COMMONWEALTH OF MASSACHUSETTS

DEPARTMENT OF PUBLIC HEALTH

Evaluation of Brain/CNS and Breast Cancer Incidence in Walpole, MA 1998 - 2002

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I.   Introduction

At the request of the Walpole Health Department, the Community Assessment Program (CAP), a division within the Massachusetts Department of Public Health, Center for Environmental Health (MDPH, CEH), reviewed brain/central nervous system (CNS) cancer and breast cancer incidence data for the 5-year time period 1998-2002 for the town of Walpole. This evaluation was initiated based on community concerns about the patterns of these two cancer types at the neighborhood level and elevated incidence rates reported for the town as a whole in the Massachusetts Cancer Registry's (MCR) most recent report *Cancer Incidence in Massachusetts 1998-2002: City and Town Supplement* (MCR 2005).

II.   Methods

Brain/CNS and breast cancer incidence data for the town of Walpole were obtained from the MCR. The MCR is a division of the MDPH Center for Health Information, Statistics, Research, and Evaluation and is a population-based surveillance system that has been monitoring cancer incidence in the Commonwealth since 1982. All new diagnoses of invasive cancer, along with several types of *in situ* (localized) cancer, occurring 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.

In order to determine whether cancer incidence in a community is occurring at a higher or lower rate than expected, a statistic called the standardized incidence ratio (SIR) is calculated using data from the MCR. More specifically, an SIR is the number of observed cancer cases in a town (or census tract) divided by the number of expected cases based on the population of the town (or census tract) and the state's cancer rates. An SIR greater than 100 indicates that more cancer cases occurred than expected; an SIR less than 100 means that fewer cases occurred than expected. For example, an SIR of 150 is interpreted as 50 percent more cases than expected; an SIR of 90 indicates 10 percent fewer cases than expected.   When an SIR is statistically significant, as indicated in the report tables by an asterisk symbol (*), there is less than a 5% chance that the observed number of cases is due to chance alone. It is the policy of the MDPH

Center for Health Statistics, Research, and Evaluation not to calculate SIRs and 95% confidence intervals (CIs) when fewer than five cases are observed, due to the instability of the rates. A more detailed explanation of SIRs and 95% CIs is provided in Appendix A.

Because accurate age-group and gender-specific population data are required to calculate SIRs, the census tract (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 by the U.S. Census Bureau. CTs usually contain between 2,500 and 8,000 persons and are designed to be homogeneous with respect to population characteristics. According to the U.S. Census, the town of Walpole is subdivided into three CTs (U.S. DOC 2000a). The town boundaries and census tract locations for Walpole are illustrated in Figure 1. SIRs were calculated for both brain/CNS and breast cancer for the town of Walpole as a whole and for each CT.

In addition to calculating SIRs, place of residence at the time of diagnosis was mapped for each resident diagnosed with brain/CNS or breast cancer in Walpole using a computerized geographic information system (GIS) (ESRI 2004). This allowed assignment of census tract location for each individual as well as a qualitative evaluation of the spatial distribution of diagnoses at a smaller geographic level (i.e., neighborhoods). Cases for which census tract designation was not possible due to inadequate address information were included in the town totals for Walpole. For this evaluation, there were three individuals diagnosed with breast cancer who could not be assigned to a Walpole census tract (all individuals diagnosed with brain/CNS cancer were mapped). The geographic pattern was determined using a qualitative evaluation of the point pattern of individuals diagnosed with cancer to assess any possible concentrations of cases. The MDPH is bound by law not to make public names or 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 locations of individuals diagnosed with cancer. However, a summary of this evaluation with any notable findings is presented in this report.

Available information from the MCR related to cancer risk factors was also evaluated for residents of Walpole who were diagnosed with brain/CNS and breast cancer and compared to

known or established incidence patterns for these diseases. Risk factor information reported to the MCR at the time of diagnosis and evaluated for brain/CNS cancer included the individual's age, histology (cell-type), and whether they had a previous cancer diagnosis. For breast cancer, specific case information reviewed included the age distribution and the stage of cancer at the time of diagnosis. In addition, because a higher socioeconomic status is correlated with risk factors for increased incidence of breast cancer, available information on education and income from the 2000 federal census was also evaluated for Walpole.

