



Data Brief: Response to Community Concerns Related to Vapor Intrusion in the Nonantum Area of Newton

Massachusetts Department of Public Health

OCTOBER 2019

The Massachusetts Department of Public Health, Bureau of Environmental Health (MDPH/BEH) conducted a screening-level review of the incidence of three types of cancer and the prevalence of congenital heart defects in response to community concerns about indoor air exposure to the chemical trichloroethylene (TCE) in the Nonantum area of Newton. These specific health outcomes were selected because studies show that they could be associated with TCE exposure.

This bulletin summarizes the incidence of kidney and renal pelvis cancer, liver and intrahepatic bile duct (IBD) cancer, and non-Hodgkin lymphoma (NHL) in one area of Newton (Census Tract 3732) using data collected by the Massachusetts Cancer Registry during three 10-year periods: 1984-1993, 1994-2003, and 2004-2013 as well as more recent years of 2014 and 2015. This bulletin also summarizes the prevalence of congenital heart defects in the same area of Newton using readily available data from the Massachusetts Birth Defects Monitoring Program during the period of 2000-2014 and provisional data from 2015 and 2016. These years constitute the most recent and complete data available at the initiation of this evaluation.

Summary of Findings

MDPH reviewed selected health outcomes that could be associated with TCE in response to community concerns about possible indoor air exposure.

- No unusual patterns were observed in the incidence of kidney and renal pelvis cancer, liver and IBD cancer, and NHL among residents of CT 3732 during 1984-2015.
- No unusual patterns were observed in the prevalence of congenital heart defects among live births or stillbirths to mothers residing in this area during 2000-2016.
- No diagnoses of these cancer types or congenital heart defects occurred among individuals living at residences within the MassDEP study area where TCE in indoor air was found above threshold levels.

Based on this screening-level review, MDPH recommends no further evaluation of health information.

Background

In 2014, the Massachusetts Department of Environmental Protection (MassDEP) began investigating groundwater contamination in the Nonantum area of Newton caused by TCE. MassDEP conducted indoor air sampling in over 160 nearby homes and buildings to determine if TCE evaporated from the groundwater and entered indoor air through seams and cracks in foundations (a process called vapor intrusion). In a small number of buildings (approximately 11) where contamination was found above threshold levels, MassDEP installed systems to prevent vapors from entering the buildings. The approximate area that MassDEP studied within CT 3732 is shown in yellow in Figure 1. See the MassDEP fact sheet [TCE Contamination, Nonantum Area of Newton, October 2017](#) for more details regarding the groundwater investigation.

Incidence of Three Types of Cancer in CT 3732

Kidney and Renal Pelvis Cancer

The incidence of kidney and renal pelvis cancer among males in CT 3732 was as expected during 1984-1993, statistically significantly elevated during 1994-2003 (10 observed versus 4 expected), and elevated but not statistically significantly during 2004-2013 (8 observed versus 5 expected). Among females, the incidence of kidney and renal pelvis cancer was as expected (within 1 or 2 diagnoses) during all three time periods (Table 1).

Table 1: Incidence of Kidney and Renal Pelvis Cancer in CT 3732 from 1984-2013

	1984-1993				1994-2003				2004-2013			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Males	3	3.0	NC	NC -- NC	10	3.8	266*	127--489	8	5.4	149	64--293
Females	1	2.1	NC	NC -- NC	3	2.8	NC	NC -- NC	5	3.5	141	45--329

Source: Massachusetts Cancer Registry, Office of Data Management and Outcomes Assessment, MDPH

Obs = Observed number of diagnoses
Exp = Expected number of diagnoses
SIR = Standardized Incidence Ratio

95% CI = 95% Confidence interval
* = Statistical significance
NC = Not calculated

