

Massachusetts  
Department  
Of  
Public Health



**Evaluation of Brain and Central  
Nervous System Cancer Incidence  
in Arlington, Massachusetts**

**1982-2001**

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## **I. INTRODUCTION**

At the request of concerned residents and the Director of Public Health in Arlington, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Center for Environmental Health, conducted an evaluation of brain and other central nervous system (CNS) cancers for the town of Arlington. This evaluation was initiated because of community concerns about a suspected increase in the incidence of cancer among individuals living in the neighborhood surrounding the former Reeds Brook Landfill (located in the northwest corner of the town) and the incidence of brain and CNS cancers town-wide.

This investigation provides a review of the pattern of brain and CNS cancers in the town of Arlington and compares their incidence with the incidence of this cancer type in the state of Massachusetts as a whole. Available information about risk factors, including environmental factors, related to the development of this cancer type was also evaluated. The town of Arlington is divided into eight smaller geographic areas or census tracts (CTs). Cancer incidence rates were evaluated for each census tract separately as well as for the town as a whole. Cancer incidence data for Arlington were obtained from the Massachusetts Cancer Registry (MCR) for the years 1982 – 2001, the time period for which the most recent and complete data were available at the initiation of this analysis. Three smaller time periods were evaluated, 1982 – 1988, 1989 – 1995 and 1996 – 2001, to assess possible trends over time.

In addition to calculation of cancer incidence rates, a qualitative analysis of the geographic distribution of individuals diagnosed with brain and CNS cancers was conducted by “mapping” their residence at time of diagnosis. This was done to determine if any unique geographic patterns existed in a particular area of town.

Finally, to further address resident concerns about the pattern of cancer near the former Reeds Brook Landfill and specific reports of individual diagnoses in the adjacent neighborhood, a qualitative evaluation of all cancer types was conducted for the streets immediately surrounding the landfill.

## **II. METHODS FOR ANALYZING CANCER INCIDENCE**

### **A. Case Identification/Definition**

Cancer incidence data (i.e., reports of new cancer diagnoses) for the years 1982 – 2001 were obtained for the town of Arlington from the Massachusetts Cancer Registry (MCR), a division of the Center for Health Information, Statistics, Research and Evaluation within the MDPH. Brain and CNS cancers were evaluated in this investigation. [Coding for this cancer type in this report follows the International Classification of Diseases for Oncology (ICD-O) system. See Appendix A for the incidence coding definitions used in this report for this cancer type.] As mentioned above, this cancer type was selected for evaluation based on concerns from residents of the community. Only individuals reported to the MCR as having a diagnosis of a primary brain or CNS cancer and being a resident of Arlington were included in the analysis. Cases were selected for inclusion based on the address reported to the hospital or reporting medical facility at the time of diagnosis (last updated April 6, 2004).

The MCR is a population-based surveillance system that began collecting information on Massachusetts residents diagnosed with cancer in the state in 1982. All newly diagnosed cancer cases 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). This information is kept in a confidential database. Data are collected on a daily basis and are reviewed for accuracy and completeness on an annual basis. This process corrects misclassification of data (i.e., city/town misassignment). Once these steps are finished, the data for that year are considered “complete.” Due to the volume of information received by the MCR, the large number of reporting facilities, and the six-month period between diagnosis and required reporting, the most current registry data that are complete will inherently be a minimum of two years prior to the current date. The 20-year period 1982 – 2001 constitutes the period for which the most recent and complete cancer incidence data were available from the MCR at the time of this analysis.

The term "cancer" is used to describe a variety of diseases associated with abnormal cell and tissue growth. Epidemiologic studies have revealed that different types of cancer are individual diseases with separate causes, risk factors, characteristics and patterns of survival (Berg 1996). 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). Cancers that occur as the result of the metastasis or the spread of a primary site cancer to another location in the body are not considered as separate cancers and therefore were not included in this analysis.

### **B. Calculation of Standardized Incidence Ratios (SIRs)**

To determine whether elevated numbers of brain and CNS cancer diagnoses occurred in Arlington, cancer incidence data were tabulated by gender according to eighteen age groups to compare the observed number of cancer diagnoses to the number that would be expected based on the statewide cancer rate. Standardized incidence ratios (SIRs) were then calculated for the period 1982 – 2001 for Arlington as a whole as well as for each census tract (CT) within Arlington. SIRs were also calculated for three smaller time periods, 1982 – 1988, 1989 – 1995 and 1996 – 2001, in order to evaluate patterns or trends in cancer incidence over time.

In order to calculate SIRs, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated based on 1980, 1990, and 2000 U.S. census data for each CT in Arlington and for the town as a whole (U.S. DOC 1980, 1990, and 2000). Midpoint population estimates were calculated for each time period evaluated (i.e., 1985, 1992 and 1999). To estimate the population between census years, an assumption was made that the change in population occurred at a constant rate throughout the ten-year interval between each census.<sup>1</sup>

Because accurate age group and gender-specific population data are required to calculate SIRs, the CT is the smallest geographic area for which cancer rates can be accurately calculated. Specifically, a CT is a smaller statistical subdivision of a county as defined

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<sup>1</sup> Using slightly different population estimates or statistical methodologies, such as grouping ages differently or rounding off numbers at different points during calculations, may produce results slightly different from those published in this report.

by the U.S. Census Bureau. CTs usually contain between 2,500 and 8,000 persons and are designed to be homogenous with respect to population characteristics (U.S. DOC 1990). According to the 1990 U.S. Census, the town of Arlington was subdivided into seven census tracts (i.e., CTs 3561 – 3567). During the 1990 U.S. Census, the Census Bureau further divided one Arlington CT (3566) into two, resulting in CTs 3566.01 and 3566.02. The split in this census tract produced a total of eight census tracts in Arlington (CT 3561-3565, 3566.01, 3566.02 and 3567). In order to evaluate cancer incidence by census tract over time, population data for the split CT were combined for the year 2000 to remain consistent with the 1980 and 1990 population data and CT designations. Therefore, for the purpose of this evaluation SIRs were calculated according to the 1990 census tract designations for seven Arlington census tracts (CT 3561-3567). The town boundaries and census tract locations for Arlington are illustrated in Figure 1.

