Massachusetts
Department Of
Public Health

Health Consultation: Evaluation of the Incidence of Brain and Other Nervous System Cancers in Medfield, Massachusetts and Review of Tetrachloroethylene (PCE) in Municipal Drinking Water

Medfield, Norfolk County, Massachusetts

Bureau of
Environmental Health,
Community Assessment
Program

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BACKGROUND

This investigation is to follow up on a recent request to the Massachusetts Department of Public Health Bureau of Environmental Health (MDPH/BEH) from a resident for information on the incidence of brain cancer in a neighborhood in Medfield and the possibility of an association with historical exposures to tetrachloroethylene (also known as PCE, perchloroethylene, PERC and tetrachloroethene) in the municipal drinking water supply[1]. PCE is a synthetic chemical that is widely used in the dry cleaning process and as a degreasing solvent. PCE can travel through soils easily and as a result, contaminate groundwater (ATSDR 1997).

The Community Assessment Program (CAP) is a division within MDPH/BEH that investigates reports of suspected disease clusters in communities throughout Massachusetts. In response to this request, CAP staff contacted the Medfield Department of Public Works (DPW) and the Massachusetts Department of Environmental Protection (MDEP) to obtain available information about any current or historic levels of PCE in the municipal drinking water within the community. In addition, CAP staff reviewed available cancer incidence data from the Massachusetts Cancer Registry (MCR) for the community of Medfield to assess whether an atypical pattern of cancer may be occurring there. This report summarizes the results of MDPH/BEH's evaluation.

DISCUSSION

Tetrachloroethylene (PCE) in Drinking Water

The municipal drinking water for Medfield is supplied by five groundwater wells located within the community. Wells 1, 2, and 6 are located within the Charles River aquifer while Wells 3 and 4 are located within the Neponset River aquifer (Figure 1). The water system also includes five pumping facilities, two water storage tanks, and approximately 76 miles of water main pipe (Town of Medfield 2009).

MDPH obtained available analytical PCE data from the MDEP Drinking Water Program for the nine-year period 1988-1996. Wells 1 and 2, which are only used during peak summer demand, have been treated historically for the removal of PCE. The source of the PCE that was detected in these two Medfield wells is unknown.

The Massachusetts Maximum Contaminant Level (MMCL) is a state-wide standard that applies to drinking water provided by public water systems. Of the 15 samples collected from Well 1 during 1988-1996, one had levels of PCE that exceeded the MMCL of 5 ppb. This sample was collected in 1995 and had a PCE concentration of 9.4 ppb (Table 1). PCE exceedances above the MMCL in water from Well 2 occurred in 6 of 11 samples collected during this time period. The earliest exceedances were detected in 1991, with a maximum concentration of 9.9 ppb detected in 1995 (Table 2) (D. Guterman, MDEP, personal communication, 2010; MDEP 2010). Therefore, a completed exposure pathway existed in the past due to PCE in drinking water. Overall,

[1] This report was supported in part by funds from a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Department of Health and Human Services. This document has not been reviewed and cleared by ATSDR.

the average concentration of PCE in the two wells combined during the nine-year period was 3.5 ppb.

In 1996, an aeration tower was installed to remove PCE from the drinking water from Wells 1 and 2 prior to distribution. The aeration tower was in use by December 1996 as indicated by samples collected both before and after treatment (MDEP 2010). Sampling results from December 1996 to October 2009 indicate that no detectable levels of PCE were found after this treatment process was put into place. As of November 2009, the aeration tower is no longer in use, and PCE is sampled on a quarterly basis per agreement with MDEP. If the average concentration of PCE detected in the previous four quarters is greater than or equal to ½ the MMCL (2.5 ppb), then the aeration tower will be turned back on (K. Feeney, Medfield DPW, personal communication, 2010). As a result, present and future exposures to PCE in drinking water from Wells 1 and 2 were eliminated as exposure pathways.

In the past, it is possible that residents of Medfield could have been exposed to PCE in municipal water from Wells 1 and 2 via ingestion, inhalation (i.e., while showering), and/or dermal contact (i.e., washing hands or bathing with water containing PCE) during periods of peak demand (i.e., summer months). A completed exposure pathway whereby PCE was detected above the MMCL is documented from about 1991 through 1996, at which time the aeration tower was put in use. However, it is unlikely that a resident would have ingested the maximum concentration of PCE due to mixing with water from other wells and due to the intermittent use of the backup wells. While houses in close proximity to Wells 1 and 2 are likely to receive more of their water from these two wells than houses farther away, mixing with water from other wells would have likely reduced the concentration of PCE. This is supported by data showing samples from Wells 1 and 2 during 1995 ranging from 3.6 to 9.9 ppb whereas levels of PCE detected in samples collected in November 1995 from the taps of 6 residences located throughout the distribution system ranged from non-detect to 1.4 ppb (D. Guterman, MDEP, personal communication, 2010; MDEP 2010).

