

A RETROSPECTIVE STUDY OF WOBURN

1949 - 1968

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I. INTRODUCTION

This is the fifth part in a series of reports by the Massachusetts Department of Public Health on the possible association between the cancer incidence and the environmental hazards in Woburn. The initial investigation stemmed from public concern over the discovery of toxic wastes in town dump sites and the presence of chlorinated organic compounds in the drinking water in 1979. The citizens of Woburn had complained often about the quality of the drinking water and were concerned that this may be deleterious to their health. A local clergyman, Bruce Young, was alarmed by the number of childhood leukemias that he had heard of in Woburn and asked the Massachusetts Department of Public Health to investigate the situation. A local factory had also been concerned with a group of three (3) kidney cancers in their workers. Along with help from CDC, an investigation was carried out in 1980 and presented in the last MDPH report of January 23, 1981.¹

The study revealed a cluster of childhood leukemia cases in West Woburn, as well as an elevated incidence of kidney cancer. A thorough case-control study was carried out on the leukemia cases in an attempt to uncover any environmental factors which could be implicated in the etiology of the cases. No significant factors were identified. There still remained a question as to what effects the contaminated drinking water may have had on the occurrence of cancer. Since the wells were dug in the mid-1960's, a retrospective mortality study of the 1950's and 1960's was necessary in order to examine the temporal trends of cancer mortality in Woburn before, during, and after the use of the contaminated drinking water.

II. ENVIRONMENTAL HISTORY

Woburn is located 12 miles northwest of Boston (Fig. 1).¹ Originally settled as an agricultural community, it grew into a center for the leather industry and accompanying chemical industry in the late 19th and 20th centuries.^{2,3} The major industrial park today is located in the northeast section of town and is known as the Industri-plex site. On this 800 acre parcel companies have produced a variety of products ranging from chemicals for the leather and paper

industries, arsenical pesticides, TNT, and animal glues (Fig.2).³ The common practice of waste disposal was to dump them into lagoons and let the toxins settle. Unfortunately many compounds can seep through the ground and gain access to aquifers, thereby contaminating drinking water supplies. The tragedy of the Love Canal incident made the citizens of Woburn concerned that their community may be in as grave a danger and so they asked for a study to determine the extent of the pollution.

Most toxic wastes in the Industri-plex site are heavy metals (arsenic, chromium, lead) and have been deposited in discrete lagoons. Although the concentrations at the site are very high, these compounds are virtually insoluble in water and tend to remain where they have been dumped and so offer little threat of further contamination (Fig. 3).³ Large piles of animal hides had been unearthed during the excavation process for the new industrial park. These released high concentration of hydrogen sulfide (rotten egg smell) which was responsible for the horrendous “Woburn Odor.” Especially during the summer, the smell would drift across Route 93 and into Reading. An injunction was filed against the developer to stop further excavation and to cover the decaying hides with large amounts of lime and earth, but not until the buildings had been completed did the smell abate. There was also release of methane gas from the anaerobic decay process of the hides, and concentrations in one of the buildings had reached explosive levels, mandating application of additional sealant to the buildings and ventilation systems to control the accumulation of gas.³

WATER QUALITY

The people of Woburn had been complaining of the odor and color of the water for years.⁴ Most of the problem came from the presence of natural organic materials (fulvates and humates), iron and manganese which gave the water a brownish tinge. Since these trace contaminants do not pose any health hazard, the ground water is not subject to further purification steps like that of surface water.

In the early 1960’s the city of Woburn had to expand its water supply to meet the demands of its rapidly growing population. Two wells were dug in the Aberjona River Basin and connected to the water mains of Woburn. Although no definite boundary existed, the distribution is roughly that seen in Figure 4.¹ Well G was dug in 1964 and used intermittently during the

summer drought season. Well H was dug in 1967, used for approximately six (6) months and then closed until 1975. However by the late 1970's, both wells were contributing approximately one fifth of the total water needs of Woburn.⁵ Because of the concern that toxic wastes may be seeping into the Aberjona aquifer from the Industri-plex area (Fig. 5),³ the wells were tested for more than one hundred (100) chemicals on the EPA's priority pollutants list. Trace amounts of TCE (100-267 ppb) were found in both wells and they were permanently shut down in May, 1979.²

Trichloroethylene (TCE) is an industrial solvent which is used in many manufacturing processes as well as in septic tank cleaners and as a component of some of the early sealants used in underground pipe joints. It has been responsible for the closing of many public wells across Massachusetts. Like many halogenated organics that have found to be carcinogenic in experimental animals, the risk to human health is not well known.⁸ A review of the cancer incidence of various communities with different levels of halogenated organics in their drinking water found a suggestion of higher colorectal and bladder cancer in the heavily chlorinated water supplies. However this has not been seen in all studies.

