The Legacy of Woburn, Massachusetts and Trichloroethylene

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Abstract

Woburn, Massachusetts is an industrial, suburban city with a population of about 40,000. As populations grew in the city during the 1960's, the municipal water supply was stretched beyond its capacity. Two new wells were drilled and developed for additional community water supply. Wells G and H were drilled along the Aberjona River in a historically industrial area. Almost immediately, residents began to complain about the water. Some began to suspect the water was responsible for the occurrence of childhood leukemia and the increase in birth defects. Ten years after their use began, the Massachusetts Department of Public Health determined these wells were contaminated and shut the wells down. One of the contaminants in the wells was trichloroethylene. Hundreds of thousands of tons of trichloroethylene are produced in the US every year. Trichloroethylene is found in over 60 percent of all Superfund Sites in the US. Studies have linked trichloroethylene with leukemia and birth defects; yet, today Trichloroethylene remains listed by EPA only as a *possible* human carcinogen.

Introduction

The City of Woburn is located 10 miles north of Boston along the upper Mystic Valley of east central Massachusetts. Despite the rolling hills, waterways, and trees,

less than 2% of the city area is dedicated to recreation. Over16% of the land in the City is used for commercial or industrial production (Massachusetts Office of Environmental Affairs). Heavy industrial use is not new to the City or the area. Incorporated in 1642, Woburn was an early manufacturing center, tanning leather and making shoes. Production of shoes was so large that, during the reign of King Philip, the town taxes were partially paid in shoes for the monarchy. The Middlesex Canal from Boston opened in 1803 and the Boston and Lowell Railroad ran through Woburn in 1835. With the increased transportation options, Woburn increased the shoe and boot production. By 1865, the town of Woburn was a world renowned leather producer, shipping over $1.7 million dollars in tanned leather and leather products. In 1901, Henry Thayer , a resident and tanner of Woburn, created chrome tanning which quickly replaced bark tanning.

By 1915, the City began diversifying its industrial and commercial endeavors. soon included the production of uniforms and uniform cleaning, manufacture of machine tools, paper products, metal parts, plastics, and food products. Woburn's extensive

industrial history, spanning over 130 years, has resulted in the deposition of hazardous materials and waste products including heavy metals, volatile organics, and radionuclides.

A Civil Action, a book and recent box office hit, tells a small part of the Woburn industrial contamination story. The characters in the book and the movie are lawyers and larger than life corporate players. The real life characters are ordinary people – families with small children suffering from childhood leukemia, ordinary people who lived and worked in Woburn and who drank city supplied water. This paper explores the contamination of drinking water wells in Woburn. Specifically, trichloroethylene (TCE) contamination and its impacts on human health will be explored.

Background

In May 1979, state investigators discovered that two municipal wells in Woburn were contaminated with industrial solvents. Residents of the neighborhood served by the wells had long complained that the water was malodorous and foul-tasting. They also complained that the water ruined their clothes. Later that same year, two very large toxic waste sites were discovered fueling suspicions that local industries had polluted the drinking water wells. When researchers from the Harvard School of Public Health discovered that families who received most of their drinking water from the two contaminated wells suffered far more than the normal number of leukemia cases and immune-system disorders, Woburn entered the public consciousness of environmental disaster areas alongside Love Canal and Times Beach.

Health of Woburn residents

More than a dozen environmental health studies involving the residents of Woburn have been conducted by the Massachusetts Department of Public health (MDPH) since 1979. The most notable health issue has the been the increased incidence of childhood leukemia. Between 1969 and 1979, twelve cases of childhood leukemia were diagnosed in Woburn. Six of these cases resided in a six-block area served directly by Wells G and H. A 1981 case-control study by MDPH confirmed the elevated incidence of childhood leukemia in Woburn, especially among males. By 1986, nine additional cases had been diagnosed. There were nineteen cases of childhood leukemia over a fifteen-year period in Woburn; only six would have been expected in a normal population. The number of leukemia deaths among children under 21 years of age increased from only one in 1969-1973 to five in 1974-1978. During 1974 to 1978, 12 females and 6 males died from leukemia when forecasts indicated only 3 leukemia deaths should have occurred.