III.   Brain/CNS Cancer Results

Table 1 summarizes brain/CNS cancer incidence data for the town of Walpole as a whole and for each CT for the 5-year time period 1998-2002. The table provides information on the number of individuals diagnosed with brain/CNS cancer in Walpole (observed), the number of cancer cases expected based on the population of Walpole and the statewide cancer experience, and the Standardized Incidence Ratio (SIR). SIRs were not calculated for some census tracts due to the small number of observed cases (less than five). However, the expected numbers of cases were computed to determine whether excess numbers of cancer cases were occurring.

A. Cancer Incidence

The rate of brain/CNS cancer was elevated among males and females combined in the town of Walpole as a whole during 1998-2002 (14 diagnoses observed vs. 9.1 expected, SIR=154), however this elevation was not statistically significant. Slight elevations were also observed among males and females when evaluated separately. Specifically, eight males were diagnosed during 1998-2002, whereas five would have been expected. Six females were diagnosed with brain/CNS cancer when approximately four were expected. As with the town-wide rates, neither of these elevations was statistically significant.

Brain/CNS cancer incidence was greater than expected in two of three Walpole census tracts. In the third census tract, brain/CNS cancer was lower than expected. Specifically, seven individuals were diagnosed with brain/CNS cancer in CT 4111 whereas approximately three would have been expected (SIR=234). In CT 4113, six individuals with brain/CNS cancer were

observed versus approximately four expected. Neither elevation was statistically significant. One individual was diagnosed with brain/CNS cancer in CT 4112 during 1998-2002, whereas approximately two would be expected.

B. Review of Risk Factor Information

After a peak in childhood (generally under 10 years of age), the risk of developing brain cancer increases with age from age 25 to 75. Brain cancers occur with different frequencies among the various age-groups, and most brain cancer occurs more frequently in older populations (i.e., those over 50 years of age) (Ries et al. 2005). Statewide, cancers of the brain/CNS are the second most common type of cancer in children. From 1998-2002, the average age at diagnosis among the 14 individuals with brain/CNS cancer in Walpole was 55, with a range of 6 to 89 years of age at diagnosis. Two children (i.e., ages 0-19) were diagnosed with brain/CNS cancer in Walpole and reported to the MCR during 1998-2002. The majority of individuals (64%, n = 9) were 50 years of age or older at the time of diagnosis.

According to the scientific literature, the most common primary brain tumors are gliomas. Gliomas are a general classification of malignant tumors that include a variety of types such as glioblastoma, astrocytoma, oligodendroglioma, and ependymomas. In adults, the most frequent types of brain tumors are astrocytic tumors (mainly astrocytomas and glioblastoma multiforme). About half of all childhood brain tumors are astrocytomas and 25% are medulloblastomas (ACS 2004b).

All of the 14 individuals reported with brain/CNS cancer in Walpole during 1998-2002 were diagnosed with cancer of the brain. A review of the various histologies revealed that for both children and adults, no unusual patterns of histology were observed. Specifically, all 14 individuals were diagnosed with a glioma, the most common type of brain cancer. The majority of adults (58%, n = 7) were diagnosed with glioblastoma, and other histology subtypes included an astrocytoma, oligodendroglioma, and types of glioma that were not otherwise specified (NOS). The two Walpole children reported to the MCR with a diagnosis of brain cancer during 1998-2002 were diagnosed with the astrocytoma sub-type of glioma.

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One of the well established risk factors (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) (Preston-Martin & Mack 1996). Most radiation-induced brain tumors are caused by radiation to the head from treatment of other cancers (ACS 2004a). Review of specific patient information from the MCR identified one individual in Walpole diagnosed with brain cancer during 1998-2002 who had been previously diagnosed with cancer. This patient may have received treatment that could have contributed to their subsequent diagnosis of brain cancer. It is important to note, however, it is not possible to determine whether this individual actually received radiation therapy for their cancer.

