethylene (Without Epichlorohydrin) in F344/N Rats and</u> <u>B6C3Fl Mice (Gavage Studies)</u>. NTP Technical Report 243, Research Triangle Park NC.

23) National Toxicology Program, 1985. <u>Toxicology and Carcinogenesis</u> <u>Studies of Trichloroethylene in Four Strains of Rats</u>. NTP Tehcnical Report 273, Research Triangle Park NC.

24) National Toxicology Program, 1985. <u>Toxicology and Carcinogenesis</u> <u>Studies of Tetrachloroethylene (Perchloroethylene) in F344/N Rats and B6C3F1</u> <u>Mice (Inhalation Studies)</u>. NTP Technical Report 311, Research Triangle Park NC.

25) National Cancer Institute, 1977. <u>Bioassay of Tetrachloroethylene for</u> <u>Possible Carcinogenicity</u>. Technical Report 13, Bethesda MD. References, page 3.

26) Brown D.P. and S.D. Kaplan, 1987. Retrospective cohort mortality study of dry cleaner workers using perchloroethylene. J Occup Med 29(6):535-541.

27) Duh R.W. and N.R. Asal, 1984. Mortality among laundry and dry cleaning workers in Oklahoma. <u>Am J Public Health</u> 74:1278-1280.

28) Katz R.M. and D. Jowett, 1981. Female laundry and dry cleaning workers in Wisconsin: a mortality analysis. <u>Am J Public Health</u> 71:305-307.

29) Blair A., P. Decoufle, and D. Grauman, 1968. Causes of death among laundry and dry cleaning workers. <u>Am J Public Health</u> 69:508-511.

30) Lowengart R.A., J.M. Peters, C. Cicioni, J. Buckley, L. Bernstein, S. Preston-Martin, and E. Rappaport, 1987. Childhood leukemia and parents' occupational and home exposures. <u>JNCI</u> 79(1):39-46.

31) Heath, C.W., 1982. The Leukemias. In <u>Cancer Epidemiology and</u> <u>Prevention</u>, D. Schottenfeld and J.F. Fraumeni, eds. W.B. Saunders Company, Philadelphia.

# APPENDIX I

# WATER SYSTEM DATA SUMMARIES, 1984-1985

<u>#</u>	DISTRIBUT	<u>ION</u>	<u>AVER/</u>	<u>AGE CO</u>	NCEN'	RATION	<u>1</u> *	<u># THM</u>	<u>AVG</u>
WATER SYSTEM	SAMPLES	<u>TCE</u>	<u>PCE</u>	<u>TCA</u>	MC	<u>DCEs</u>	<u>OTHER</u>	SAMPLES	<u>THM</u>
Allendale Water Department	3		1					0	
Belleville Water Department	r 2							0	
Bloomfield Water Department	r 2							0	
Elmwood Park Wat Department	ter 3							0	
Fair Lawn Water Department	7	3	3	3	3			32	67
Garfield Water Department	20	23	9	2	<1	1	а	32	25
Glen Ridge Water Department	2							0	
Hackensack Water Company	: 14	1	1					64	70
Haledon Water Department	4				<1			31	69
Hawthorne Water Department	50	17	12	6		1	Ъ	32	1
Ho-ho-kus Water Department	6		16	28		3		6	4
Lodi Water Department	7	46	5		1	11	с	32	57
Montclair Water Bureau	14	1	3		5			32	48
Newark Water Department	3							32	76
N.J.D.W.S.C. – Wanaque North	5				<1	1		33	39

Appendix I, page 2.