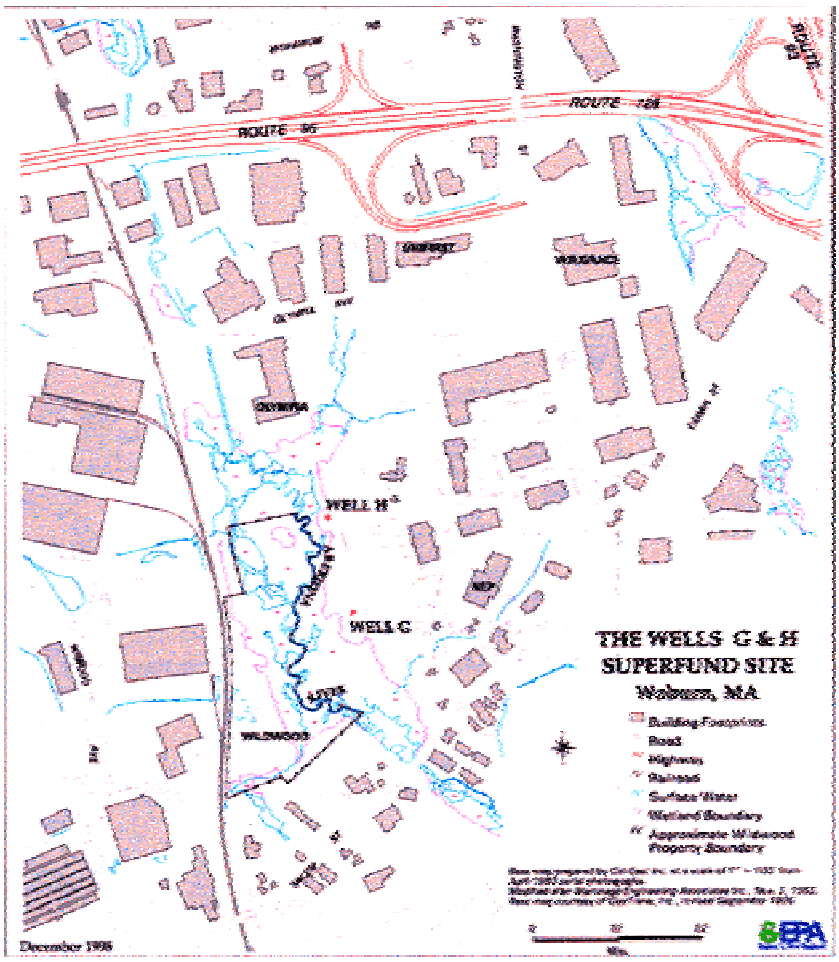
There have also been reports of excessive rates of other diseases such as certain birth defects and adult liver and kidney cancer. According to a MDPH report, deaths from cancer (not including leukemia) in Woburn increased by 17 percent from 1974 to 1978; a time when cancer deaths nationwide were on the decrease. From 1969 to 1973, four women died from cancer of the kidneys – only one death was expected. Significant incidence of prostate and urinary tract cancers occurred in the Woburn male population. From 1969 to 1973, fifteen males in Woburn died from prostate or urinary tract cancer. However, from 1974 to 1978 the number of deaths doubled to thirty.

Initially, MDPH reported that there were some elevated rates of cancer and leukemia, but none were “statistically significant”. Upon review of the 1979 MDPH

report, state statistician Robert Beattle discovered the population data used for the reports was from a 1970 census. With the closure of several manufacturing facilities, the population in Woburn had diminished since 1970. Beattle corrected the calculations using more recent census data and discovered that the death rates from leukemia and other cancers were statistically significant. MDPH reported there was a health problem in Woburn and it seemed to be clustered in the residents receiving most of their drinking water from Wells G and H. Further, they concluded that the risk of developing childhood leukemia was greater for a child whose mother drank water from the contaminated wells during pregnancy. The released study also suggested the greater the amount of contaminated water provided to the house and available for use while the mothers were pregnant, the greater the risk of the child developing leukemia.

Wells G and H

The National Priority List (NPL) site, Wells G and H, is located along the Aberjona River. The wells were used for Woburn municipal water supply from 1964 to 1979 . The wells supplied nearly 30 percent of the city's drinking water during these years. In 1976, while testing an experimental instrument (designed to detect small



quantities of organic solvent chemicals), a state health official detected inexplicably high readings from Wells G and H. But, rather than explore the source of the readings he assumed the readings were incorrect and used them to recalibrate the instrument. In 1979, city police found several 55-gallon drums of industrial waste abandoned on a vacant lot immediately adjacent to the wells. As a result of the discovery, the wells were tested and found to be contaminated. Both the wells were shut down and a supplemental water supply was arranged by the city. Further investigations in the area found five separate properties that were contributing to the contamination of the aquifer that supplied the two wells.