### **C. Interpretation of a Standardized Incidence Ratio (SIR)**

An SIR is an estimate of the occurrence of cancer in a population relative to what might be expected if the population had the same cancer experience as a larger comparison population designated as "normal" or average. Usually, the state as a whole is selected to be the comparison population. Using the state of Massachusetts as a comparison population provides a stable population base for the calculation of incidence rates.

Specifically, an SIR is the ratio of the observed number of cancer cases in an area to the expected number of cases multiplied by 100. The population structure of each town is adjusted to the statewide incidence rate to calculate the number of expected cancer cases. The SIR is a comparison of the number of cases in the specific area (i.e., city/town or census tract) to the statewide rate. Comparisons of SIRs between towns or census tracts are not possible because each community has different population characteristics.

An SIR of 100 indicates that the number of cancer cases observed in the population being evaluated is equal to the number of cancer cases expected in the comparison or "normal" population. An SIR greater than 100 indicates that more cancer cases occurred than were expected, and an SIR less than 100 indicates that fewer cancer cases occurred than were

expected. Accordingly, an SIR of 150 is interpreted as 50% more cancer cases than the expected number; an SIR of 90 indicates 10% fewer cancer cases than expected.

Caution should be exercised, however, when interpreting an SIR. The interpretation of an SIR depends on both the size and the stability of the SIR. Two SIRs can have the same size but not the same stability. For example, an SIR of 150 based on four expected cases and six observed cases indicates a 50% excess in cancer, but the excess is actually only two cases. Conversely, an SIR of 150 based on 400 expected cases and 600 observed cases represents the same 50% excess in cancer, but because the SIR is based upon a greater number of cases, the estimate is more stable. It is very unlikely that 200 excess cases of cancer would occur by chance alone. As a result of the instability of incidence rates based on small numbers of cases, SIRs were not calculated when fewer than five cases were observed for a particular cancer type.

#### **D. Calculation of the 95% Confidence Interval**

To help interpret or measure the stability of an SIR, the statistical significance of each SIR was assessed by calculating a 95% confidence interval (95% CI) to determine if the observed number of cases is “significantly different” from the expected number or if the difference may be due solely to chance (Rothman and Boice 1982). Specifically, a 95% CI is the range of estimated SIR values that have a 95% probability of including the true SIR for the population. 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% chance that the observed difference (either increase or decrease) is the result of random fluctuation in the number of observed cancer cases.

For example, if a confidence interval does not include 100 and the interval is above 100 (e.g., 105–130), there is a statistically significant excess in the number of cancer cases. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45–96), the number of cancer cases is statistically significantly lower than expected. If the confidence interval range includes 100, the true SIR may be 100. In this case, it cannot be determined with certainty that the difference between the observed and



expected number of cases reflects a real cancer increase or decrease or is the result of chance. It is important to note that statistical significance does not necessarily imply public health significance. Determination of statistical significance is just one tool used to interpret SIRs.

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval, such as 103–115, allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval, for instance 85–450, leaves considerable doubt about the true SIR, which could be much lower than or much higher than the calculated SIR. This would indicate an unstable statistic. Again, due to the instability of incidence rates based on small numbers of cases, statistical significance was not assessed when fewer than five cases were observed.

#### **E. Evaluation of Cancer Risk Factor Information**

Available information reported to the MCR related to risk factors for cancer development was reviewed and compared to known or established incidence patterns for brain and CNS cancers. This information is collected for each individual at the time of cancer diagnosis and includes age at diagnosis, stage of disease, previous cancer diagnoses and occupation. One or even several factors acting over time can be related to the development of cancer. However, information about personal risk factors such as family history, hormonal events, diet, and other factors that may also influence the development of cancer is not collected by the MCR, and therefore, it was not possible to evaluate them in this investigation.

#### **F. Determination of Geographic Distribution of Cancer Cases**

In addition to calculation of SIRs, address at the time of diagnosis for each individual diagnosed with brain or CNS cancer was “mapped” using a computerized geographic information system (GIS) (ESRI 2002). This allowed assignment of census tract location as well as an evaluation of the spatial distribution of individuals at a smaller geographic level (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cancer diagnoses. In instances where the address

information from the MCR was incomplete (i.e., did not include specific streets or street numbers), efforts were made to research those cases using telephone books issued within two years of an individual's diagnosis. For confidentiality reasons, it is not possible to include maps showing the locations of individuals diagnosed with cancer in this report. [Note: MDPH is bound by law not to reveal the name or identifying information of an individual diagnosed with cancer and reported to the MCR.]

### **G. Cancer Incidence in the Reeds Brook Landfill Neighborhood**

Specific information on several Arlington residents diagnosed with cancer was provided to the MDPH from area residents along with concerns about the former Reeds Brook Landfill. Although it is not possible to calculate incidence rates beyond the year 2001 (i.e., the year for which complete data were available at the time of data analysis), the MCR is a continual surveillance system for cancer and it is possible to review individual case reports for more recent years (i.e., 2002 – present). Therefore, to address specific community concerns about a suspected increase in cancer incidence in neighborhoods in close proximity to the former Reeds Brook Landfill site, MCR data files were reviewed for residents who had been diagnosed with any cancer type from 1982 to the present whose residence was reported on streets in close proximity to the Reeds Brook Landfill (last updated October 6, 2005).<sup>2</sup> Specifically, to determine whether any cancer type appeared to be concentrated within this area of Arlington, place of residence at the time of diagnosis was “mapped” and evaluated for all individuals diagnosed with cancer in the area that is bordered to the east by Wright Street, to the south by Summer Street, to the west by the Lexington town line and to the north by the Winchester town line (see Figures 1 and 2). As previously stated, for confidentiality reasons, maps of the location of individuals diagnosed with cancer cannot be provided in this report.