A secondary pathway was investigated, as PCE can also leach into drinking water from vinyl-lined asbestos cement water mains. This type of lined pipe was used historically in the water distribution systems of several communities in Massachusetts. It was confirmed, however, with the Medfield DPW that there is no vinyl lining in the drinking water pipes within the community of Medfield (K. Feeney, Medfield DPW, personal communication, 2010). Therefore, past, present or future exposures to PCE from vinyl-lined asbestos cement water mains were eliminated as exposure pathways.

PCE Exposures and Noncancer Health Effects

In order to evaluate the potential for noncancer health effects, exposure doses were estimated and compared to health guideline values for estimating noncancer health risks. Using highly conservative assumptions that an adult ingested 2 liters of water and a child ingested 1 liter of water containing the maximum concentration of PCE detected according to available records (i.e., 9.9 ppb in Well 2 in 1995) for 150 days per year (May through September) for a maximum potential exposure duration (9 years from

1988-1996), the estimated noncancer effects exposure dose is 0.0001 milligrams per kilograms per day (mg/kg/day) for adults and children. This estimated daily exposure dose is substantially less than the U.S. EPA chronic oral RfD (0.01 mg/kg/day), which represents an estimate of a daily oral exposure that is not expected to result in adverse noncancer health effects (USEPA 1988). As a result, noncancer health effects from past exposure to PCE in Medfield municipal water are not expected. It is important to note that using the maximum concentration that was detected in Wells 1 and 2 likely overestimates the exposure dose since the available information indicates that the overall concentration of PCE reaching nearby homes would have been reduced by mixing with water from other wells in the distribution system. See attachment A for more information on the noncancer effects exposure dose calculation.

It should also be noted that sodium (a nutrient) was detected at elevated concentrations in Wells 1 and 2. A maximum concentration of 40 ppm of sodium was measured in these wells. This concentration exceeds the Massachusetts guideline for sodium in drinking water of 20 ppm. Sodium is a naturally occurring element found in water and soil. It is an essential mineral, which is necessary for the normal functioning of the body and maintenance of body fluids. The Massachusetts guideline of 20 ppm in drinking water represents a level of sodium in water that physicians and sodium-sensitive individuals should be aware of in cases where sodium exposures are carefully controlled. People who have difficulty regulating fluid volume as a result of several diseases such as hypertension and kidney failure are particularly affected by elevated levels of sodium (MDPH 2007). MDPH's fact sheet on sodium in drinking water is included in Attachment B.

PCE Exposures and Carcinogenic Health Effects

PCE has been classified as a probable human carcinogen by the International Agency for Research on Cancer (IARC) and is reasonably anticipated to be a carcinogen by the National Toxicology Program (NTP) of the U.S. Department of Health and Human Services. These evaluations were based on limited evidence in humans and sufficient evidence in experimental animals. Although brain and other nervous system cancers are not cancer types that are likely to be associated with exposure to PCE based on the epidemiological literature, exposure doses were estimated and compared to health guidelines for estimating cancer risk (ATSDR 1997, 2011; Cantor et al. 2006; Siemiatycki et al. 2006) Extensive literature exists on the toxicity and carcinogenicity of PCE. For a full discussion, refer to the ATSDR Toxicological Profile (1997) at http://www.atsdr.cdc.gov/ToxProfiles/tp18.pdf.

For the purposes of evaluating carcinogenic health effects, a conservative approach was employed. The exposure dose received from inhalation and dermal exposures while showering were considered to be equal to the estimated ingestion exposure dose (USEPA 2000). Under the same assumptions as for the above noncancer health effects and using the California EPA cancer slope factor ($0.54\ [mg/kg/day]^{-1}$), the estimated cancer risk is 1.6×10^{-5}, indicating that past opportunities for PCE exposure via municipal drinking water are unlikely to result in unusual cancer risks for either adults or children. The theoretical cancer risk calculation estimates an excess cancer risk in terms of the

proportion of the population that may be affected by a carcinogenic substance over a lifetime of exposure (ATSDR 2005). In other words, the estimated cancer risk of 1.6 in 100,000 ($1.6x10^{-5}$) means that there is a probability of about one or two additional cancer diagnoses over background levels in a population of 100,000 people. See Attachment A for more information on the calculation of the estimated cancer risk.