WATER	SYSTEM	DATA	SUMMARIES.	1984-1985
-------	--------	------	------------	-----------

WATER SYSTEM	<u># DISTRIE</u> <u>SAMPI</u>	BUTION LES TCH	AVER <u>PCE</u>	AGE CO TCA	<u>MC</u>	TRATION DCEs	<u>1</u> * <u>OTHER</u>	<u>#</u> <u>THM</u> <u>SAMPLES</u>	<u>AVG</u> <u>THM</u>
Nutley Water Department	2							0	
Passaic Valley Water Commissi	, .on 9				1			32	48
Ramsey Water Department	3			5				15	4
Ridgewood Wate Department	r 18		3	1	1			34	3
Saddle Brook W Department	ater 3							0	
Totowa Water Department	3							0	
Waldwick Water Department	10	1	5		1			32	3
Wallington Wat Department	er 6	14	12	<1	1	44	d	32	41
West Paterson Water Departme	nt 2				2			0	

 \* ALL RESULTS IN PARTS PER BILLION (ppb) TCE - trichloroethylene PCE - tetrachloroethylene (perchloroethylene) TCA - 1,1,1-trichloroethane MC - methylene chloride DCEs - dichloroethylenes

 a 1,2-dichloroethane - 5 ppb carbon tetrachloride, benzene, trichlorobenzene, xylenes - <1 ppb</li>

```
b carbon tetrachloride - 1 ppb
c carbon tetrachloride - 4 ppb
d vinyl chloride - <1 ppb</pre>
```

```
benzene - 1 ppb
```

# APPENDIX II

# WATER SYSTEMS SERVING EACH TOWN, 1984-85 (Source of purchased water in parentheses)

TOWN	SERVED BY	APPROX. 8 OF TOWN	SOURCE WATER
Bergen County			
Allendale	Allendale Water Department	60	groundwater
	Ramsey Water Department	40	groundwater
Elmwood Park	Elmwood Park Water Department (Passiac Valley Water Commiss:	100 ion)	surface water
Fair Lawn	Fair Lawn Water Department	45	groundwater
	Passaic Valley Water Commissio Hackensack Water Company	on \ / 55	surface water
Garfield	Garfield Water Department	65	groundwater
	Passaic Valley Water Commissio	on 35	surface water
Glen Rock	Ridgewood Water Department	100	groundwater
Ho-ho-kus	Ho-ho-kus Water Department	100	groundwater
Lodi	Lodi Water Department	60	groundwater
	Passaic Valley Water Commissio	on 40	surface water
Midland Park	Ridgewood Water Department	100	groundwater
Ridgewood	Ridgewood Water Department	100	groundwater
Rochelle Park	Hackensack Water Company	100	surface water
Saddle Brook	Saddle Brook Water Department (Hackensack Water Company)	100	surface water
Waldwick	Waldwick Water Department	100	groundwater
Wallington	Wallington Water Department	50	groundwater
	Passaic Valley Water Commissio	n 50	surface water
Wyckoff	Ridgewood Water Department	100	groundwater
<u>Essex</u> <u>County</u>			
Belleville	Belleville Water Department (Newark Water Department)	100	surface water

# Appendix II, page 2.

WATER SYSTEMS SERVING EACH TOWN, 1984-85

TOWN	SERVED BY	APPROX. <u>8</u> OF TOWN	SOURCE WATER
Bloomfield	Bloomfield Water Department (Newark Water Department)	100	surface water
Glen Ridge	Glen Ridge Water Department (N.J.D.W.S.C Wanaque North)	100	surface water
Montclair	Montclair Water Bureau N.J.D.W.S.C Wanaque North	20 80	groundwater surface water
Nutley	Nutley Water Department (Newark Water Department) (Passaic Valley Water Commissi	100 .on)	surface water
Passaic County			
Clifton	Passaic Valley Water Commissio	n 100	surface water
Haledon	Haledon Water Department Passaic Valley Water Commissio	50 m 50	surface water surface water
Hawthorne	Hawthorne Water Department	100	groundwater
Passaic	Passaic Valley Water Commissio	n 100	surface water
Paterson	Passaic Valley Water Commissio	n 100	surface water
Prospect Park	Passaic Valley Water Commissio Hawthorne Water Department	n 99 1	surface water groundwater
Totowa	Totowa Water Department (Passaic Valley Water Commisss (N.J.D.W.S.C Wanaque North)	100 ion)	surface water
West Paterson	West Paterson Water Department (Passaic Valley Water Commissi (N.J.D.W.S.C Wanaque North)	60 on)	surface water
	Commonwealth W. C Little Fa (Passaic Valley Water Commissi (Montclair Water Bureau) (N.J.D.W.S.C Wanaque North)	lls 40 on)	surface water

.