About Kidney and Renal Pelvis Cancer

Kidney and renal pelvis cancer is more common among older people and males. The average age at diagnosis is 64 and it is very uncommon in people under the age of 45. Men are almost twice as likely to develop this type of cancer during their lifetime as women. Renal cell carcinoma (RCC) is by far the most common type of kidney and renal pelvis cancer and accounts for 90% of all diagnoses. Transitional cell carcinoma is another type that starts in the renal pelvis (where the kidney meets the ureter). Smoking is a major risk factor for kidney and renal pelvis cancer, and the risk seems related to how much is smoked. Additional established risk factors include family history, certain hereditary conditions, advanced kidney disease, and obesity. Possible risk factors may include high blood pressure and workplace exposures to substances such as cadmium (a type of metal), some herbicides, and organic solvents (like TCE). See the [American Cancer Society](#) website and the risk factor summary on [Massachusetts Environmental Public Health Tracking](#) for more information.

Consistent with national trends, the average age of the 18 males diagnosed with kidney and renal pelvis cancer in CT 3732 during 1994-2013 was 65 and none were younger than 45. Nearly 90% were diagnosed with the most common type called renal cell carcinoma (RCC). The remaining 2 males diagnosed with kidney and renal pelvis cancer during this time period had a type called transitional cell carcinoma and may have had a possible occupational exposure. Of the 16 males for whom tobacco use history was available, 9 reported using tobacco in the past or present, which could have been a contributing factor*.

An analysis of the residential address at the time of diagnosis did not reveal any unusual spatial or temporal patterns for those diagnosed with kidney and renal pelvis cancer in CT 3732 during the 30-year time period of 1984-2013.

*An evaluation of the tobacco use history information reported to the MCR indicates that the category of “never smoker” is less reliable than other reporting categories (such as current or former smoker). Many individuals are reported as never having smoked when, based on medical record reviews, they are individuals who are not current smokers but whose past tobacco use is unknown. These individuals should more accurately be reported as having an unknown tobacco use history rather than being categorized as never having used tobacco products. This misclassification is expected to result in an overestimate of “never smokers” and an underestimate of “former smokers” (MCR 2013).

The geographic distribution of diagnoses generally followed the pattern of population density with no unusual spatial clustering.

Liver and IBD Cancer

The incidence of liver and IBD cancer was less than or as expected (within 1 or 2 diagnoses) among males and females in CT 3732 during each 10-year time period from 1984-2013 (Table 2).

Table 2: Incidence of Liver and IBD Cancer in CT 3732 from 1984-2013

	1984-1993				1994-2003				2004-2013			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Males	0	0.8	NC	NC -- NC	0	1.6	NC	NC -- NC	3	3.0	NC	NC -- NC
Females	2	0.4	NC	NC -- NC	0	3.8	NC	NC -- NC	2	1.2	NC	NC -- NC

Source: Massachusetts Cancer Registry, Office of Data Management and Outcomes Assessment, MDPH

Obs = Observed number of diagnoses
 Exp = Expected number of diagnoses
 SIR = Standardized Incidence Ratio

95% CI = 95% Confidence interval
 * = Statistical significance
 NC = Not calculated

About Liver and Intrahepatic Bile Duct Cancer

Liver and IBD cancer is more common among men and older adults. Hepatocellular carcinoma is the most common type among adults, accounting for 80% of all diagnoses, while intrahepatic cholangiocarcinomas account for about 10-20%. Cirrhosis (a disease in which liver cells are damaged and replaced by scar tissue) is a major risk factor for the development of liver and IBD cancer. Most cirrhosis in the U.S. occurs as a result of long-term infection with the hepatitis B virus (HBV) or hepatitis C virus (HCV) or heavy alcohol consumption. Other conditions that may lead to cirrhosis include certain inherited metabolic diseases and autoimmune diseases. Additional established risk factors for liver and IBD cancer include tobacco use, obesity, long-term exposure to drinking water contaminated with arsenic, exposure to vinyl chloride (used in making some plastics and strictly regulated), and exposure to thorium dioxide, also called Thorotrast (a chemical injected into some patients in the past for certain x-ray tests but no longer used). Possible risk factors may include Type 2 diabetes, infection with a parasite called a liver fluke, long-term use of anabolic steroids, and TCE exposure. See the [American Cancer Society](http://www.americancancer.org) website and the risk factor summary on [Massachusetts Environmental Public Health Tracking](http://www.mass.gov/dep/ehp/ehp_tracking) for more information.