## **III. FORMER REEDS BROOK LANDFILL**

The Reeds Brook property, formerly known as the Reeds Brook and Summer Street Landfill, is a 20-acre parcel of land located on Summer Street in the northwest section of

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The data summarized in this section are drawn from data entered on MCR computer files before October 6<sup>th</sup>, 2005. The numbers presented in this report may change slightly in future reports, reflecting late reported cases, address corrections, or other changes based on subsequent details from reporting facilities.

Arlington in census tract (CT) 3565. The site was operated as a municipal sanitary landfill from approximately 1950 through 1969. The landfill accepted household waste including garbage, paper, metal, wood, rubber, and plastics. Some evidence, however, of the illegal dumping of construction debris, appliances, and yard waste within limited areas of the site has been documented (Metcalf & Eddy, 1996). During this time, the property was privately owned but the landfill was operated by the town. In the mid-1990s, the town bought the property to facilitate proper closure of the landfill and future development of the property.

For many years, residents living in the neighborhoods adjacent to Reeds Brook Landfill were reportedly faced with significant flooding of their homes during storms (Arlington Online, 2005). Beginning in 1993, several site investigations were undertaken to assess the impact of the landfill on groundwater, soil, sediment, and air quality and to plan for closure of the landfill. These comprehensive site assessments resulted in a two-phase site remediation and redevelopment project. The first phase involved the construction of a storm drainage system on the property, to improve site drainage, which includes a storm water detention basin covering approximately four acres of the site. In addition, the site topography was totally reconfigured and the entire property was covered with three feet of clean soil. The second phase, redevelopment, is essentially complete with the construction of a park onsite that includes athletic fields and other recreational uses (O'Brien, K., 2005). The identification, assessment, and remediation of such contamination are regulated by the Massachusetts Department of Environmental Protection (MDEP).

#### **IV. RESULTS OF CANCER INCIDENCE ANALYSIS**

The following sections present cancer incidence rates for Arlington and its individual census tracts during the 20-year time period 1982 – 2001. These data were analyzed by three time periods, 1982 – 1988, 1989 – 1995 and 1996 – 2001. SIRs were not calculated for some time periods and/or census tracts due to the small number of observed cases (less than five). However, the expected number of cases was calculated during each time period, and the observed and expected numbers of cases were compared to determine whether excess numbers of cancer cases were occurring.

### **A. Brain and CNS Cancer Incidence in Arlington**

In the town of Arlington as a whole, the incidence of brain and CNS cancer among males occurred about as expected when compared to the statewide brain/CNS cancer experience for each of the three time periods. Women were diagnosed slightly more often than expected in each of the three time periods, 1982 – 1988, 1989 – 1995, and 1996 – 2001 (see Table 1). None of these elevations were statistically significant.

### **B. Cancer Incidence in Arlington Census Tracts**

The majority of CTs experienced brain or CNS cancer incidence at or below the expected when compared to the state during each of the three time periods evaluated (see Tables 2 – 4). Elevations were noted however in some CTs during the three different time periods. During the 1989 – 1995 time period, a statistically significant elevation occurred among females in CT 3562 (6 diagnoses observed vs. 1.9 expected, SIR = 313, 95% CI 114-682). In the subsequent time period, between 1996 and 2001, among males and females combined in CT 3562, four individuals were diagnosed with cancer versus 2.2 expected. However, this elevation was not statistically significant.

During the most recent time period in CT 3561, a statistically significant elevation occurred among men and women combined (5 diagnoses observed vs. 1.4 expected, SIR = 361, 95% CI 116-844). In the two earlier time periods, cancer occurred less often than expected among males and females combined in CT 3561. In CT 3564 there was an elevation among males and females combined during the earliest time period, 9 observed versus 5.2 expected. This elevation was not statistically significant. Slight elevations occurred in CT 3564 during the subsequent two time periods evaluated. From 1989 - 1995, seven individuals were diagnosed with brain or CNS cancer while 5.3 were expected. This elevation was due to an elevation among men (6 observed versus 2.7 expected) and was not statistically significant. During the most recent time period, 1996 – 2001, the incidence of brain and CNS cancer occurred about as expected. In CT 3565, the census tract in which the former Reeds Brook Landfill is located, the incidence for all three time periods was about as expected. See Tables 2-4 for a summary of the results for each CT in Arlington by time period.

### **C. Review of Diagnosis Information**

As previously mentioned, cancer is not just one disease, but is a term used to describe more than 100 different diseases. As such, studies have generally shown that different cancer types have different causes, patterns of incidence, risk factors, latency periods (the time between exposure and development of disease), characteristics, and trends in survival. In order to better understand the patterns of brain and CNS cancer incidence in Arlington, factors that are thought to contribute to an individual's risk for developing these types of cancer were evaluated. Risk factors reviewed include gender, age, histology, previous cancer diagnosis and occupation. These data are collected by the MCR and were reviewed for Arlington residents diagnosed with brain and CNS cancer. However, risk factors such as heredity, consumption of nitrites and other N-nitroso compounds found in some foods (e.g., cured meats), as well as exposure to ionizing radiation, electromagnetic fields, and some occupational exposures (e.g., lead) which have been associated with an increased risk of developing brain or CNS cancer in some studies, could not be evaluated because information related to these factors for individuals with brain or CNS cancer in Arlington was not available. Therefore, the extent of the role these factors may have played in the incidence of brain and CNS cancer in Arlington is unclear. For more information regarding potential risk factors for brain and CNS cancer, see Appendix B.

#### *1. Age and Gender*

Brain and CNS cancers are slightly more common among males and, after a peak in childhood (which occurs under age 10), risk declines slightly until around age 25, and increases consistently with age thereafter (Preston-Martin & Mack 1996). From 1982 to 2001, males and females statewide were diagnosed with brain and CNS cancer about equally. A review of gender patterns among brain and CNS cancer cases in Arlington revealed that slightly more females than males were diagnosed with brain and CNS cancer during that time period (57% vs. 43%, respectively). The ages at diagnosis for individuals with brain or CNS cancer in Arlington were consistent with the pattern expected based on the scientific literature for this cancer type. All but two of the individuals diagnosed with brain or CNS cancer were diagnosed prior to age 10 or after

age 25. During the 20 year period from 1982 – 2001, seven children ages 19 or younger were diagnosed with brain or CNS cancer while approximately five would have been expected. Brain and CNS cancers are the most common solid tumors diagnosed among children and the second most common childhood cancer. The average age of diagnosis among adults, 20 years of age and older, living in Arlington diagnosed with brain or CNS cancer between 1982 and 2001 was approximately 62 years old, which is slightly older than the state average of 59 for the same time period.