The NPL site covers a total area of 330 acres. The site includes commercial and industrial parks, a recreational area, and some residential gardens. The site is defined by the boundaries of Route 128 to the north, Route 93 to the east, the Boston and Main Railroad to the west and Salem Street to the South. Figure 2 provides a map of the site. The Aberjona River flows through the middle of the site. Surface water from the site is generally directed toward the River. The Aberjona River provides significant recharge to the aquifer. Consequently, contamination reaching the Aberjona River substantially impacts the quality of water in the wells.

The US EPA identified five properties from which it believed most of the pollutants in the wells had originated. Those sites were - a machine-tool plant operated by W.R. Grace, a vacant 15 acre site that was part of a tannery owned by Beatrice Foods (the site had been used for illegal waste disposal), an industrial dry-cleaning operation owned by UniFirst, a manufacturing facility of solid vinyl siding owned by New

England Plastics Corporation, and an undeveloped parcel of land and a leased trucking terminal owned by the Olympia Nominee Trust Corporation.

Contaminants in Wells G and H

Wells G and H were contaminated with trichloroethylene (TCE), tetrachloroethylene (PERC), chloroform, arsenic, 1,2 dichloroethene, and other organics and heavy metals.

Tetrachloroethylene (PERC)

UniFirst Corporation used and stored the dry-cleaning agent tetrachloroethylene. In 1979, the facility reported a 100 gallon spill. The spill was cleaned up by the Interstate Uniform Service Corporation. In 1988, Ebasco Services Inc. reported the recovery of 2 liters of a dense nonaqueous phase liquid from a monitoring well installed near the location of the removed PERC storage tank. The liquid contained 19,000,000 μg/l of PERC.

The Department of Health and Human Services has determined that Tetrachloroethylene may reasonably be considered a carcinogen. It has been proven to result in kidney and liver tumors in mice. The maximum concentration of PERC allowed in drinking water is 5 μg/l. The concentration of PERC in Wells G and H was determined to be 20.8 μg/l

Trichloroethylene (TCE)

The W.R. Grace company used trichloroethylene as a degreasing agent in its Woburn facility. The Grace company used a pit behind the plant for waste disposal and discharged some wastes to the city's sewer system. In June 1983, six 55-gallon drums of liquid waste (containing TCE) were removed from the pit. The New England Plastics

Corporation industrial well was tested and confirmed to be contaminated with various volatile organic compounds, mostly tetrachloroethylene and trichloroethylene.

TCE is used mainly as a degreasing agent but is also an ingredient in adhesives, paint removers, and spot removers. Research has indicated that TCE may cause cancer of the liver and lungs. TCE exposure is believed to be responsible for youth leukemia in several contaminated areas including Woburn. The maximum allowable level of TCE in drinking water is 5 μg/l. In the infamous 1979 sample, the groundwater in wells G and H contained as much as 267.4 μg/l TCE.

Chloroform

Chloroform is used in the manufacture of plastics, most notably vinyls. The New England Plastics Corporation manufactured solid vinyl siding and other plastic products. Chloroform was used by the company during the manufacture of several of its products.

Exposure to chloroform may result in cancer of the liver and kidneys. Inhalation of chloroform may also cause damage or cancer in the lungs. Drinking water limit for chloroform is 100 μg/l. Chloroform in the 1979 groundwater sample was found to be only 11.8 μg/l. Later tests revealed much higher concentrations of chloroform.

1,2 dichloroethene

1,2 dichloroethene is used in the manufacture of chemicals and solvents. While none of the manufacturers identified by EPA were directly manufacturing 1,2 dichloroethene, several used solvents that may have contained the compound. Additionally, two of the identified sites contained significant quantities of 55-gallon drummed waste, paints, sludges and other industrial wastes.

The long-term effects of human exposure to 1,2 dichloroethene is not known. However, some laboratory studies have shown a decreased number of red blood cells and effects on the liver in animals exposed to cis-1,2 dichloroethene. The maximum allowable level of cis-1,2 dichloroethene in drinking water is 0.07 mg/l. The maximum allowable level of trans-1,2 dichloroethene in drinking water is 0.1 mg/l. Trans-1,2 dichloroethene was found in the groundwater of Well G at a concentration of 53 mg/l.