An analysis of the residential address at the time of diagnosis did not reveal any unusual spatial or temporal patterns for those diagnosed with liver and IBD cancer in CT 3732 during 1984-2013.

Non-Hodgkin Lymphoma

Among males in CT 3732, the incidence of NHL occurred less than or as expected (within 1 or 2 diagnoses) for all three 10-year time periods from 1984-2013. Among females in CT 3732, NHL occurred as expected during 1984-1993 and 2004-2013 but was statistically significantly elevated during 1994-2003 (11 observed versus 5.3 expected) (Table 3).

Table 3: Incidence of NHL in CT 3732 from 1984-2013

	1984-1993				1994-2003				2004-2013			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Males	3	3.9	NC	NC -- NC	2	5.1	NC	NC -- NC	5	6.0	84	27 -- 196
Females	6	4.1	148	54 -- 322	11	5.3	207*	103 -- 371	8	5.7	140	60 -- 276

Source: Massachusetts Cancer Registry, Office of Data Management and Outcomes Assessment, MDPH

Obs = Observed number of diagnoses
 Exp = Expected number of diagnoses
 SIR = Standardized Incidence Ratio

95% CI = 95% Confidence interval
 * = Statistical significance
 NC = Not calculated

About Non-Hodgkin Lymphoma (NHL)

NHL is a group of all lymphomas (cancers that occur in white blood cells) except Hodgkin lymphoma. Although most diagnoses of NHL occur in older adults in their 60s and older, it is one of the more common cancers among children, teens, and young adults. In the United States, B-cell lymphomas account for 85% of all NHL diagnoses and T-cell lymphomas account for less than 15%. People with weakened immune systems have a higher risk of developing NHL. These include individuals taking immunosuppressant drugs following an organ transplant, those with inherited immunodeficiency syndromes, and individuals infected with human immunodeficiency virus (HIV). Other established risk factors include infection with the Epstein-Barr virus (EBV), some autoimmune disorders such as rheumatoid arthritis and systemic lupus erythematosus (SLE, or lupus), and certain chemotherapy drugs used to treat other cancers. Possible risk factors include workplace exposures to chemicals such as benzene, TCE, and some herbicides and insecticides. See the [American Cancer Society](#) website and the risk factor summary on [Massachusetts Environmental Public Health Tracking](#) for more information.

Of the 11 females in CT 3732 diagnosed with NHL during 1994-2003, the ages at the time of diagnosis and type of NHL follow what would be expected based on national trends. The average age at the time of diagnosis was 65 and nearly 75% occurred in older adults age 60 and over. Of the 7 females for whom a specific type of NHL was reported, nearly all consisted of B-cell lymphomas. Although almost half of the females diagnosed during this time period had a previous cancer diagnosis, it is not possible to determine if they received chemotherapy for their previous cancer (a possible risk factor for NHL) using readily available information from the MCR.

An analysis of the residential address at the time of diagnosis did not reveal any unusual spatial or temporal patterns for those diagnosed with NHL in CT 3732 during the 30-year time period of 1984-2013. The geographic distribution of diagnoses generally followed the pattern of population density with no unusual spatial clustering.

Update of Cancer Incidence Data in CT 3732

Since the initiation of this evaluation, the MCR released two additional years of cancer incidence data for 2014 and 2015. During 2014 and 2015, three diagnoses of kidney and renal pelvis cancer, one diagnosis of liver and IBD cancer, and one diagnosis of NHL occurred among residents of CT 3732. In general, the ages at the time of diagnosis and histology for each of the three cancer types followed what would be expected based on national statistics and the epidemiological literature. The geographic distribution of residential address at the time of diagnosis did not reveal any unusual patterns.