A host of other halogenated organics were also found in trace amounts (Fig. 6).¹ A thorough search for a possible source of these waste products turned up little new information. One hundred eighty barrels of polyurethane waste were found approximately one (1) mile north of the wells, but no source with enough TCE to explain the contamination was found.¹

The levels of heavy metals were all less than the guidelines established by the federal drinking water regulations (Fig. 7)⁶; so no significant seepage had occurred from the Industri-plex lagoons. Further samples taken at various sites along the Aberjona River as it makes its way to the Mystic Lakes show the distribution of contaminants (Fig. 8).⁶

No further public wells have been dug in the Aberjona aquifer and the water is used only for industrial processes (mainly cooling). The rest of the city's wells are concentrated around the Horn Pond aquifer, where extensive water supplying has proved the water to have no harmful contaminants and to conform to the federal drinking water standards.

AIR QUALITY

The noxious fumes of hydrogen sulfide from the decaying hide gave the “Woburn Odor” its characteristic rotten egg smell. Levels of 0.1 – 0.5 ppm have been recorded with an odor detection level of 0.0005 ppm.⁷ These levels may cause headache, eye irritation and bronchitis in susceptible individuals. However, more serious health effects usually aren’t seen until the 10 ppm level is reached. The exposed hides were also responsible for trace amounts of toluene (1-4 ppm) and methane. Analysis of other pollutants (including arsenic and lead) revealed no measurable amounts.³ Continual monitoring of the area is being carried out by DEQE.

OTHER ENVIRONMENTAL CONCERNS

The main concerns of the citizens of Woburn is what the impact of these toxins will have on them. In order to minimize exposure and further spread of heavy metal in the Industri-plex area, these lagoons should be covered with a thick application of clay. This would prevent further run-off and airbourne dust. Since the animal hides contain organic substances such as toluene, further excavation of them would increase release of toxins. Therefore, the exposed piles will probably be handled in a similar manner. No further exposure to the public could occur if these steps were taken.

The seepage into the Aberjona River is of concern because of its course through the residential areas of Woburn and into the Mystic Lakes. The high level of contamination is mostly confined to the Industri-plex area and the amount that remains in the river is not considered hazardous. In the event of further pollution the contaminated area should be partitioned off and a clean-up process undertaken. However the waste sites in the Industri-plex area are not considered to pose increased threat to the water quality of the Aberjona River.

There are numerous other industries scattered throughout Woburn, many are light-industrial type including tanneries and machine shops. Most are in compliance with EPA and DEQE standards and pose no threat to the public. The usage of the MDC sewer system has been questioned as a source of industrial wastes, yet no significant leaks have been detected from these pipes.

III. SUMMARY OF CANCER INCIDENCE DATA – 1969 - 1978

In 1979 the MDPH, with assistance from CDC, launched a study to determine if there was an excess incidence of cancer in Woburn during the last decade. The study was prompted by the information provided by Rev. Bruce Young, a local clergyman, who had heard of ten (10) cases of childhood leukemia in the town. A careful ascertainment of all cases of leukemia was carried out and the results showed a 7-fold excess of cases in the East Woburn area around Walker Pond (Fig 9).¹ A case-control study was undertaken in order to uncover any possible factors that may have contributed to this occurrence. No significant differences could be found between cases and two sets of controls.

Concern over kidney cancer arose from the finding of 3 cases in one plant (Tabby Cat Food – now closed). A similar study revealed a possible clustering of kidney cases in the central Woburn area by place of residence 20 years prior to diagnosis. Exposure to environmental contamination may have resulted in the kidney cancer after a latent period of 20 years. Interviews with cases and next-of-kin, however, revealed no significant exposure to an environmental toxin. Every male case had given a history of having worked in an occupation where possible exposure to a known carcinogen would have occurred. Without knowing the occupations of a control group, no conclusions can be drawn from this observation.