Arsenic

Inorganic arsenic is used to treat wood and in insecticides and weed killers. In 1971, a 55-gallon drum was found on the Olympia Nominee Trust Corporation property. The drum was found to contain arsenic. In 1985, the EPA found an additional 10 rusted 55-gallon drums and pesticide caps on the property. The drums contained residues of arsenic and pesticides.

High levels of inorganic arsenic can be fatal to humans. In less than fatal doses, arsenic causes a decrease in red blood cells, blood vessel damage, abnormal heart rhythm, and skin irritations. Arsenic is a known carcinogen. The EPA has set a drinking water limit of 0.05 parts per million arsenic. Groundwater from the wells indicated arsenic levels of 10 μg/l.

TCE Focus

While all of the contaminants in Wells G and H pose a human health hazard, this paper will focus on the volatile organic contaminant Trichloroethylene. TCE is one of the most prevalent compounds found at NPL sites. In fact, 861 of the 1428 NPL sites show the presence of TCE (HazDat 1996). However, not all the NPL sites have been

evaluated for this compound. As more sites test for TCE, the number of sites with TCE contamination may increase.

Most of the TCE used in the US is released into the atmosphere by evaporation during use. In the atmosphere, TCE has a short half life, approximately 7 days. TCE deposited on the soil or in surface water volatilizes readily into the atmosphere. However, in subsurface environments, TCE is very slow to degrade and is relatively persistent. A summary of US groundwater analyses from federal and state reports shows that TCE is the most frequently detected organic solvent and the one present in the highest concentrations (Dyksen and Hess 1982). TCE was detected in 28% of surface water reporting stations in the United States. The 1984 EPA Groundwater Supply Survey of finished water from 945 drinking water systems nationwide using groundwater sources found TCE in 91 water systems.

TCE has been detected in dairy products, meat, oils, beverages (canned fruit drink, beer, instant coffee, tea, and wine), fruits and vegetables (potatoes, apples, pears, tomatoes), and bread (McConnell et al. 1975). Samples of other foods such as Chinese-style sauces, jelly, and chocolate sauce contained TCE (Entz and Hollifield 1982). TCE has been found in corn meal, fudge brownie mix, bleached flour, and cake mixes (Heikes and Hopper 1986).

TCE is clearly found in the groundwater of the US and may be in the drinking water of thousands of people nationwide. It is also found in many food products. Yet, TCE remains an enigma for risk assessment managers. The carcinogen assessment summary for TCE has been withdrawn by EPA for additional review and reference doses for chronic oral exposure and inhalation exposure have not been established.

Discussion

What is TCE?

Trichloroethylene is a chlorinated, colorless solvent. It has been produced commercially since the 1920s by chlorination of ethylene or acetylene. It has an odor that is similar to chloroform. TCE is produced naturally in the environment by several species of marine macro and microalgae (Abrahamsson et al 1995).

Used primarily as a degreasing agent, TCE was used as a dry-cleaning agent in the 1930s-1950s. Currently, approximately 80% of the world wide use is for degreasing during metal machining. TCE can also be used as building blocks for other organic chemicals such as polyvinyl chloride, some pharmaceuticals, flame retardant chemicals, and insecticides. It can be found in household products such as typewriter correction fluid, paint removers, adhesives, carpet cleaners, and spot removers.

Prior to 1977, trichloroethylene was used as a general anesthetic, a grain fumigant, skin wound disinfectant, pet food additive, and extractant of spice oleoresins in food and for the production of decaffeinated coffee. These uses were banned by a US Food and Drug Administration regulation promulgated in 1977 (IARC 1979).

TCE and the Environment

The biggest source of TCE in the environment is evaporation from factories that use it to remove grease and oils from metal parts. Trichloroethylene also enters the environment from improper waste disposal and from waste disposal sites. TCE is commonly found in landfill leachate. Measured leachate concentrations range from 0.7 to 7,700 ug/l.

Atmospheric levels are highest in areas of concentrated industrial activities and high populations. Generally, TCE levels are lower in rural and remote regions of the

world. The general population is exposed to trichloroethylene by contact and/or consumption of contaminated water or foods.