In CT 3562, the census tract in which there was a statistically significant elevation among females during the 1989 – 1995 time period, all of the individuals diagnosed were aged 25 and older. One individual was diagnosed at age 27 and the remainder of the individuals were over 70 years old when diagnosed. No children were diagnosed with brain or CNS cancer in this CT during this time period and the average age at diagnosis, for individuals 20 years of age and older, was approximately 66 years. All of the individuals diagnosed in this CT during the 1989 – 1995 time period were females.

As mentioned above, there was also a statistically significant elevation among males and females combined in CT 3561 in the most recent time period evaluated. The average age at diagnosis for these individuals was approximately 52 years of age and none of the individuals diagnosed were children. Additionally, of the five individuals diagnosed with brain or CNS cancer in CT 3561 between 1996 and 2001, three were males and two were females.

## *2. Occupation*

Research has shown that some occupational exposures may increase the risk of developing brain and CNS cancer among individuals employed in such industries as plastics manufacturing, aspartame (or artificial sweetener) manufacturing, electrical work, veterinary or agricultural work, as well as among some workers in the nuclear and rubber industries, in certain health professions, and in individuals who have an occupational exposure to lead. Among the 92 adults in Arlington diagnosed with brain or CNS cancer during the 20 year period evaluated, an occupation was reported for 50 individuals. Of these, nine individuals reported an occupation where possible exposures

to radiation or employment in one of the industries listed above may have occurred, based on the available information. Occupation was reviewed for the individuals in the two census tracts with statistically significant elevations. In CT 3562 during the 1989 – 1995 time period, occupational data were reported for three of the six individuals diagnosed with brain and CNS cancer. For one of the three individuals, an occupation was reported where possible exposures consistent with the literature may have occurred in the workplace. In CT 3561 during the 1996 – 2001 time period, occupation was reported for three of the five individuals and none of these individuals reported an occupation where a possible exposure was likely.

Therefore, although the information reviewed suggests the possibility that occupational exposures may have contributed to the development of brain or CNS cancer among some individuals, it is difficult to determine what role occupational exposures played in the incidence of brain or CNS cancer in Arlington overall. Occupation was not reported for 46% of the individuals (42 out of 92) diagnosed with brain or CNS cancer over the 20-year time period evaluated.

### *3. Previous Cancer Diagnosis*

Patients who are treated with radiation therapy to the head and/or neck area for other cancers have a higher risk of developing brain cancer later in life. Review of data from the MCR identified seven individuals (8%) diagnosed with brain and CNS cancers in Arlington during 1982 – 2001 who had a previous cancer diagnosis reported to the MCR. These patients may have received treatment for their initial cancer that could have contributed to their subsequent brain or CNS cancer diagnosis. However, because medical records were not readily available, it is not possible to determine whether these individuals actually received radiation therapy for their previous cancer.

### *4. Histology*

In general, each diagnosis of cancer is classified by its histology, which is the tissue or cell type from which the cancer originates. In the brain most tumors develop from glial cells. There are four different types of glial cells: astrocytes, oligodendrocytes, ependymal cells and microglia. Each type of cell has a particular role supporting and/or

protecting the neurons in the brain. Tumors of the glial cells are called gliomas, which are a general classification of malignant tumors that include three main sub-types: astrocytomas, oligodendrogliomas, and ependymomas. In addition to these sub-types of brain cancer, there are a number of less common sub-types, including medulloblastomas, meningiomas, and primitive neuroectodermal tumors (PNET).

The distribution of brain and CNS cancers by cell type was reviewed for each individual diagnosed with brain and CNS cancer in Arlington. Patterns of disease were compared to known or established incidence trends to assess whether any unexpected patterns exist. In adults, the most frequent types of brain tumors are astrocytomas and about half of all childhood brain tumors are also astrocytomas (ACS, 2004a; ACS, 2004b).

Of the 92 individuals in Arlington reported to the MCR with a diagnosis of brain or CNS cancer from 1982 to 2001, approximately 80% (n=74) were diagnosed with a glioma. Specifically, 57 individuals (62%) were diagnosed with an astrocytoma, 4 individuals (4%) were diagnosed with an oligodendroglioma, and 13 individuals (14%) were diagnosed with a non-specified type of glioma. The remaining 20% of individuals diagnosed with brain or CNS cancer were diagnosed with a variety of other less common histology types. This is similar to the histologic distribution for the state of Massachusetts as a whole. Specifically, 76% (n=7367) of individuals diagnosed with brain or CNS cancer in Massachusetts were diagnosed with a glioma and the remaining 24% of individuals were diagnosed with a less common histology type. Additionally, similar trends were seen in both the town and the state when each of the smaller time periods were analyzed individually. Among children (age 19 or below) diagnosed with brain and CNS cancers in Arlington during 1982 – 2001, 6 of the 7 children were diagnosed with astrocytomas. The seventh child was diagnosed with a glioma, not otherwise specified.

Similar histologic trends were observed in the two census tracts with statistically significant elevations. Specifically, during the 1989 – 1995 time period, five individuals (83%) residing in CT 3562 were diagnosed with a glioma (3 astrocytomas and 2 non-specified gliomas). The other individual was diagnosed with a non-specified type of



meningioma. In CT 3561, during the most recent time period, four of the five individuals diagnosed with brain or CNS cancer were diagnosed with gliomas (3 astrocytomas and 1 non-specified glioma). The fifth individual diagnosed with brain and CNS cancer in this CT was diagnosed with the subtype meningioma.