Review of Massachusetts Cancer Registry Incidence Data

Cancer is not just one disease, but is a term used to describe a variety of different diseases. As such, studies have generally shown that different cancer types have different causes, patterns of incidence, risk factors, and latency periods (time between exposure to a cancer-causing agent and the development of the disease). Despite numerous scientific and medical studies, the causes of brain and other nervous system cancers are still largely unknown. Most brain and other nervous system cancers develop for no apparent reason and are not associated with anything that the person did or didn't do, or with any known exposures in the environment. The most established risk factor for brain and other nervous system tumors is high-dose exposure to ionizing radiation such as that used for the treatment of other cancers (ACS 2009a, b). According to the medical literature, the latency period for brain and other nervous system cancers could range anywhere between 10 and 40 or 50 years. Although exposure to PCE is not likely to be associated with the development of brain and other nervous system cancers, the incidence of these specific cancer types was evaluated by MDPH in response to a request of a concerned resident.

The MCR is a division in the MDPH Bureau of Health Information, Statistics, Research, and Evaluation (BHISRE). It is a population-based surveillance system that has been monitoring cancer incidence in the Commonwealth since 1982. All new diagnoses of invasive cancer, as well as certain *in situ* (localized) cancers, among Massachusetts residents are required by law to be reported to the MCR within six months of the date of diagnosis (M.G.L. c.111. s 111b). The MCR also gathers background information (e.g. gender, age, and address at diagnosis) on each individual reported. This information is kept in a confidential database. Data are collected daily and reviewed for accuracy and completeness on an annual basis. Due to the high volume of data collected and the six-month period between diagnosis and required reporting, the most current registry data that are complete will be a minimum of 2 years prior to the current date. At the time of this investigation, 2002-2006 constitutes the 5-year time period for which the most recent and complete cancer incidence data are available.

To assess the incidence of cancer in a community, the number of observed diagnoses is compared to the number of expected diagnoses, which is calculated based on the statewide cancer experience and adjusted for the population structure of the community. The incidence of malignant brain and other nervous system cancers in the community of Medfield, which had a population of about 12,300 in 2000 and encompasses approximately 14.5 square miles, was reviewed for the 5-year period from 2002-2006. During this time period, brain and other nervous system cancers occurred approximately as expected among males (4 observed vs. 3 expected) and as expected among females (2 observed vs. 2 expected). Although the number of observed diagnoses among males exceeded the number of expected diagnoses by one, this was likely a result of random

fluctuation and represents natural variation. It should be noted that secondary brain tumors, which originate elsewhere in the body and then metastasize to the brain, are not included.

Primary brain and other nervous system tumors consist of two main histology (or tissue) types: gliomas and meningiomas. Gliomas are a general classification of brain and other nervous system tumors that includes astrocytomas, oligodendrogliomas, and ependymomas. According to the American Cancer Society (ACS), gliomas account for approximately 80% of malignant brain and other nervous system tumors. Astrocytomas are the most common type of glioma. Glioblastoma multiforme (also referred to as glioblastoma for short) is a high grade, aggressive form of astrocytoma. Glioblastomas account for about two-thirds of all astrocytomas and are the most common malignant brain tumors in adults. Meningiomas arise from the meninges, the layers of tissue that surround the outer part of the brain and spinal cord. Approximately 80% of meningiomas are non-malignant. Brain and other nervous system tumors are the second most common cancer type among children (after leukemia) and account for over 20% of childhood cancers. After a peak in childhood, the risk of brain and other nervous system cancers increases with age between 25 and 75 years (ACS 2009a, 2009b).

The histology types and age patterns of those individuals diagnosed with malignant brain and other nervous system cancers in Medfield during 2002-2006 appear to be consistent with what would be expected based on the medical literature and national cancer statistics. All six individuals, the majority of whom were adults, were diagnosed with gliomas. To protect the privacy of the Medfield residents diagnosed with brain and other nervous system cancers, their specific cancer subtypes will not be discussed here.