An analysis of the cancer mortality rate (as opposed to the incidence of cancer cases) from the 1970's was also undertaken and showed Woburn to have an approximately 13% excess of cancer over the Massachusetts rates. Cancer mortality from kidney and female reproductive organs (other than uterus) were elevated at a statistically significant level, while higher rates were also noted for leukemia, prostate, breast, lung, pancreas and stomach but not at a statistically significant level. Fewer deaths than expected occurred from cancer of the large intestine and rectum, cervix uteri, and bladder. No significant time trends were noted.

These results raised a number of questions. The main concern was the apparent cluster of childhood leukemia cases in East Woburn. Since this was in the distribution area of water from Wells G and H, it was theorized that the contaminants may be responsible for the appearance of the cancer. Cancer rates from the 1950s to 1960s were needed to examine temporal trends of cancer whether they correlated with use of water from the contaminated wells. This led to the

present study where a retrospective cancer mortality study from the Woburn area was carried out for the time period 1949 – 1968.

IV. CANCER MORTALITY STUDY

As mentioned above, this study was undertaken to analyze cancer mortality in Woburn from 1949 – 1968. Of major concern was the pattern of childhood leukemia. Since the survival from childhood leukemia was 5% in 1950, mortality rates closely reflect the actual incidence figures for that time. Much improvement in the treatment of leukemia has occurred since that time and the 5 year survival rate in 1975 was greater than 70%.⁹ Therefore, one can rely on mortality figures for the 1950s and 1960s, but needs to analyze total incidence for the 1970s.

METHOD

A systematic search through all death certificates at the Woburn City Hall was carried out for the period of 1949 – 1968. Every certificate that mentioned cancer was abstracted on a form with pertinent information (name, address, age, sex, cause of death, year). A distinction was made between cancer as a cause of death and mentioned cancer as a cause were included in this study. Most of the deaths where cancer was only an incidental finding were from cancers which turned up at the time of autopsy, but weren't thought to have been a threat to the patient at the time of death from other causes—e.g., prostatic cancer of males.

The data were processed on a computer tape and analyzed according to the site of cancer, year of death, age at time of death and sex. These observed deaths were compared to the expected number of deaths, by computing standardized mortality ratios (SMRs).

The expected deaths were computed from the mortality rates on a tape generously provided by Dr. Richard Monson at the Harvard School of Public Health. He had collected data from death certificates on all causes of death since 1925 and generated age and sex-specific mortality rates. These were computed for each 5-year interval from 1925 – 1975. Massachusetts mortality rates were used for most cancers except where the numbers were so small as to become

statistically unstable, in which case the U.S. mortality figures were substituted. These tapes had been used in a previous publication for Boston and Massachusetts mortality profiles.^{10,11}

COMPARIBILITY

The mortality rates were based on information from the underlying cause of death on the death certificate. In order to make a valid comparison with this data, only those death certificates that included cancer as an underlying cause of death were included in this analysis.

Verifications of the death certificates were carried out where feasible. Approximately 80% of childhood leukemia (6/8) and kidney cancer deaths (6/9) were verified and in only one case was the cause of death changed, (a case attributed to kidney cancer, which was protatic cancer according to the hospital records).

CASE DEFINITION

A cancer case was defined as being listed on the death certificate under one of the immediate causes of death between 1949 – 1968. Childhood leukemia included all cases of leukemia 19 years of age and under.

RESULTS

A collection of 886 deaths were attributable to cancer in Woburn between 1949 – 1969. The tabulation according to CA site, listed by sex and 5-year interval is seen in Figs. 10a, b, c. The calculated SMRs have been graphically displayed for selected sites and include the SMRs for the 1970s from the previous Woburn report (Fig. 11,12,16). The use of the SMR is to enable one to compare the ratio of observed to expected deaths. An SMR of 100 means that the observed deaths equaled the number expected.

A. ALL MALIGNANT NEOPLASM

Analysis of all cancers showed remarkably stable SMRs over time. There was a gradual trend from a 5% excess in 1949 – 1953 to a 23% excess in 1974 – 1978 with a slight predominance of males greater than females as seen in Fig. 11.