#### **D. Geographic Distribution of the Cancer Incidence in Arlington**

In addition to determining census tract-specific incidence rates for brain and CNS cancers, a qualitative evaluation was conducted to determine whether the individuals diagnosed with these cancers appeared to be spatially concentrated in any one area of Arlington during the time period evaluated in this report, 1982 – 2001. Specifically, place of residence at the time of diagnosis was “mapped” for all individuals diagnosed with cancers of the brain or CNS to assess any possible geographic concentrations.

In general, review of the geographic distribution of brain and CNS cancer diagnoses in Arlington revealed no apparent spatial patterns at the neighborhood level that were not likely attributed to factors such as areas of higher population density. For example, although a statistically significant elevation among females was observed in CT 3562 during the 1989 – 1995 time period, this census tract is very densely populated and the diagnoses were fairly evenly distributed throughout the census tract. A similar trend was observed when looking at the distribution of residences of males and females combined who lived in CT 3561 and were diagnosed in the later time period when a statistically significant elevation occurred. These individuals also live in a very densely populated area of Arlington. In addition, varying years of diagnosis and histologies for these individuals do not suggest a common factor (environmental or non-environmental). The geographic distribution of the residences for the seven children diagnosed with brain or CNS cancer from 1982 – 2001 was evaluated and similarly did not reveal any atypical spatial patterns.

#### **E. Cancer Incidence in the Neighborhoods near the Reeds Brook Landfill**

To further address the concerns of residents living in close proximity to the former Reeds Brook Landfill, an analysis of all types of cancers diagnosed among residents of this neighborhood was completed for the years 1982 to the present. The site is located in CT

3565 in the northwestern portion of Arlington bordering the towns of Lexington and Winchester. For this evaluation, the pattern of all cancer diagnoses was reviewed for the area that is bordered to the east by Wright Street, to the south by Summer Street, to the west by the Lexington town line and to the north by the Winchester town line (see Figure 2). Fifteen different streets, in part or whole, are included in this area.

From 1982 – 2004, a total of 18 different types of cancer were diagnosed among residents of this area, representing the occurrence of many different diseases. The most commonly reported diagnoses included cancers of the lung and bronchus, breast, prostate, and colon/rectum. These are the four most common types of cancer diagnosed among men and women in Massachusetts and this pattern is consistent with national and statewide trends in cancer incidence (ACS, 2002). Together, these cancer types represented more than half (58%) of the cancer diagnoses in this area. There were also a number of other cancer types diagnosed among residents of this area of Arlington over the 23 year period reviewed including cancers of the bladder, brain, cervix, esophagus, kidney and renal pelvis, oral cavity and pharynx, ovaries, pancreas, stomach, thyroid, and ureter as well as Hodgkin's disease, melanoma of the skin, and mesothelioma. However, the types of cancer that occurred varied in nature and there was no specific pattern or geographic concentration of any one cancer type within this neighborhood. Also, the years of diagnosis for these individuals varied throughout the 23 years reviewed, indicating no apparent trend or pattern in the time of diagnosis.

The majority of cancer types diagnosed among residents of the neighborhood surrounding the Reeds Brook Landfill site are predominantly associated with non-environmental factors such as family history, smoking, diet, and other lifestyle behaviors. Because the MCR collects some information related to risk factors (e.g., smoking history) for individuals diagnosed with cancer, these data were reviewed to better characterize the incidence patterns of cancer in this area of Arlington. This included a review of age at diagnosis, gender, smoking history, and occupation.

Age is an important risk factor in many cancers. Different cancers occur with different frequencies among the various age groups, and most cancer types occur more frequently

in older populations (i.e., age 50 and over). The average age at diagnosis among individuals in this neighborhood with any type of cancer was 68 and the majority of individuals (93%) were age 50 or older when they were diagnosed. Review of the age and gender pattern among these individuals indicates that the incidence of cancer in this area is consistent with established patterns of disease in the general population.

Because cigarette smoking is also an important risk factor in the development of several cancer types, including cancers of the lung and bronchus, oral cavity and pharynx, esophagus, colon/rectum, bladder, kidney, stomach, and pancreas, smoking history was reviewed for each individual in the neighborhood surrounding the Reeds Brook Landfill site who had been diagnosed with a smoking-related cancer. Of the 37 individuals with a smoking-related cancer, 30 reported a smoking status. Of those, 19 (64%) were current or former smokers and 36 % were nonsmokers. Smoking status was unknown for seven individuals. Therefore, it is likely that smoking played a role in the development of cancer among some residents of the neighborhood surrounding the Reeds Brook Landfill. In addition, some occupational exposures, such as jobs involving contact with chemicals, have been associated with an increased risk for developing certain types of cancer. A review of occupation as reported to the MCR showed that some of the individuals diagnosed with cancer in this area of Arlington worked in jobs that could be related to an increased risk for developing their cancer. Finally, analysis of the geographic distribution of place of residence for individuals diagnosed with cancer did not reveal any atypical spatial patterns that would suggest an association with a common factor in the town of Arlington as a whole or in the neighborhoods surrounding the Reeds Brook Landfill.

We were not able to confirm the diagnoses of all individuals reported to the CEH by Arlington residents. There may be several reasons for this. At the time of this evaluation, the MCR data were complete through 2001; however, as discussed earlier, this is an on-going surveillance system that collects reports on a daily basis. Although we reviewed the MCR data for cancer diagnoses among residents living in the former Reeds Brook Landfill area of Arlington through the present time, it is possible that some residents of this neighborhood with cancer may not be included in the MCR files. For example, some of these individuals may have been diagnosed prior to 1982 when the

MCR began collecting information on individuals in the state diagnosed with cancer. It is also possible that some individuals with recent cancer diagnoses may not have been reported to the MCR yet. Finally, some individuals may have resided at or reported an address other than the Reeds Brook neighborhood at the time of their diagnosis (e.g., a P.O. Box). or a diagnosis of cancer may have been incorrectly reported for some individuals.