C. Geographic Distribution

A review of the geographic distribution of individuals diagnosed with brain/CNS cancer in Walpole during 1998-2002 revealed no unusual patterns at the neighborhood level that would suggest the presence of a common factor. Although brain/CNS cancer incidence occurred more often than expected in CTs 4111 and 4113, the locations of individual diagnoses generally matched closely with the pattern of population density within those census tracts. For example, more individuals diagnosed with brain/CNS cancer were located in the northern part of CT 4111 where population density is higher.

IV.   Breast Cancer Results

Table 2 summarizes breast cancer incidence data for the town of Walpole as a whole and for each CT for the 5-year period 1998-2002. Similar to Table 1, Table 2 provides information on the number of females diagnosed with breast cancer in Walpole (observed), the number of cancer cases expected based on the population of Walpole and the statewide cancer experience, and the Standardized Incidence Ratio (SIR).

A. Cancer Incidence

More breast cancer was observed than expected in the town of Walpole as a whole during 1998-2002. Specifically, 113 females were diagnosed with breast cancer whereas approximately 98

would have been expected (SIR=116). This elevation was not statistically significant. When breast cancer was evaluated for individual Walpole census tracts, a statistically significant elevation was observed among females in CT 4112 (45 diagnoses observed vs. 24.3 expected, SIR=185, 95%CI=135-248). Fewer females were diagnosed with breast cancer in CT 4111 (22 diagnoses observed vs. 31.5 expected, SIR=70), and breast cancer occurred approximately as expected among females in CT 4113 (43 diagnoses observed vs. 41.2 expected, SIR=104). There were no males diagnosed with breast cancer in Walpole during 1998-2002.

B. Review of Risk Factor Information

According to the scientific literature, incidence rates of breast cancer increase notably both nationally and in Massachusetts among females ages 45 to 79 (MCR 2002; Ries et al. 2005). Age at diagnosis is collected by the MCR for each individual reported with a diagnosis of cancer. Approximately 84% (n = 95) of females in Walpole diagnosed with breast cancer during this time period were age 45 or older at diagnosis. Approximately 37% (n = 42) of females in Walpole were 65 years of age or older at the time of diagnosis. The average age at diagnosis was approximately 60 years old with a range of 30 to 97 years of age. These trends are consistent with those observed among individuals diagnosed with breast cancer state-wide. Specifically, the average age at diagnosis among Massachusetts residents diagnosed with breast cancer was 62 years old with a range of 20 to 104 years of age.

To better understand the pattern of breast cancer in Walpole, stage of cancer at the time of diagnosis was also reviewed. The staging of breast cancer categorizes the extent of the disease and its spread at the time of diagnosis. Communities in which large portions of women receive routine breast cancer screening are expected to have a greater number of women diagnosed at the early stages of the disease. Likewise, communities with low screening rates would be expected to have fewer cases diagnosed and more of those diagnosed at the later stages of disease. Invasive breast cancer is typically classified as one of four stages of disease: localized, regional, distant, and unknown. Localized breast cancer represents a diagnosis in which the tumor is invasive but the cancer is confined to the breast. Regional refers to a tumor that has spread beyond the organ of origin (breast), including spread to adjacent tissues and organs, lymph nodes, or both. Distant stage breast cancer is a cancer that has metastasized or spread to organs

other than those adjacent to the organ of origin, to distant lymph nodes, or both (MCR 1996). Some of the cases are reported to the MCR with an unknown stage meaning that, at the time of reporting by a hospital or other facility (e.g. physician's office), the tumor had not been staged.