Air

According to the Toxic Chemical Release Inventory database, an estimated 49 million pounds of TCE was released to the air in the United States in 1988 (TRI88 1990). The level dropped to 30.2 million pounds in 1993 (TRI93 1995). Not all facilities that potentially release TCE are required to report for the TRI database. Once in the atmosphere, TCE reacts with photochemically produced hydroxyl radicals. The half-life is estimated to be approximately 7 days. This short half life indicates that long-range global transport of the compound is unlikely. However, the constant release of TCE and its role as an intermediate in tetrachloroethylene degradation may account for its persistence and presence in more rural areas. Due to its moderate solubility, precipitation often contains TCE.

Water

The Henry's law constant value of 2.0×10^{-2} atm-m^3/mol suggests that trichloroethylene partitions rapidly to the atmosphere from surface water. Volatilization is the primary route of removal of TCE from surface water bodies. In groundwater, TCE does not readily volatilize but can be biotransformed. Biotransformation of TCE in groundwater generally results in the metabolites vinyl chloride, ethylene, and dichloroethylene. Neither biodegradation nor hydrolysis occurs at a fast rate in groundwater conditions; thus, most concentrations of TCE in groundwater are fairly indicative of the contamination source concentration.

Soil and sediment

The majority of trichloroethylene on soil surfaces will volatilize into the atmosphere or leach into the subsurface. Because TCE is a dense nonaqueous phase liquid (DNAPL), it can move through the unsaturated soil zone into the saturated zone rapidly. Once in the saturated zone, TCE will displace the soil pore water (Wershaw et al. 1994). Biodegradation of TCE in soil is favored only under very limited conditions. In some cases, such as landfills, bacteria can use methane as an energy source to degrade trichloroethylene (Alverz-Cohen 1991). Biodegradation of TCE in soil is very sensitive to the balance of cosubstrate – too little and the degrading enzymes are not activated, too much and the enzymes out race the TCE inhibiting its decomposition (Ensley 1991).

Human Exposure

For members of the general population, the most important routes of exposure to trichloroethylene appear to be inhalation of the compound in air and ingestion of drinking water. Data indicates that dermal exposure is not a very important route for most people. Because of the high volatilization of TCE from water, inhalation may be a major route of exposure in homes with contaminated water. Hot running water, such as a shower, has been found to increase TCE levels inside homes 161 times in less than 30 minutes (Andelman 1985). One study concluded that showering for 10 minutes in water contaminated with TCE could result in a daily exposure by inhalation comparable to that expected by drinking contaminated water. The people of Woburn were exposed to TCE through their drinking water as well as through the air from using the municipal water for baths, showers, and clothes washing.

In addition to water and air exposure, some consumer products have been found to contain trichloroethylene. These products can be easily purchased at hardware or discount stores. The products include wood stains, varnishes, paint remover, adhesives, lubricants, and cleaning products. Some food products have also shown detectable levels of TCE. Exposure by consumption of processed food products is not a major route of exposure for humans. However, use of contaminated water as a home garden irrigation source may prove a significant exposure route for individuals who grow their own food.

Human Health Effects

Inhalation, oral, and dermal studies in animals and humans indicate that trichloroethylene is rapidly absorbed into the bloodstream regardless of the route of entry. Much of the TCE in the blood will be volatilized in the lungs and exhaled. The liver changes most of the remaining TCE into metabolites that are generally discharged in the urine within a day of exposure. However, some of the trichloroethylene or its metabolites can be stored in the body fat and may build up as exposure continues.

<u>Death</u>

Humans have died from breathing high concentrations of trichloroethylene fumes. Most of the reported deaths have been associated with accidental breathing. Generally, people exposed to large amounts of TCE become dizzy or sleepy and may become unconscious when exposed to very high levels. Death can occur from inhalation of high concentrations (10,000 ppm or more). Several reported deaths have occurred after TCE exposure ended but the subject engaged in physical exertion (Smith 1966). Deaths have also been recorded from the early use of trichloroethylene as an

anesthetic as well as the intentional inhalation of concentrated fumes. Death associated with liver damage has also been reported (US Department of Health and Human Services, 1997).

Death can also result from ingestion of trichloroethylene. Death associated with ingestion of TCE is generally attributed to hepatorenal failure. Very rare cases of myocardial infarction have been reported after accidental consumption of TCE.