Prevalence of Congenital Heart Defects in CT 3732

Fewer than 6 cases of congenital heart defects were identified among live births to mothers residing in CT 3732 during 2000-2014. To protect the privacy of those individuals diagnosed with birth defects and their parents and families, counts are suppressed and prevalence rates are not calculated when fewer than six diagnoses are observed in a geographic area. Statewide, the prevalence rate for congenital heart defects was 54 per 10,000 live births over this 15-year time period. No congenital heart defects were identified among stillbirths in CT 3732 during this time period.

Update of Congenital Heart Defects in CT 3732

A review of more recent provisional data from 2015 and 2016 did not reveal any diagnoses of congenital heart defects among live births or stillbirths to mothers residing in CT 3732.

DISCUSSION

Many cancers occur because of changes to cells that happen by random chance. These are called sporadic or spontaneous mutations and are not due to any particular exposure to a cancer-causing agent (i.e., carcinogen). Other times, exposure may be an initiating or contributing factor to the development of cancer in an individual. The latency period is the time interval between an initiating event (such as a random cellular mutation or exposure to a carcinogen) and the appearance of symptoms of the disease or its diagnosis. Cancer, in general, has a long latency period but it may vary depending on the type, magnitude, and timing of the exposure. Cancers that are solid tumors are believed to have a long latency period, estimated to be no shorter than 10 years and possibly as long as 50 years or more. For hematopoietic or blood-related cancers, such as NHL, experts think that the general latency period may be shorter, most commonly on the order of 5 to 10 years (Hall 2006; NRC 2005; UNSCEAR 2000; Bang 1996; Frumkin 1995). Due to the long latency period for most types of cancer, it is difficult to identify exposures that may have contributed to an individual's cancer development.

A risk factor is anything that increases a person's chance of developing cancer and can include hereditary conditions, medical conditions or treatments, infections, lifestyle factors, or environmental exposures. It is likely that multiple risk factors influence the development of most cancers. In addition, an individual's risk of developing cancer may change over time and may depend on a complex interaction between their genetic makeup and exposure to a cancer-causing agent.

Available information reported to the MCR related to risk factors for cancer development was reviewed for residents of CT 3732 who were diagnosed with a cancer type that could be associated with TCE during 1984-2013. This information is collected for each individual at the time of diagnosis and may include the individual's age, history of tobacco use, and occupation. However, information about personal risk factors such as family history, medical conditions, diet, and other factors that may also influence the development of cancer is not collected by the MCR. Therefore, it was not possible to consider their contributions to cancer development in this evaluation.

This screening-level review of cancer incidence data was used to evaluate the pattern of cancer in a geographic context and determine whether further public health investigations or actions may be warranted. This descriptive analysis of cancer incidence data alone cannot be used to establish a causal link between a particular risk factor (either environmental or non-environmental) and the development of cancer. It also cannot determine the cause of cancer in any one particular individual. A review of the geographic distribution of addresses at the time of diagnosis was conducted for all three cancer types during the 30-year time period of 1984-2013. Place of residence at the time of diagnosis was also compared with TCE concentrations in groundwater at the same location. Very few diagnoses occurred among individuals at residences located within areas of elevated TCE levels in groundwater as measured

since 2014. It should be noted, however, that TCE levels in groundwater prior to 2014 are unknown. The geographic extent of contamination in the past is likely to have been the same or smaller. Historical concentrations in shallow groundwater are unknown and may have changed over time. It should also be noted that residential history prior to the time of diagnosis is unknown.

Similarly, with regard to the prevalence of congenital heart defects in CT 3732, it is important to note that census tract of mother's residence at the time of birth is a relatively poor proxy for exposure to TCE since the mother may have moved during pregnancy and/or the indoor air within the home may or may not have been contaminated.