A review of the geographic distribution of residences of individuals diagnosed with brain or other nervous system cancers during this time period was based upon address at the time of diagnosis. The spatial distribution was assessed by qualitatively evaluating the point pattern of diagnoses using a computerized geographic information system (GIS). The geographic distribution was generally consistent with the pattern of population density. No unusual spatial pattern or concentration of diagnoses was observed. The MDPH is bound by law not to make public the names or any other information (e.g., place of residence) that could personally identify individuals with cancer whose diagnoses have been reported to the MCR (M.G.L. c.111. s. 24A). Therefore, for confidentiality reasons, it is not possible for the MDPH to release maps showing the residential locations of individuals diagnosed with cancer.

CAP staff also conducted a qualitative review of more recent diagnoses of brain and other nervous system cancers that occurred in Medfield from 2007 to present. As mentioned previously, the MCR data file for these more recent years had not yet been closed at the time of this investigation. For that reason, an expected number of diagnoses for these years could not be calculated. Based on the available information, although some diagnoses occurred among individuals whose residences at the time of diagnosis were in relative close proximity to one another, the geographic distribution of these more recent diagnoses generally followed the pattern of population density. The histology types and

age patterns of those individuals also appear to be consistent with what would be expected.

CONCLUSIONS

1) PCE was detected at levels above the MMCL in Wells 1 and 2 from about 1991 through 1996, at which time an aeration tower was put in place. Based on highly conservative assumptions about the concentration, frequency and duration of potential exposures, no unusual risks of cancer or other adverse non-cancer health effects are expected to result from these past exposures.

2) During the 5-year period from 2002-2006, the incidence of brain and other nervous system cancers in the community of Medfield was approximately as expected among males (4 observed vs. 3 expected) and as expected among females (2 observed vs. 2 expected) when compared to the statewide cancer experience. Although the number of observed diagnoses among males exceeded the number of expected by one, this was likely a result of random fluctuation and represents natural variation. The histology types and age patterns of those individuals diagnosed during this time period appear to be consistent with state and national trends.

3) Analysis of the geographic distribution of place of residence for individuals diagnosed with brain and other nervous system cancers in Medfield during 2002 to present did not reveal any unusual spatial patterns. In general, the geographic distribution was consistent with the pattern of population density.

Based on the MDPH's evaluation of the available environmental data, the exposure pathway analysis, and risk factor information related to brain and other nervous system cancers, the MDPH concludes that:

For adults and children in the community of Medfield, drinking, touching (i.e., washing hands or bathing), or breathing while showering with municipal water containing PCE at levels reviewed for this evaluation in the past is not expected to result in health effects. Based on the available information, potential exposures are unlikely to be of sufficient concentration and duration to result in health effects.

RECOMMENDATIONS

The MDPH recommends no further investigation of the incidence of brain and other nervous system cancers in the community of Medfield at this time, but will continue to monitor the incidence of all cancer types in the community of Medfield through city/town cancer incidence reports published by the Massachusetts Cancer Registry.

For more information about recent water quality tests conducted on drinking water in the community of Medfield, contact the Medfield Department of Public Works at 508-359-8505.

PUBLIC HEALH ACTION PLAN

The Public Health Action Plan contains a description of actions to be taken by the MDPH subsequent to completion of this Health Consultation. The purpose of the Public Health Action Plan is to ensure that this health consultation not only identifies public health hazards, but also provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of the MDPH to follow up on this plan to ensure that it is implemented. The public health actions to be implemented by MDPH are as follows:

- The MDPH will continue to monitor the incidence of all cancer types in the community of Medfield through city/town cancer incidence reports published by the Massachusetts Cancer Registry.

REFERENCES

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Figure 1
Public Water Supplies
Medfield, Massachusetts