## **V. DISCUSSION**

The causes of brain and CNS cancer remain largely unknown. However, some risk factors have been identified. The only established environmental risk factor is exposure to radiation (e.g., from radiation therapy to the head and neck) (ACS, 2004a; Chow et al., 1996). In addition, rare cases of brain and CNS cancer run in some families. Some types have also been associated with certain rare genetic disorders, such as neurofibromatosis type 1, von Hippel-Lindau disease, and Li-Fraumeni syndrome (ACS, 2004a; Chow et al., 1996). Environmental exposures, such as vinyl chloride, aspartame (a sugar substitute), and electromagnetic fields, have been suggested as risk factors for brain cancer, but the evidence to support these associations is inconsistent (ACS, 2004a). For more information regarding risk factors for brain and CNS cancer, please refer to Attachment B.

According to statistics from the American Cancer Society, cancer is the second leading cause of death in Massachusetts and the United States. Not only will one out of three females and one out of two males develop cancer in their lifetime, but cancer will also affect three out of every four families. For this reason, cancers often appear to occur in “clusters,” and it is understandable that someone may perceive that there are an unusually high number of cancer cases in their neighborhood or town. Upon closer examination, many of these “clusters” are not unusual increases, as first thought, but are related to such factors as local population density, variations in reporting or chance fluctuations in occurrence. In other instances, the “cluster” in question includes a high concentration of individuals who possess behaviors or risk factors that put them at high risk for cancer. Some cancer distributions, however, are unusual; that is, they represent a true excess of cancer in a workplace, a community, or among a subgroup of people. A suspected cancer

cluster is more likely to be a true cluster if it involves a large number of cases of one type of cancer diagnosed in a relatively short time period rather than several different types diagnosed over a long period of time (i.e., 20 years), a rare type of cancer rather than common types, and a large number of cases diagnosed among individuals in age groups not usually affected by that cancer. These types of clusters may warrant further public health investigation.

## **VI. CONCLUSIONS**

Based on the information reviewed in this report, there does not seem to be an atypical pattern of brain and CNS cancer in the town of Arlington as a whole, in any of the census tracts, or an atypical pattern of cancer in the neighborhood surrounding the former Reeds Brook Landfill. Although there were some elevations in diagnoses of brain and CNS cancer during certain time periods, in general, the incidence of brain and CNS cancer occurred about as expected when compared to the state as a whole. When elevations were observed, most increases were not statistically significant, with two exceptions. These involved two different census tracts over two different time periods. When these were analyzed further, no atypical patterns were noted for the various factors considered in our analysis. These factors included year of diagnosis, age at diagnosis, gender frequency, histology types, geographic distribution, occupation, and smoking history. Based on our analysis, it does not appear that any one environmental factor played an important role in the occurrence of these elevations of cancer during the time periods evaluated.

## **VII. RECOMMENDATION**

Based on the results of this investigation, the MDPH does not recommend any further evaluation of brain or CNS cancer incidence in Arlington at this time. However, the MDPH will continue to monitor the incidence of all cancer types in the town through city/town cancer incidence reports published by the Massachusetts Cancer Registry.

## VIII. REFERENCES

American Cancer Society (ACS). 2004a. Brain/CNS Tumors in Adults. Available at: <http://www.cancer.org>.

American Cancer Society (ACS). 2004b. Brain/CNS Tumors in Children. Available at: <http://www.cancer.org>

American Cancer Society (ACS). 2002. Lung Cancer. Available at: <http://www.cancer.org>.

Arlington Online, 2005. 2004 State of the Town Address at [www.town.arlington.ma.us/Public\\_Documents/ArlingtonMA\\_Selectmen](http://www.town.arlington.ma.us/Public_Documents/ArlingtonMA_Selectmen).

Berg JW. 1996. Morphologic classification of human cancer. In: Cancer Epidemiology and Prevention. Schottenfeld D and Fraumeni JF Jr (eds). New York: Oxford University Press, 1996:28-44.

Chow WH, Linet MS, Liff JM, and Greenberg RS. 1996. Cancers in children. In: Schottenfeld D and Fraumeni JF (eds). *Cancer Epidemiology and Prevention, 2<sup>nd</sup> edition*. New York: Oxford University Press.

Environmental Systems Research Institute (ESRI). 2002. ArcGIS, Arcview license, ver. 8.3, Redlands, California.

Metcalf & Eddy, 1996. Draft Comprehensive Site Assessment (CSA) for the Reeds Brook Property. Prepared for the Town of Arlington Department of Community Planning and Development. July 1996.

O'Brien, Kevin, 2005. Personal communication with Kevin O'Brien, Director of Planning and Community Development, Town of Arlington. August 4, 2005.

Preston-Martin S, Mack W. 1996. Neoplasms of the nervous system. In: *Cancer Epidemiology and Prevention*. 2nd Ed, edited by Schottenfeld D, Fraumeni JF. New York: Oxford University Press: 1996.

Rothman K and Boice J. 1982. *Epidemiological Analysis with a Programmable Calculator*. Boston: Epidemiology Resources, Inc. 1982.

U.S. Department of Commerce (U.S. DOC). 1980. *Census of Population: General Population Characteristics, Massachusetts*. U.S. Department of Commerce, Washington, DC: US Government Printing Office.

U.S. DOC. 1990. *Census of Population: General Population Characteristics, Massachusetts*. U.S. Department of Commerce, Washington, DC: US Government Printing Office.

U.S. DOC. 2000. Census of Population: General Population Characteristics, Massachusetts. U.S. Department of Commerce, Washington, DC: US Government Printing Office.

## **Appendix A**

### **Cancer Incidence Coding Definition**



**Appendix A:  
Coding Definitions of Cancer Site/Type\***

Cancer Site / Type	ICD-O-1 and Other Pre-ICD-O-2 Codes		ICD-O-2 Codes		ICD-O-3 Codes	
	Site code	Histology code	Site code	Histology code	Site code	Histology code
Brain & Central Nervous System (CNS)	191.0-192.9	See Table 1 below	C70.0-C72.9	See ICD-O codes in Table 1 below	C70.0-C72.9	See Table 2 below

\*Note: Includes invasive tumors only, selected by excluding in situ stages J0, S0, TTISNXM0, TTANXMX, TTANXM0, TTAN0MX, TTISN0M0, TTISNXMX, TTISN0MX, TTISN0M0, TTIN0M0, TTIN0MX, TTINXM0, and TTINXMX (1982-1994 data) or by specifying behavior code (1995-present data).