B. LEUKEMIA

Analysis of the SMRs for leukemia shows a gradual increase from 76 to 123 in the period 1949 – 1968. These are separated into adult and childhood leukemias and the results for the 30-year period 1949 – 1978 are plotted on Fig. 12. This shows the increase in mortality of childhood leukemia was greater than the more gradual increase in adult leukemia. Spatial analysis of these cases is seen in Fig. 13. Both census tracts 3335 and 3336 had 9 leukemia deaths, where 4 of 9 in census tract 3335 were childhood leukemia. No childhood leukemia deaths were seen in the Walker Pond neighborhood where the case clusterings in the 1970s were observed. Only one case (jh 6/67) appeared in the distribution area of water from Wells G and H during the time that they were operational. Although all 4 cases of childhood leukemia appeared in a single census tract (3335) during the 1960s, the significance of this is not known.

The overall pattern of the adult leukemia deaths appears random, although there is a slight predominance in the census tract 3336. This is made more apparent by the map of the adult leukemias during the 1970s (Fig.14) which shows a shift of deaths from the northern section of Woburn to the center of town. This is confirmed by the calculated SMRs by census tract (Fig.15).

C. KIDNEY CANCER

The SMRs of kidney cancer as plotted graphically for the period 1949 – 1978 is seen in Fig. 16. While the mortality ratio was elevated in the 1970s, it was consistently lower than expected in the preceding 20 years. A map of the residence at the time of death and 20 years prior to diagnosis is seen in Fig. 17. For both time periods the deaths appear to be randomly distributed, although the numbers are small.

D. ALL OTHER CANCERS

Analysis of the SMRs for cancer deaths from other sites shows that most are within the expected range, with the following exceptions. Esophageal cancer shows increase with time mostly due to a higher number of male deaths. Liver cancers started with an SMR of 150, during the 1950s, then declined to 60 in the 1960s, only to become elevated again to 250 in the 1970s.

Bone cancer remained elevated throughout the 1950s and 1960s but the numbers were small and the significance of the high SMR is unknown.

V. DISCUSSION

A. ALL CANCERS

Overall cancer mortality in the city of Woburn, as measured by SMRs, closely reflects the average rate of the state of Massachusetts in the period 1949 – 1968 (Figs. 10a, b, c). The fluctuations in the SMR go from an excess of 9% in the 1950s to 13% in the 1970s. As will be seen, however, the pattern for each site of cancer is extremely variable. The most obvious reason is that as the numbers get smaller, they get more unstable and vary through what may appear extreme ranges, but is only due to the lack of statistical power. This paper excludes any analysis of measure of numerical stability (i.e. X^2 test), and the interpretation of the SMRs generated lies in the judgement of the reader.

B. LEUKEMIA

The previous report established that during the years 1969 – 1979 there was clustering of childhood leukemia cases in eastern Woburn. This was evidenced by the 7-fold increase in the incidence rates in the census tract 3334 from that expected from the TNCS. It is important to remember that the survival period of childhood leukemia has lengthened dramatically in the last 10 years, so that cases collected in the recent past will not necessarily be reflected in the same period. Mortality rates of childhood leukemia for the pre-1970s accurately depict the incidence at the time because virtually all cases died within 5 years.

The temporal pattern of childhood leukemia in the 1950s and 60s showed an increase from an SMR of 76 to 123 (Fig. 10c). While the numbers are rather low and therefore the SMRs unstable, the spatial distribution of cases shows that they are dispersed rather randomly throughout the city, and reflect the population density (Fig. 13). An important observation is that there was only 1 out of 8 cases that occurred in the distribution system of Wells G and H during the time that they were on-line. If one suspects an environmental toxin such as the contaminated

drinking water to have been a factor in the occurrence of childhood leukemias, then there must have been a sufficient latent period such that they didn't show up until the late 1970s.

However in looking for a suspected carcinogen it is important to consider adult forms of the disease as well. Virtually all known leukemogens (radiation, benzene and other agents) act on all age groups. If there was an agent responsible for the childhood leukemia in East Woburn, one would expect to see the same pattern reflected in the adult leukemia cases. While the overall adult leukemia mortality rate increased slightly between the 1960s and 1970s, the distribution of cases showed a different pattern than the childhood leukemias. During the 1970s the SMRs for adult leukemia showed a low - normal value in north and east Woburn (c. t. 3336 & 3339) but an intermediate - high value in the west and central areas (3331 – 3333), and only 5 out of 30 cases were within the distribution system of wells G and H. (Fig. 4).