Data on stage of cancer at the time of diagnosis for individuals diagnosed with breast cancer in Walpole during 1998-2002 indicated that the majority of females were diagnosed at earlier stages of disease rather than later stages. Specifically, from 1982-2002, approximately 64% of breast cancer diagnoses among females in Walpole were diagnosed at the local stage of disease, approximately 31% were diagnosed at the regional stage, and approximately 4% were diagnosed at the distant stage. Stage at diagnosis was unknown for one individual (see Figure 2). This distribution is similar to that observed statewide during this time period (66% local, 27% regional, 4% distant, and 3% unknown). Localized breast cancer was also diagnosed more often than regional or distant breast cancer in Walpole CT 4112, where a statistically significant elevation in breast cancer occurred during 1998-2002. Specifically, among females with breast cancer in CT 4112, approximately 62% were diagnosed at the localized stage, 31% were diagnosed at the regional stage, approximately 4% were distant, and approximately 2% were unknown.

The medical literature has shown a correlation between elevated breast cancer incidence rates and higher socioeconomic status (Henderson et al 1996). Therefore, potential differences in education level and median household income were assessed for CT 4112 where a statistically significant rate of breast cancer occurred and compared to Walpole's other two census tracts. According to the 2000 census, CT 4112 had the highest proportion of residents above the age of 25 with a bachelor's or other advanced degree (see Table 3). Specifically, 43% of the population over the age of 25 years in CT 4112 had a bachelor's, masters, professional, or doctorate degree whereas 39% of the populations in CTs 4111 and 4113 had these advanced degrees (U.S. DOC 2000b). While all three census tracts had a higher median income than that of the state of Massachusetts as a whole ($50,502), CT 4112 did not have the highest of the three Walpole census tracts (see Table 4). Specifically, based on 2000 federal census data, the 1999 median household income was $68,768 in CT 4111, $75,463 in CT 4112, and $77,521 in CT 4113 (U.S. DOC 2000b). Thus, while data on higher education level is consistent with a higher risk of

breast cancer in CT 4112, available information on median household income suggests the correlation with a higher socioeconomic status is less clear.

C. Geographic Distribution

A review of the geographic pattern of females diagnosed with breast cancer in Walpole during 1998-2002 revealed no unusual spatial concentrations at the neighborhood level. As with the brain/CNS cancer observations, any apparent concentrations were likely attributable to factors such as a higher population density. Although the incidence of breast cancer was statistically significantly elevated in CT 4112 during this time period, no atypical patterns with respect to place of residence were noted in this part of town.

V.   Discussion and Conclusions

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 people develop cancer in their lifetime, but cancer will 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 surrounding neighborhoods or towns. Upon close 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 related behaviors or risk factors for cancer. Some, 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 cluster is more likely to be a true cancer 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.

Review of the most recent available cancer incidence data for Walpole indicated that town-wide rates of both brain/CNS and breast cancer were elevated during the five-year time period 1998-2002. However, neither elevation was statistically significant. The elevation of brain/CNS cancer was attributed to non-statistically significant elevations observed in two of three Walpole census tracts, CTs 4111 and 4113. Therefore, the overall elevation could not be attributed to one particular area of Walpole. Slightly less brain/CNS cancer was observed in CT 4112 than expected. While the elevated rate of breast cancer did not reach statistical significance for the town of Walpole as a whole, a statistically significant rate of breast cancer occurred in CT 4112, located on the eastern side of town. Breast cancer rates were near or below what would be expected for the other two Walpole census tracts.

Despite the elevations observed for brain/CNS cancer (town-wide and in CTs 4111 and 4113) and breast cancer (town-wide and CT 4112) in Walpole, evaluation of the geographic distribution of individuals at the neighborhood level did not reveal any atypical spatial patterns that would suggest a common factor (environmental or nonenvironmental) related to the incidence of these cancer types. In general, any spatial patterns that were observed were consistent with what would be expected based on the population distribution and areas of higher population density. In addition, when available risk factor information for those diagnosed with brain/CNS and breast cancer was evaluated, cancer trends in Walpole were similar to those seen in the general population. A review of specific case information for individuals with brain/CNS cancer in Walpole did not reveal any unusual geographic patterns with respect to age, date of diagnosis or histology sub-types.