Table 1: Histology codes for Brain and Central Nervous System (pre-ICD-O-3)

ICD-O O 9370, 9380, 9381, 9382, 9390, 9391, 9392, 9400, 9401, 9403, 9410, 9411, 9420, 9421, 9422, 9423, 9424, 9430, 9440, 9441, 9442, 9443, 9450, 9451, 9460, 9470, 9471, 9472, 9473, 9480, 9481, 9490, 9500, 9501, 9502, 9503, 9530, 9539, 9540, 9560, 9561.  
SNOP P 9363, 9383, 9393, 9403, 9413, 9423, 9433, 9443, 9453, 9463, 9473, 9483, 9493, 9503, 9533, 9543, 9563.  
HLTHSTT B 9383, 9393, 9403, 9433, 9443, 9453, 9463, 9473, 9483, 9493, 9503, 9530, 9533, 9537, 9543, 9563.

Table 2: Histology codes for Brain and Central Nervous System (ICD-O-3)

ICD-O O 9370, 9371, 9372, 9380, 9381, 9382, 9390, 9391, 9392, 9400, 9401, 9410, 9411, 9420, 9421, 9423, 9424, 9430, 9440, 9441, 9442, 9450, 9451, 9460, 9470, 9471, 9472, 9473, 9474, 9480, 9490, 9500, 9501, 9502, 9503, 9530, 9539, 9540, 9560, 9561, 9571.

## **Appendix B**

### **Risk Factor Information for Brain and Central Nervous System (CNS) Cancer**

## Appendix B

### Risk Factor Information For Brain and CNS Cancer

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Brain and central nervous system (CNS) tumors can be either malignant (cancerous) or benign (non-cancerous). Primary brain tumors (i.e., brain cancer) comprise two main types: gliomas and malignant meningiomas. Gliomas are a general classification of malignant tumors that include a variety of types, named for the cells from which they arise: astrocytomas, oligodendrogliomas, and ependymomas. Meningiomas arise from the meninges, which are tissues that surround the outer part of the spinal cord and brain. Although meningiomas are not technically brain tumors, as they occur outside of the brain, they account for about 50% of all reported primary brain and spinal cord tumors. The majority of meningiomas (about 85%) are benign and can be cured by surgery. Therefore, approximately 7.5% of brain and CNS tumors are malignant meningiomas. In addition to these main types, there are a number of rare brain tumors, including medulloblastomas, which develop from the primitive stem cells of the cerebellum and are most often seen in children. Also, 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 (ACS, 1999a). The American Cancer Society estimates that 18,500 Americans (10,620 men and 7,880 women) will be diagnosed with primary brain cancer (including cancers of the central nervous system, or spinal cord) and approximately 12,760 people (7,280 men and 5,480 women) will die from this disease in 2005 (ACS, 2005).

Brain and spinal cord cancers account for over 20% of all cancer types diagnosed among children aged 0-14 (ACS, 2005). About half of all childhood brain tumors are astrocytomas and 25% are medulloblastomas (ACS, 1999b). After a peak in childhood (generally under 10 years of age), the risk of brain cancer increases with age from age 25 to age 75. In adults, the most frequent types of brain tumors are astrocytic tumors (mainly astrocytomas and glioblastoma multiforme<sup>1</sup>). Incidence rates are higher in males than in females for all types. In general, the highest rates of brain and nervous system cancer tend to occur in whites. However, this varies somewhat by type; the incidence of gliomas is lower among black men and women than whites, but for meningiomas, the reverse is true (Preston-Martin and Mack, 1996).

Despite numerous scientific and medical investigations, and analyses, the causes of brain cancer are still largely unknown. Among the possible risk factors investigated in relation to this type of cancer are ionizing radiation, electromagnetic fields, occupational exposures, exposure to N-nitroso compounds, head trauma, and genetic disorders.

The most established risk factor (and only established environmental risk factor) for brain tumors (either cancerous or non-cancerous) is high-dose exposure to ionizing radiation (i.e., x-rays and gamma rays). Most radiation-induced brain tumors are caused by radiation to the head from the treatment of other cancers (ACS, 1999a). Meningiomas are the most common type of tumors that occur from this type of exposure, but gliomas may also occur (Preston-Martin and Mack, 1996). Among adults, the risk of developing meningiomas has been associated with full-mouth dental x-rays taken decades ago when radiation doses were higher than today. Although the relationship between low-dose

## Appendix B

### Risk Factor Information For Brain and CNS Cancer

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radiation exposure and increased risk of brain tumors has been debated in several studies, prenatal exposure from diagnostic x-rays has been related to an increase in childhood brain tumors (Preston-Martin and Mack, 1996).

In recent years, there has been increasing public concern and scientific interest regarding the relationship of electromagnetic fields (EMF) to brain cancer. However, results from recent epidemiological investigations provide little or no evidence of an association between residential EMF exposure (e.g., from power lines and home appliances) and brain tumors (Kheifets, 2001). Studies also suggest that the use of handheld cellular telephones is not associated with an increased risk of primary brain cancer (Muscat et al., 2000). However, given the relatively recent use of cellular phones, evidence is preliminary and few studies have been conducted.

Other environmental factors such as exposure to vinyl chloride (used in the manufacturing of some plastics) and aspartame (a sugar substitute) have been suggested as possible risk factors for brain cancer but no conclusive evidence exists implicating these factors (ACS, 1999a). Although some occupational studies have suggested that electrical and electric utility workers may be at a slightly increased risk of brain cancer, these studies have important limitations, such as exposure misclassifications and a lack of dose-response relationships (Kheifets, 2001). Some researchers have also reported an increased risk of brain tumors in adults among veterinarians and farmers. Exposures to farm animals and pets have been considered as possible risk factors because of their association with bacteria, pesticides, solvents, and certain animal oncogenic (cancer-related) viruses (Yeni-Komshian and Holly, 2000). However, the relationship between farm life and brain cancer remains controversial.