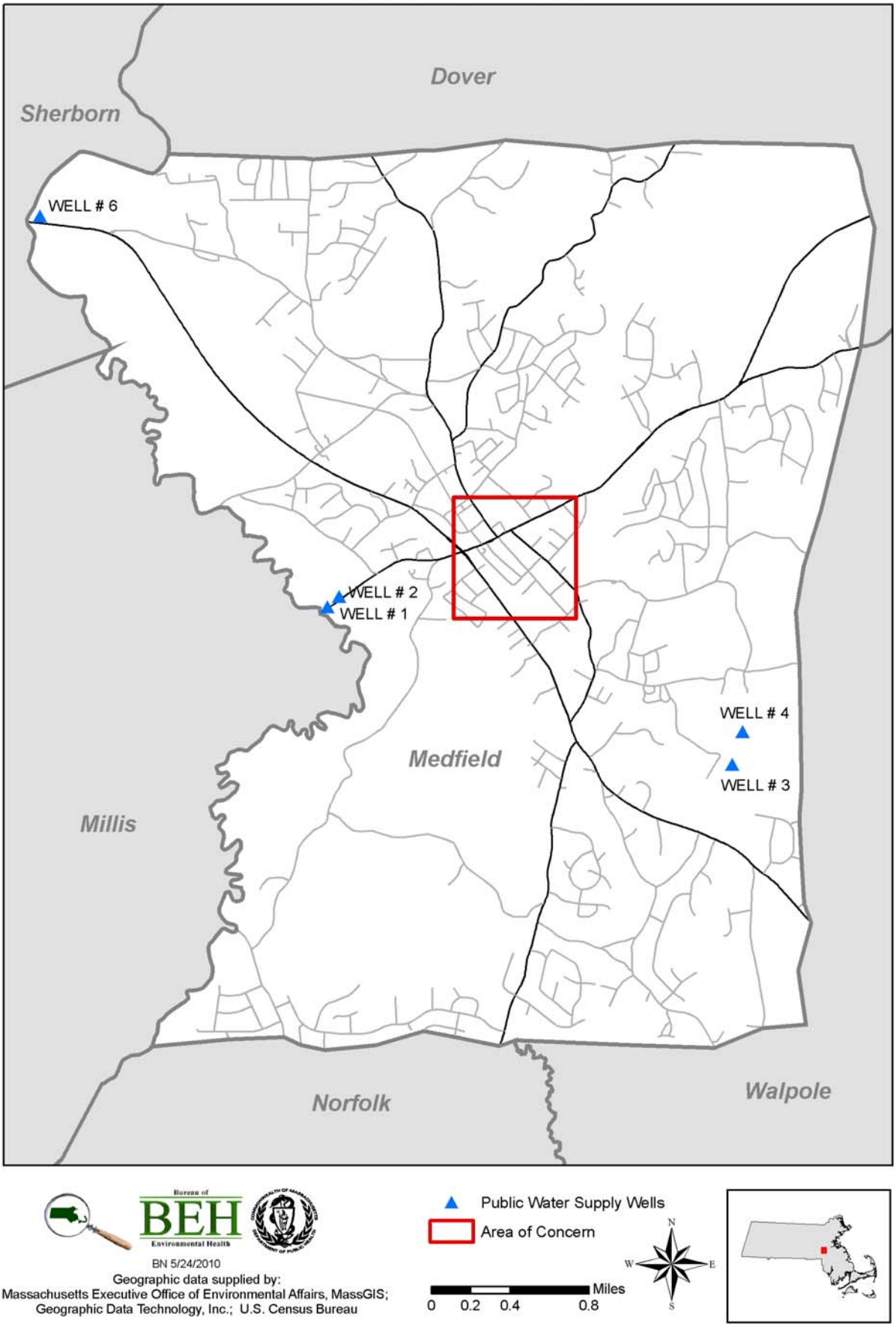

Table 1
Concentrations of Tetrachloroethylene (PCE) Detected in Well 1, 1988-1997
Medfield, Massachusetts

Date	PCE (ppb)
3/21/1988	0.25*
8/15/1990	0.5
1/3/1991	1.0
6/17/1992	0.6
3/17/1993	0.7
4/21/1993	0.8
9/1/1993	1.1
12/16/1993	1.2
1/17/1995	9.4
4/5/1995	3.6
11/16/1995	3.6
3/26/1996	3.0
6/26/1996	4.2
9/19/1996	1.6
12/19/1996	2.0
3/21/1997	0.25*‡

* Non-detect. Result is 1/2 of the detection limit.

‡ Sample was collected after treatment.

Table 2
Concentrations of Tetrachloroethylene (PCE) Detected in Well 2, 1988-1997
Medfield, Massachusetts

Date	PCE (ppb)
3/21/1988	0.25*
8/15/1990	4.0
1/3/1991	5.1
6/17/1992	5.2
3/17/1993	4.3
4/21/1993	3.3
9/1/1993	8.8
12/16/1993	8.9
1/17/1995	9.9
4/5/1995	8.2
12/12/1996	0.25*‡
3/21/1997	0.25*‡

* Non-detect. Result is 1/2 of the detection limit.

‡ Sample was collected after treatment.