CONCLUSIONS

The incidence of kidney and renal pelvis cancer, liver and IBD cancer, and NHL in CT 3732 during the 10-year time periods of 1984-1993, 1994-2003, and 2004-2013 was either less than or as expected based on the statewide experience except for the following elevations, two of which were statistically significant:

- Kidney and renal pelvis cancer was statistically significantly elevated among males during 1994-2003 with 10 observed diagnoses compared to 4 that would be expected.
- Kidney and renal pelvis cancer was elevated (but not statistically significant) among males during 2004-2013 with 8 observed diagnoses compared to 5 that would be expected.
- NHL was statistically significantly elevated among females during 1994-2003 with 11 observed diagnoses compared to 5 that would be expected.

Statistical significance does not necessarily imply public health significance. Determination of statistical significance is just one tool used to interpret SIRs. Closer examination of risk factor information for each of these three elevations as well as the spatial and temporal distributions did not reveal any unusual patterns or suggest that a common environmental factor played a primary role in these cancer diagnoses. It is possible that tobacco use may have been a contributing factor for some of the males diagnosed with kidney and renal pelvis cancer during the time periods for which it was elevated. Similarly, treatment for a previous cancer diagnosis may have been a contributing factor for some of the females diagnosed with NHL during 1994-2003.

It is important to note that the majority of the diagnoses of these three cancer types that occurred in CT 3732 during 1984-2015 were among individuals living outside the MassDEP study area. Of those who were within the MassDEP study area, about 1/3rd were at residences located within areas of elevated TCE levels in groundwater as measured since 2014 and none were living at residences where TCE in indoor air was found above threshold levels.

Fewer than 6 cases of congenital heart defects were identified in CT 3732 during 2000-2014 and provisional data for 2015 and 2016 did not reveal any congenital heart defects. To protect patient privacy, the exact count was suppressed and the prevalence rate was not calculated. It is important to note that no cases occurred within the MassDEP study area.

Based on the findings of this screening-level review of selected cancer incidence data and congenital heart defect prevalence data, MDPH recommends no further evaluation of health information.

DATA SOURCES AND METHOD NOTES

Data Sources:

Massachusetts Cancer Registry, Office of Data Management and Outcomes Assessment, MDPH.

Massachusetts Center for Birth Defects Research and Prevention, Bureau of Family Health and Nutrition, MDPH.

Method Notes for Cancer Incidence:

All new diagnoses of invasive cancer, as well as certain in situ (localized) cancers, 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). Individuals diagnosed with cancer were selected for inclusion based on the residential address provided to the hospital or reporting medical facility at the time of diagnosis. The year 2013 was the most recent year for which complete cancer incidence data were available at the initiation of this analysis. An SIR is the ratio of the observed number of cancer diagnoses in an area to the expected number of diagnoses, multiplied by 100. Age-specific statewide incidence rates were applied to the population distribution of CT 3732 to calculate the number of expected cancer diagnoses. It is standard MCR policy not to calculate rates with fewer than five observed diagnoses due to instability. The statistical significance of an SIR is assessed by calculating a 95% confidence interval (CI) to determine if the observed number of diagnoses is “statistically significantly different” from the expected number or if the difference may be due solely to chance. If the 95% CI range does not include the value 100, then the study population is significantly different from the comparison or “normal” population. “Significantly different” means there is less than a 5% percent chance that the observed difference (either increase or decrease) is the result of random fluctuation in the number of observed cancer diagnoses. The MDPH is bound by state and federal patient privacy and research laws not to make public the names or any 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).