No studies have indicated humans have died as a result of dermal exposure to TCE.

Central Nervous System

In the case of acute TCE exposure, the subject often reports dizziness, loss of facial sensation, and difficulty swallowing. More severe cases of nausea and unconsciousness have also been reported. Some permanent nerve damage resulting from TCE exposure has been reported (Feldman et al. 1985). The problems include neuro-ophthalmological impairments, facial numbness, jaw weakness, memory loss, and cranial nuclei degeneration in the brain stem. In some rare cases, acute exposure has resulted in psychotic behavior with impaired cognitive function (Steinberg 1981).

Chronic exposure to trichloroethylene can also result in central nervous system effects. Case studies have reported dizziness, loss of coordination, sleepiness, blurred vision, headaches, and confusion. Some people in Woburn showed damage to cranial nerves which manifested itself in decreased blink reflexes. Decreased intelligence test scores have been reported for some exposed individuals (Kilburn and Warshaw 1993). Among some exposed children, a significant impairment in hearing has been correlated to exposure to TCE (Burg et al. 1995). Finally, increases in mood swings have been

attributed to individuals who have been exposed to a contaminated drinking water supply.

Reproductive

Increases in miscarriages have been reported among people exposed to unspecified concentrations of trichloroethylene. Despite some of the claims of the people of Woburn, no positive correlation has been determined between the occurrence of birth defects and acute exposure of parents to TCE.

There is evidence that exposure to trichloroethylene in drinking water may cause certain types of birth defects. These birth defects include neural tube defects, oral clefts, cardiac defects, ear defects, and congenital heart disease. Despite the correlation between birth defects and exposure of the fetus to TCE, some investigators conclude that the research is far from conclusive and that there is insufficient evidence to determine whether or not there is an direct causal relationship between TCE and birth defects (US Department of Health and Human Services, 1997).

Respiratory

Some studies suggest an increased incidence of asthma, bronchitis, and pneumonia in children with chronic exposure to a TCE contaminated water supply (Byers et al. 1988). Other respiratory effects due to TCE exposure include shortness of breath, labored breathing, and respiratory edema. An increased susceptibility to respiratory infections appear to be a secondary effect on the human immune system. Despite these study results and the results of animal testing the EPA has not established a reference concentration for chronic inhalation exposure (RfC).

Cancer

The link between exposure to TCE and the incidence of cancer in humans is controversial. As with the residents of Woburn, people in a New Jersey cancer study saw an increased incidence of childhood leukemia in towns with high exposure to TCE (Fagliano et al. 1990). Another study of 1.5 million resident in 75 towns monitored between 1979 and 1987 reported a significant elevation of total leukemia, childhood leukemia, acute lymphatic leukemia, and non-Hodgkin's lymphoma in females exposed to more than 5 ppb TCE (Cohen et al. 1994). The Massachusetts Department of Public Health (MDPH) reported in 1996 that the incidence of childhood leukemia in Woburn was 8 times higher when the child was exposed to TCE in utero than non-exposed children. It concluded that these results consistently support the hypothesis that childhood leukemia in Woburn was directly related to the mother's exposure to TCE contaminated drinking water during pregnancy.

Other types of cancers have been found in humans exposed to TCE. These cancers included prostate, urinary tract, cervical, kidney, respiratory, and liver. Investigations into human TCE exposure have not found statistically significant increases in cancer occurrences. However, much anecdotal evidence suggests a correlation between TCE exposure concentration and duration and several cancers. The EPA considers trichloroethylene as an intermediate between a probable and possible human carcinogen. TCE is classified by EPA as a Group 2A compound: *The agent is probably carcinogenic to humans. The exposure circumstance entails exposures that are probably carcinogenic to humans.* This category is used when there is limited evidence of carcinogenicity in humans but sufficient evidence of carcinogenicity in laboratory animals.

Extrapolation of animal toxicity data to predict human risk is controversial and is difficult. In the case of trichloroethylene, it is especially difficult. Some of the mechanisms implicated in animal effects do not exist in humans. Additionally, some laboratory test animals metabolize TCE in pathways that are similar to humans but internal organ morphologies are disparate. These limitations in laboratory animal testing have made TCE cancer risk development problematic. While the debate wages on, people may be exposed to a carcinogen in their drinking water, on their jobs, in their gardens, and in their showers. Recognizing these public health concerns and the apparent ubiquitous distribution of TCE, the EPA is currently revising its cancer risk assessment for TCE (IRIS).