The fact that the survival period of adult leukemia has not improved much in the last 10 years allows the mortality figures to correctly reflect the underlying incidence in the most recent years. However, as seen in radiation induced leukemia, the latent period may be somewhat longer in adults than in children.¹³ Therefore, an increase in adult leukemia may not have appeared yet.

Further comparison of the distributions of leukemia cases shows an apparent shift in the predominance of cases in the northern section during 1949 – 1968 to the center of town in the 1969 – 78 period (Fig. 14 & 15). While there is no suspected environmental agent responsible for this pattern, one may be concerned about the proximity of the Industri-plex area in the north. As mentioned previously, the pollutants in this area are mainly heavy metals and have remained confined in the lagoons. While these toxins are not known to be leukemogenic, arsenic in very high quantities is a proven carcinogen in humans.¹⁴ From the available environmental data, the levels of arsenic to which the public would be exposed in Woburn are in the range of natural background levels and pose no known risk to developing cancer. Although there is no reason to expect a leukemogen in this area, it is reassuring to see the numbers of cases fall off to almost nil in the 1970s.

In view of the fact that there is no evidence of any known leukemogen at large in Woburn, the most probable reason for these migratory patterns of leukemia are statistical variation over time and space.

The problem of the cluster of childhood leukemia cases still remains an enigma. The previous MDPH report examined in detail, all known risk factors in the etiology of leukemia, but was not able to identify any feature that was particular to the cases except for their proximity in time and space. Although similar clusters of childhood leukemia have been described previously, no consistent factors have been observed. The report of cases in Niles, Illinois in 1963, stated that a higher proportion of cases were members of the same church.¹⁵ Suspicious features in a recent cluster in Rutherford, N.J. (1980) were exposure to pesticide spraying and Rubella vaccination.¹⁶ However these were not strong associations and have not been reported in other epidemiological investigations.

Of the known etiologies of leukemia, perhaps the strongest is genetic. A twin of a child under 5 with leukemia, has a 20% chance of developing the disease within 6 months.¹⁷ A non-twin sib has roughly twice the risk of the general population (this holds true for most childhood cancers).¹⁸ While equally dramatic examples of genetic risks of cancer are seen in such diseases as retinoblastoma and Gardner's syndrome, these diseases are few in number and heredity does not explain the vast majority of cancers.¹⁹⁻²¹

Radiation is probably the leukemogen that we know the most about. Well established dose response relationships have been documented in the survivors of the A-bomb explosions in Nagasaki and Hiroshima and following therapeutic x-irradiation for patients with ankylosing spondylitis. A current summary is provided in the BEIR III report of 1980.¹³ However at a risk of 1 – 2 leukemia cases /10⁶ p-yr-rems over background, the amount of radiation needed to explain the cluster of cases in East Woburn would be in the range of dropping an A-bomb on Somerville.

Toxins, such as benzene, have been shown to be leukogenic in man after chronic occupational exposure.²² While the risk of developing leukemia in a benzene-laden environment has led to a greater than 5-fold excess, it has not been demonstrated as a factor in the leukemia cases occurring outside of an occupational setting.²³ There was a report of a privately owned well in the Aberjona aquifer being contaminated with benzene.²⁴ The analysis of water from Wells G and H however, has failed to detect any measurable amount of this toxin. Data are not available on the water prior to 1979, but there is no reason to believe that it contaminated the water in quantities high enough to have caused leukemia and then to have vanished.

Other toxins which are suspected of being possibly leukemogenic in humans, but for which there is no firm evidence at present are: Heptachlor,²⁵ Epichlorohydrin²⁶ and ethylene oxide.²⁷ For completeness, there are 2 drugs with an association with aplastic anemia and subsequent leukemia: Chloramphenicol²⁸ and phenylbutazone.²⁹

Finally, there are maternal factors which are considered as risk factors in the subsequent development of childhood leukemia. Radiation to the fetus in utero has been documented to increase the relative risk of leukemia and it is thought that the fetus may be an order of magnitude more sensitive to the leukemogenic effects of irradiation.³⁰⁻³² However, the case-control study effectively rules this out as an important factor in the Woburn cases. Analysis of cohorts born to mothers that were pregnant at the time of known influenza epidemics also has shown an increased risk of subsequent development of childhood leukemia.³³ The relative risk of 3.4 has been impressive in the face of possible underestimation due the inclusion of some mothers who weren't actually infected.³⁴ However no convincing evidence support an association has been established in case-control studies.