Method Notes for Congenital Heart Defects Prevalence:

All birth defect diagnoses are required by law to be reported to the Massachusetts Birth Defects Monitoring Program (MBDMP) within 30 days of the date of diagnosis (M.G.L. c.111.s67E). Birth defect data are collected when all of the following criteria are met: the infant was live born or the fetus was stillborn with a gestational age greater than or equal to 20 weeks or with a weight of at least 350 grams; the infant or fetus had a birth defect that met diagnostic criteria; the diagnosis was made before the infant reached one year of age; and the infant’s mother had a permanent address in Massachusetts at the time of delivery. The time period 2000-2014 constitutes the period for which the most recent and complete birth defects prevalence data were available at the initiation of this analysis. Birth defects prevalence data for the years 2015 and 2016 are provisional and subject to revision. Census tract of residence at the time of delivery were obtained from vital records data (i.e., birth and fetal death certificates). For the purposes of this evaluation, congenital heart defects were identified using ICD-9-CM/BPA[†] birth defect codes in the range of 745.000-747.880, excluding patent ductus arteriosus and patent foramen ovale. The MDPH is bound by state and federal patient privacy and research laws not to make public the names or any information (e.g., place of residence) that could personally identify individuals whose child was diagnosed with a birth defect that was reported to the MBDMP (M.G.L. c.111. s. 24A).

REFERENCES

- Agency for Toxic Substances and Disease Registry. 2014. Toxicological Profile for Trichloroethylene (TCE) (Draft for Public Comment). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available at: <https://www.atsdr.cdc.gov/toxprofiles/tp19.pdf>
- American Cancer Society. 2018a. Kidney Cancer. Available at <https://www.cancer.org/cancer/kidney-cancer.html>.
- American Cancer Society. 2018b. Liver Cancer. Available at <https://www.cancer.org/cancer/liver-cancer.html>.
- American Cancer Society. 2018c. Non-Hodgkin Lymphoma (Adults). Available at <https://www.cancer.org/cancer/non-hodgkin-lymphoma.html>
- Bang KM. 1996. Epidemiology of Occupational Cancer. *Occupational Medicine*. 11(3):467-485.

[†] International Classification of Diseases, 9th Revision, Centers for Disease Control, Clinical Modification/ British Pediatric Association

Frumkin H. 1995. Carcinogens. In: Levy BS and Wegman DH, editors. Occupational Health- Recognizing and Preventing Work-Related Disease. 3rd ed. Boston: Little, Brown and Company. p.293

Hall EJ. 2006. Radiobiology for the radiologist. 6th ed. Philadelphia: Lippincott Williams & Wilkins; p. 138

Massachusetts Cancer Registry. 2013. "CDC/CER Innovative Uses of Cancer Registries for Public Health Applications: Primary Payer at Diagnosis Data Quality Evaluation for Invasive Colorectal and Breast Cancer Cases in the Massachusetts Cancer Registry (MCR) from 2005 to 2009." MCR Fall Educational Workshop. December 5, 2013.

National Research Council (NRC). 2005. Health risks from exposure to low levels of ionizing radiation. BEIR VII Phase 2. Washington, DC: National Academies Press.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2000. Sources and Effects of Ionizing Radiation. Volume I. New York: United Nations Scientific Committee on the Effects of Atomic Radiation.

RESOURCES

For information on this bulletin or other environmental health concerns:

MDPH Bureau of Environmental Health
250 Washington Street, 7th Floor
Boston, MA 02108
Tel. (617) 624-5757

www.mass.gov/dph/environmental_health

For information on the TCE groundwater investigation or indoor air testing:

MassDEP Northeast Regional Office
205B Lowell Street
Wilmington, MA 01887
Tel. (978) 694-3200

<https://www.mass.gov/locations/massdep-northeast-regional-office>

For additional cancer incidence or birth defect prevalence data:

Massachusetts Environmental Public Health Tracking
250 Washington Street, 7th Floor
Boston, MA 02108
Tel. (800) 319-3042

www.mass.gov/dph/matracking

This publication was made possible by Grant Number [6 NU61TS000276-03-01] from the Agency for Toxic Substances and Disease Registry. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the Agency for Toxic Substances and Disease Registry, or the Department of Health and Human Services.