Controls

There are several regulations and guidelines for trichloroethylene exposure.

Agency	Water	Food	Air	Other
OSHA			100 ppm – PEL 300 ppm - STEL	
FDA		Indirect food additive for use only as an adhesive		
EPA ODW	0.005 mg/l -MCL			
EPA OSW	Toxic pollutant		Hazardous Air pollutant	Hazardous waste Hazardous constituent Right-to Know reporting required
EPA OERR				Reportable quantity 1000 pounds

PEL – permissible exposure limit, STEL – short term exposure, ODW- Office of Drinking water, MCL – maximum contaminate limit, OSW – Office of Solid Waste, OERR – Office of emergency and remedial response.

Despite these regulatory limits, TCE continues to enter the atmosphere and groundwater of the industrialized countries, including the US. There are some protections that individuals can take to minimize exposure to TCE. These protections include increased hydration with uncontaminated water, reduced use of acetaminophen (and other drugs that lower GSH levels), decreased alcohol consumption, eliminate cigarette smoking, use appropriate respiration protection when working with trichloroethylene containing products, and using paint removers, spot cleaners, and degreasers in well ventilated areas. Manufacturing facilities often install scrubbers or air strippers on stacks to reduce volatile organic emissions.

The recommended method of trichloroethylene disposal is incineration after mixing with a combustible fuel. Incomplete combustion of TCE can result in the formation of phosgene, polycyclic aromatic hydrocarbons, and perchloroaromatic. Acid scrubbers are used on incinerators burning TCE wastes to remove the haloacids produce during combustion. EPA restricts land disposal of halogenated organic solvents such as trichloroethylene. Because TCE poses a potential human cancer risk and disposal costs are high, there has been an emphasis on recovery and recycling of TCE.

Once TCE has contaminated a groundwater source, such as Wells G and H in Woburn, there are some means of control and remediation. Control and remediation of a contaminated site includes:

1) Removal of human contact (fencing or personal protection equipment)
2) Immediate removal of the contaminant source (leaking drums, pure product in a well)

3) Contamination migration control (excavation of contaminated soils, soil incineration, capping, soil vapor extraction systems)
4) Groundwater treatment (pumping, UV-chemical oxidation, carbon filtration, air stripping)

Summary

There is no doubt the people of Woburn, Massachusetts were exposed to several contaminants when municipal water supply Wells G and H went on line in 1969. Some investigators have suggested that the exposure of its residents was solely the responsibility of the City of Woburn. A water consultant's report to the City in 1958 characterized the groundwater of the Aberjona River Valley as *"…too polluted to be used for a public water supply."* (Whitman & Howard 1958). Yet, the wells were drilled and the groundwater was used as a municipal water source for ten years.

Childhood leukemia and other human health problems plagued some Woburn residents, with the number of cases increasing dramatically after the wells came on line. After ten years of constant use and human consumption, the wells were found to be contaminated and closed down.

Were the five companies named by EPA the only contributors to the groundwater contamination? Probably not. Were the contaminated wells responsible for the increased diseases and defects occurring in Woburn? EPA and MDPH are not certain, but they are certain that continued contamination of ground water, surface water and soil, and continued human exposure are not acceptable. Was trichloroethylene the sole contaminate responsible for the increased childhood leukemia in Woburn? Maybe not. Was trichloroethylene the sole contaminate responsible for the birth defects

reported by Wells G and H users? Maybe not. Can Trichloroethylene cause leukemias, cancers, and birth defects? Probably. EPA concedes that TCE is *probably carcinogenic to humans*. Yet, the agency has not established a reference dose for chronic oral exposure, reference concentration for chronic inhalation exposure, or completed a carcinogenicity assessment for lifetime exposure.

TCE is one of the most ubiquitous groundwater contaminants at NPL sites in the US. It is difficult for risk managers to assess the level of risk or determine agency acceptable remediation levels for TCE contaminated sites due to the lack of EPA risk information. Currently, EPA requires industry to report spills of 1000 pounds or more of TCE. It has been proposed that this level be reduced to 100 pounds. Until acceptable clean up levels can be determined for TCE, source control, water testing, and personal protection are the most important processes available to safeguard the public.

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