Attachment A
Exposure Dose and Cancer Risk Calculations for Exposure to PCE in Drinking Water
Medfield, Massachusetts

Exposure Dose and Cancer Risk Calculation Formulas:

Noncancer Health Effects Exposure Factor:

$$NC\_EF = \frac{F \times ED}{ED \times 365\ days}$$

Noncancer Health Effects Exposure Dose (Ingestion):

$$NC\_D = \frac{[C]_{drinking\ water} \times IR \times NC\_EF}{BW}$$

Cancer Effects Exposure Factor:

$$C\_EF = \frac{F \times ED}{70\ years \times 365\ days}$$

Cancer Effects Exposure Dose (Ingestion):

$$C\_D = \frac{[C]_{drinking\ water} \times IR \times C\_EF}{BW}$$

Cancer Risk:

$$CR = C\_D \times CSF$$

Where:

NC_EF	= Noncancer Exposure Factor (unitless)
F	= Frequency of Exposure (days/year)
ED	= Years of Exposure (years)
NC_D	= Noncancer Exposure Dose (mg/kg/day)
$[C]_{drinking\ water}$	= Maximum Analyte Concentration in Drinking Water (mg/L)
IR	= Intake Rate (L/day)
BW	= Body Weight (kg)
C_EF	= Cancer Exposure Factor (unitless)
C_D	= Cancer Exposure Dose (mg/kg/day)
CR	= Cancer Risk (unitless)
CSF	= Cancer Slope Factor ($mg/kg/day^{-1}$)

Assumptions:

1) The receptors evaluated were an adult resident and a child.
2) The drinking water concentration was assumed to be the maximum concentration of PCE detected in drinking water from Well 1 or 2 from the available records.
3) The amount of drinking water ingested was assumed to be 2 liters per day for the adult receptor and 1 liter per day for the child.
4) The exposure factor was determined assuming the adult and child receptors were exposed to contaminated drinking water for 150 days per year (during summer demand only, May through September) over a 6 year time period.
5) The average body weight was assumed to be 70 kilograms for the adult receptor and 35 kilograms for the child receptor.
6) For the purposes of evaluating cancer risk, exposure dose received from inhalation and dermal exposures while showering were considered to be equal to the estimated ingestion exposure dose.

1. Exposure Dose and Cancer Risk Calculations for Ingestion of Drinking Water Containing PCE:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{150\ \text{days/year} \times 9\ \text{years}}{9\ \text{years} \times 365\ \text{days}} = 0.41$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{0.01\ \text{mg/L} \times 2\ \text{L/day} \times 0.41}{70\ \text{kg}} = 0.00012\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{150\ \text{days/year} \times 9\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.05$$

$$\text{Cancer Effects Exposure Dose} = \frac{0.01\ \text{mg/L} \times 2\ \text{L/day} \times 0.05 \times 2}{70\ \text{kg}} = 0.00003\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00003 \times 0.54 = 0.000016$$

b. Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{150\ \text{days/year} \times 9\ \text{years}}{9\ \text{years} \times 365\ \text{days}} = 0.41$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{0.01\ \text{mg/L} \times 1\ \text{L/day} \times 0.41}{35\ \text{kg}} = 0.00012\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{150\ \text{days/year} \times 9\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.05$$

$$\text{Cancer Effects Exposure Dose} = \frac{0.01\ \text{mg/L} \times 1\ \text{L/day} \times 0.05 \times 2}{35\ \text{kg}} = 0.00003\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00003 \times 0.54 = 0.000016$$

NOTES:

1. The EPA Chronic Oral RfD for PCE is 0.01 mg/kg/day.
2. The California EPA has calculated an oral cancer slope factor of 0.54 $(\text{mg/kg/d})^{-1}$.

Sodium in Drinking Water Fact Sheet

Bureau of Environmental Health
BEH

Is sodium found in drinking water?

Yes, sodium is found in naturally occurring element found in water and soil. Drinking water contributes only a small fraction (less than 10%) to the overall daily sodium intake which ranges from 115 to 750 milligrams per day (mg/d) for infants, 325 to 2700mg/d for children and 1100 to 3300 mg/d for adults.

The Massachusetts Department of Environmental Protection (MDEP) currently requires all water suppliers to notify the Massachusetts Department of Public Health/Bureau of Environmental Health (MDPH/BEH). MDEP, and local Boards of Health of the detected concentrations of sodium in drinking water. Notification is required so that individuals who are on a sodium restricted diet or wish to monitor their sodium intake for other reasons will have this information.