Recent reports have proposed a link between occupational exposure to lead and brain cancer risk, but further analytic studies are warranted to test this hypothesis (Cocco et al., 1998). In a recent case-control study, the concentrations of metal and non-metal compounds in brain biopsies from patients with primary brain tumors were compared to results from an analysis of tumor-free brain tissue. Statistically significant associations were observed between the presence of brain tumors and the concentrations of silicon, magnesium, and calcium (Hadfield et al., 1998). However, further research using a larger sample size is needed to determine whether exposure to these elements plays a role in the development of brain cancer. Other occupations that may be associated with elevated risks include workers in certain health professions (e.g., pathologists and physicians), agricultural workers, workers in the nuclear industry, and workers in the rubber industry, although specific exposures have not been established (Preston-Martin and Mack, 1996). Studies investigating the possible association between occupational exposure of parents (in particular, paper or pulp-mill, aircraft, rubber, metal, construction, and electric workers) and the onset of brain tumors in their children have provided inconsistent results (Preston-Martin and Mack, 1996).

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### Risk Factor Information For Brain and CNS Cancer

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The association between the development of brain cancer and nitrites and other N-nitroso compounds, among the most potent of carcinogens, has been heavily researched. N-nitroso compounds have been found in tobacco smoke, cosmetics, automobile interiors, and cured meats. A recent study concluded that an increased risk of pediatric brain tumor may be associated with high levels of nitrite intake from maternal cured meat consumption during pregnancy (Pogoda and Preston-Martin, 2001). However, the role of nitrites and cured meats in the development of brain cancer remains controversial (Blot et al., 1999; Bunin, 2000). Because most people have continuous, low level exposure to N-nitroso compounds throughout their lives, further studies, especially cohort studies, are needed to determine if this exposure leads to an increased risk of brain tumors (Preston-Martin, 1996).

Injury to the head has been suggested as a possible risk factor for later development of brain tumors but most researchers agree that there is no conclusive evidence for an association (ACS, 1999b). Head trauma is most strongly associated with the development of meningiomas compared with other types of brain tumor. Several studies have found an increased risk in women with histories of head trauma; in men who boxed; and in men with a previous history of head injuries. Gliomas are the most common type of childhood brain tumor and have been positively associated with trauma at birth (e.g., Cesarean section, prolonged labor, and forceps delivery). However, other studies have found no association (Preston-Martin and Mack, 1996).

In addition, rare cases of brain and spinal cord cancer run in some families. Brain tumors in some persons are associated with genetic disorders such as neurofibromatosis types I and II, Li-Fraumeni syndrome, and tuberous sclerosis. Neurofibromatosis type I (von Recklinghausen's disease) is the most common inherited cause of brain or spinal cord tumors and occurs in about one out of every 3,000 people (Preston-Martin and Mack, 1996). The disease may be associated with optic gliomas or other gliomas of the brain or spinal cord (ACS, 1999b). Of those afflicted with the disease, about 5-10% will develop a central nervous system tumor (Preston-Martin and Mack, 1996). In addition, von Hippell-Lindau disease is associated with an inherited tendency to develop blood vessel tumors of the cerebellum (ACS, 1999a). However, malignant (or cancerous) brain tumors are rare in these disorders; inherited syndromes that predispose individuals to brain tumors appear to be present in fewer than 5% of brain tumor patients (Preston-Martin and Mack, 1996).

Other possible risk factors investigated for brain cancer have included alcohol consumption, use of barbiturates, smoking and exposure to second-hand smoke, pesticides, and infectious diseases (i.e., tuberculosis and chicken pox). To date, studies on these risk factors have yielded inconclusive results. Further, the majority of individuals diagnosed with brain cancer have no known risk factors (ACS, 1999a).

## Appendix B

### Risk Factor Information For Brain and CNS Cancer

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#### References

- American Cancer Society. 2005. *Cancer Facts & Figures 2005*. Atlanta: American Cancer Society, Inc.
- American Cancer Society. 1999a. *Brain and Spinal Cord Cancers of Adults*. Available at: <http://www3.cancer.org/cancerinfo/>.
- American Cancer Society. 1999b. *Brain/Central Nervous System (CNS) Tumors in Children*. Available at: <http://www3.cancer.org/cancerinfo/>.
- Blot WJ, Henderson BE, Boice JD, Jr. 1999. Childhood cancer in relation to cured meat intake: review of the epidemiological evidence. *Nutr Cancer* 34(1):111-8.
- Bunin G. 2000. What causes childhood brain tumors? Limited knowledge, many clues. *Pediatr Neurosurg* 32(6):321-6.
- Cocco P, Dosemeci M, Heineman EF. 1998. Brain cancer and occupational exposure to lead. *J Occup Environ Med* 40(11):937-42.
- Hadfield MG, Adera T, Smith B, Fortner-Burton CA, Gibb RD, Mumaw V. 1998. Human brain tumors and exposure to metal and non-metal elements: a case control study. *J Environ Pathol Toxicol Oncol* 17(1):1-9.
- Kheifets LI. 2001. Electric and magnetic field exposure and brain cancer: a review. *Bioelectromagnetics Suppl* 5:S120-31.
- Muscat JE, Malkin MG, Thompson S, Shore RE, Stellman SD, McRee D, Neugut AI, Wynder EL. 2000. Handheld cellular telephone use and risk of brain cancer. *JAMA* 284(23):3001-7.
- Pogoda JM, Preston-Martin S. 2001. Maternal cured meat consumption during pregnancy and risk of paediatric brain tumour in offspring: potentially harmful levels of intake. *Public Health Nutr* 4(2):183-9.
- Preston-Martin S, Mack W. 1996. Neoplasms of the nervous system. In: *Cancer Epidemiology and Prevention*. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press: 1996.
- Yeni-Komshian H, Holly EA. 2000. Childhood brain tumours and exposure to animals and farm life: a review. *Paediatr Perinat Epidemiol* 14(3):248-56.