Extensive studies of other viruses have also been reported with particular concentration on the type-C RNA tumor viruses commonly seen in animals and DNA oncogenic viruses. Most of the evidence of their role in leukemogenesis in humans remains inadequate.^{35,36} On the other hand, the associations between Epstein-Barr Virus (EBV) and both Burkitts lymphoma and nasopharyngeal carcinoma are well documented and validate the suspicion of a viral etiology of some human cancers.^{37,38}

At the present time there is no evidence to support an environmental toxin in the etiology of the cases of leukemia in Woburn.

C. KIDNEY CANCER

As reported in the last MDPH publication, the incidence of kidney cancer was elevated during 1969 – 1978. While there was no evidence of environmental toxins involved, a possible association with occupational exposure (particularly lead) was seen. In addition, there was a suggestion of a concentration of cases in an area near Horn Pond by residential location 20 years before diagnosis.

The analysis of the SMRs for kidney cancer shows a relative deficiency of cases in the 1949 – 1968 period (Fig. 17). This analysis shows that kidney cancer in Woburn during the 1950s and 1960s exhibited a random pattern in space and occurred at a lower than expected rate.

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

- 1.) Figure 1: Map of Woburn and Surrounding Communities
- 2.) Figure 3: Map of Industri-plex site showing location of lagoons and water drainage
- 3.) Figure 4: Map of Woburn showing location of public wells
- 4.) Figure 5: Map of Aquifers and wells in Woburn
- 5.) Figure 8: Map of Aberjona River with concentrations of contaminants
- 6.) Figure 9: Map of Childhood Leukemia Cases 1969-1978
- 7.) Figure 10: Modeling Summary

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

Fig. 2 - History of Industri-plex Site in Woburn

YEAR	COMPANY	PRODUCT
1853	Chemical Works	chemicals for leather, textile & paper industries
1863	Merrimac Chemical Co. (MCCo.)	rapid growth & diversity . . .
1899	MCCo.	arsenical compounds for pesticides
1915	N.E. Manufacturing Co.	TNT & explosives for WWI
1917	MCCo. Purchase of Cochrane Co.	
1929	Monsanto purchases MCCo.	
1931	Woburn plant closed	(all operations carried out in Everett)
1934	NE Chemical Co.	hide & bone glues
1936	Consolidated Chemical Industries, Inc.	glue
1960	Stauffer Chemical Co.	animal glue & grease
1968	Mark Phillip Realty Trust Co.	subdivided for use as a light-industrial park

Fig. 6 - Contaminants in Woburn Wells G & H

<u>Organics</u>	<u>Well G ppb</u>		<u>Well H</u>	
	<u>5/14/79</u>	<u>9/24/79</u>	<u>5/14/79</u>	<u>9/24/79</u>
Chloroform	11.82	1.1	ND ³	ND
Trichloroethylene	267.4	117.6	183.6	63.0
Tetrachloroethylene (Perc)	20.8	18.3	13.4	9.0
1,1,1-Trichloroethane	0.6	ND	ND	2.1
Dibromochloromethane	2.0	ND	ND	ND
Trichlorotrifluoroethane ⁴ (Freon)		22 ppb		23 ppb
Dichloroethylene ⁴		28 ppb		ND
Dichlorotrifluoroethane ⁴		< 5 ppb		ND
<u>Metals</u>				
Arsenic		.0020 ppm ⁵		.0015 ppm
Chromium		ND		ND
Lead		ND		ND

1. D.E.Q.E. Special Analysis-Woburn. Water samples taken from Wells G & H by McCall, D.E.Q.E. (5/14/79) and Erickson, D.E.Q.E. (9/24/79).
2. Micrograms per liter or parts per billion (ppb)
3. ND-None Detectable
4. U.S. E.P.A. Analysis for Volatile Organics, on Woburn P.W.S., Wells G & H. July 24, 1979. Results contained in memo from R. Siscanaw, EPA to E. Taylor, EPA.
5. milligrams per liter or parts per million (ppm)

Fig. 7.- List of Heavy Metal Concentrations in Wells G & H and the Federal Guidelines

<u>COMPOUND</u>	<u>CONCENTRATION IN WELLS G & H (µG/l.)</u>	<u>MGL (µg/l.)</u>
Arsenic	10	50
Chromium	10	50
Lead	40	50
Manganese	420-1300	
Iron	39	