What is sodium's purpose?

Sodium is an essential mineral which is necessary for the normal functioning of the body and maintenance of body fluids. Nerve function and muscle contraction are also affected by sodium intake.

Where do we get sodium?

Sodium cannot be stored or manufactured in the body and must be consumed in some drinking water and in foods such as animal foods, low-fat dairy products, some canned foods, pickles, and olives.

What is the current guideline for sodium in drinking water and who should be concerned about this guideline?

The MDEP guideline of 20 milligrams of sodium per liter of water represents a level of sodium in water that physicians and sodium-sensitive individuals should be aware of in cases where sodium exposures are carefully controlled. People who have difficulty regulating fluid volume as a result of several diseases such as hypertension and kidney failure are particularly affected by elevated levels of sodium.

Hypertension is the medical name for high blood pressure and is a common chronic medical problem in the United States. It is responsible for a major portion of cardiovascular disease and stroke deaths. Reducing sodium intake not only prevents high blood pressure, but may also prevent heart disease.

Kidney failure occurs when an excess of sodium in the body causes fluid concentrations to change and the kidney fails to remove fluid. The result is a kidney shut-down and the build-up of fluid in the body which can lead to edema and hypertension.

How to Use this Factsheet

This risk factor summary was developed to serve as a general fact sheet. It is an overview and should not be considered exhaustive. For more information on other possible risk factors and health effects being researched, please see the References section.

A risk factor is anything that increases a person's chance of developing cancer. Some risk factors can be controlled while others cannot. Risk factors can include *hereditary conditions*, *medical conditions or treatments*, *infections*, *lifestyle factors*, or *environmental factors*. Although risk factors can influence the development of cancer, most do not directly cause cancer. An individual's risk for developing cancer may change over time due to many factors and it is likely that multiple risk factors influence the development of most cancers. Knowing the risk factors that apply to specific concerns and discussing them with your health care provider can help to make more informed lifestyle and health-care decisions.

For cancer types with environmentally-related risk factors, an important factor in evaluating cancer risk is the route of exposure. This is particularly relevant when considering exposures to chemicals in the environment. For example, a particular chemical may have the potential to cause cancer if an individual breathes the chemical in. That same chemical may not increase the risk of cancer similarly if an individual comes into contact with the chemical by touching it. In addition, an individual must generally be exposed to a chemical at a sufficient dose and for a sufficient duration of time for an adverse health effect to occur.

Gene-environment interactions are another important area of cancer research. An individual's risk of developing cancer may depend on a complex interaction between their genetic make-up and exposure to an environmental agent (for example, a virus or a chemical contaminant). This may explain why some individuals have a fairly low risk of developing cancer as a result of an environmental factor or exposure, while others may be more vulnerable.

Key Statistics

The American Cancer Society estimates 22,020 individuals will be diagnosed with malignant (cancerous) tumors of the brain or other nervous system (ONS) in the U.S. in 2010: 11,980 men and 10,040 women. These numbers would likely be much higher if non-malignant (non-cancerous) tumors were also included. In Massachusetts, incidence rates of brain and ONS cancers have generally remained steady from 2003 to 2007 among adults and children combined. Nationally, brain and ONS cancers are considered to be the second most common cancers in children. After a peak in childhood (generally under 10 years of age), the risk of brain and ONS cancers increases with age between 25 and 75 years.

Types of Brain and Other Nervous System Cancers

The term "cancer" is used to describe a variety of diseases associated with abnormal cell and tissue growth. Cancers are classified by the location in the body where the disease originated (the primary site) and the tissue or cell type of the cancer (histology).

Brain and ONS tumors can be either malignant or non-malignant, and in either case can be life threatening, although malignant tumors generally present greater concerns. In addition, the brain is a site where both primary and secondary malignant tumors can arise; secondary brain tumors generally originate elsewhere in the body and then metastasize, or spread, to the brain. Primary brain and ONS tumors consist of two main types: gliomas and meningiomas. Gliomas are a general classification of tumors that include a variety of types, named for the cells from which they arise: astrocytomas, oligodendrogliomas, and ependymomas. When considering only malignant brain and ONS tumors, approximately 80% are gliomas. Meningiomas, the most common ONS tumor in adults, arise from the meninges, which are tissues that surround the outer part of the spinal cord and brain. They account for about 33% of all (malignant and non-malignant) primary brain and ONS tumors reported in adults. Men are generally more likely to develop gliomas than women, while women are more likely to develop meningiomas. Furthermore, the incidence of gliomas is highest among white individuals, whereas the incidence of meningiomas is highest among African Americans. In addition to these main subtypes, there are a number of rare brain and ONS tumors.

Established Risk Factors

Most brain and ONS cancers develop for no apparent reason and are not associated with specific risk factors.

Hereditary Conditions

Rare cases of brain and ONS cancer run in some families. Brain tumors in some persons are associated with hereditary syndromes such as neurofibromatosis types I and II, Li-Fraumeni syndrome, and tuberous sclerosis. Neurofibromatosis type I (von Recklinghausen disease) is the most common inherited cause of brain or spinal cord tumors. Von Hippell-Lindau disease is associated with an inherited tendency to develop blood vessel tumors of the cerebellum. Overall, inherited syndromes that predispose individuals to brain tumors appear to be present in fewer than 5% of brain tumor patients.

Environmental Exposures

The most established risk factor for brain and ONS tumors (either non-malignant or malignant) is high-dose exposure to ionizing radiation (i.e., x-rays and gamma rays). Most radiation-induced brain and ONS tumors are caused by radiation to the head from the treatment of other cancers. These brain tumors usually develop around 10 to 15 years after the radiation. Meningiomas are the most common type of tumors that result from high-dose exposure to ionizing radiation, but tumors of other types have also occurred, including gliomas.

Possible Risk Factors

Medical Conditions

Head injury has long been suspected to be a possible risk factor for later development of brain and ONS tumors and continues to be studied by scientists. Of those studies that have found a positive association, head trauma was most strongly linked to the development of meningiomas compared to other types of brain and ONS tumors. Overall, additional research is necessary before a definitive link can be established.

Lifestyle Factors

The association between the development of brain and ONS cancers and N-nitroso compounds has been heavily researched. These compounds and their precursors, such as nitrite, are ubiquitous in our environment and have been found in tobacco smoke, cosmetics, automobile interiors, and cured meats. Several studies have concluded that an increased risk of pediatric brain tumors is associated with high levels of nitrite intake from maternal cured meat consumption during pregnancy. However, these studies have been criticized as many years have often passed between the mother's pregnancy and her interview, making recall less accurate.

Environmental Exposures

The National Cancer Institute reports that occupational exposure to radiation or certain chemicals has been associated with increased risk of brain cancer. As a result, workers in the nuclear industry, pathologists and embalmers who work with formaldehyde, workers who make plastics using vinyl chloride, and workers who make textile and plastics with acrylonitrile may have an increased risk of brain cancer. Exposures from working in synthetic rubber manufacturing or petroleum refining/production are also being investigated.

Other Risk Factors That Have Been Investigated

With cellular phones becoming increasingly common, there is growing concern over a link between their use and brain and ONS tumors. Cell phones emit radiofrequency radiation, a form of energy on the electromagnetic spectrum between FM radio waves and those used in microwave ovens. They do not emit ionizing radiation, which has been shown to damage DNA and has the ability to cause cancer. Several recent studies have found no excess risk between cell phone use and brain and ONS tumors. However, no studies have investigated the latent effects of long-term heavy use of cell phones due to their relatively recent widespread usage.

Many studies have been conducted to investigate links between brain and ONS cancers and environmental factors, including: residential power line exposure; viruses and

infections; aspartame (a sugar substitute), and pesticides. To date, however, there is no strong evidence to link these factors to brain tumors.

Brain and Other Nervous System Cancers in Children

Brain and ONS tumors are the second most common cancers in children and account for over 20% of malignant tumors diagnosed among children less than 20 years of age. Approximately 4,030 brain and ONS tumors are diagnosed each year in children under the age of 20, with about 25% of these considered non-malignant tumors. About half of all childhood brain tumors are astrocytomas and 20% are primitive neuroectodermal tumors (PNET). Medulloblastomas are PNETs that develop in the cerebellum whereas pineoblastomas are PNETs that develop in the pineal gland. The incidence rate of brain and ONS cancers in children has not changed significantly in recent years. In general, boys are at a slightly higher risk than girls for developing brain and ONS cancers. The vast majority of brain and ONS cancers in children occurs for no apparent reason and is not associated with any specific risk factors.

For More Information / References

Much of the information contained in this summary has been taken directly from the following sources. This material is provided for informational purposes only and should not be considered as medical advice. Persons with questions regarding a specific medical problem or condition should consult their physician.

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