The majority of females diagnosed with breast cancer during 1998-2002 were age 45 or older at the time of diagnosis. This is consistent with the age distribution of females diagnosed with this cancer type in the state of Massachusetts as a whole and in the United States. In addition, similar to breast cancer diagnoses state-wide, females in Walpole as a whole and in CT 4112 (where breast cancer was statistically significantly elevated) appear to be diagnosed with breast cancer at earlier stages, which would be consistent for a community with higher screening rates. A review of 2000 federal census data indicated that CT 4112 had the highest proportion of adult residents with a bachelor's or other advanced degree which is consistent with the medical literature

correlating a higher socioeconomic status with an increased risk of breast cancer. All three Walpole census tracts had a higher median income than the state of Massachusetts as a whole, which is also consistent with the medical literature. However, when median income levels were compared between individual census tracts, CT 4112 was not the highest of the three suggesting that the potential correlation between higher socioeconomic status and the statistically significant rate of breast cancer in this census tract was less clear.

Despite numerous scientific and medical investigations, and analyses, the causes of brain cancer are still largely unknown. However, a few risk factors have been identified. The most well-established risk factor is exposure to ionizing radiation (e.g., from radiation therapy to the head and neck) (ACS 2004a). In addition, rare cases of brain 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). Environmental exposures such as vinyl chloride, aspartame (a sugar subsititute), and electromagnetic fields, have been suggested as risk factors for brain cancer, but the evidence to support these associations is inconsistent (ACS, 2004a). When available case information from the MCR, such as age at diagnosis and histology distribution, was evaluated for individuals diagnosed with brain/CNS cancer in Walpole during 1998-2002 the trends observed were consistent with those seen in the general population. However, information about personal risk factors that may also influence the development of brain cancer (e.g., family history and heredity) are not collected by the MCR or any other readily accessible source, and therefore, could not be evaluated in this investigation. Please refer to Appendix B for further information on brain/CNS cancer.

Despite the vast number of studies on the causation of breast cancer, known risk factors are estimated to account for slightly more than half of all diagnoses in the general population (Madigan et al. 1995). Researchers are continuing to examine other potential genetic, hormonal, and environmental risk factors for breast cancer. Nonetheless, it is known that a female's risk for developing breast cancer can change over time due to many factors, some of which are dependent upon well-established risk factors for this cancer type. Females with a family history of breast cancer, those who have never had children, or have had their first child after the age of 30, are at an increased risk for developing this disease (ACS 2005). Females who take

menopausal hormone therapy (estrogen plus progestin) for five or more years after menopause also appear to have an increased risk of developing breast cancer (National Cancer Institute 2003). Information related to family history of breast cancer, reproductive factors, and use of hormone replacement therapy after menopause is not included in the MCR database. Socioeconomic differences in breast cancer incidence may be a result of current screening participation rates. Currently, women of higher socioeconomic status (SES) have higher screening rates, which may result in more of the cases being detected in these women. However, women of higher SES may also have an increased risk for developing the disease due to different reproductive patterns (i.e., parity, age at first full-term birth, and age at menarche). Although women of lower SES show lower incidence rates of breast cancer in number, their cancers tend to be diagnosed at a later stage (Segnan, 1997). Hence, rates for their cancers may appear lower due to the lack of screening participation rather than a decreased risk for the disease. Moreover, it is likely that SES is not in itself the associated risk factor for breast cancer. Rather, SES probably represents different patterns of reproductive choices, occupational backgrounds, environmental exposures, and lifestyle factors (i.e., diet, physical activity, cultural practices) (Henderson et al, 1996). Please see Appendix B for more information on risk factors related to the development of breast cancer.

In summary, despite some elevations in brain/CNS and breast cancer observed in Walpole during 1998-2002, a detailed review of the geographic distribution of individuals revealed no apparent spatial patterns at the neighborhood level, and analysis of available risk factor information for these cancers suggest that the trends observed in Walpole are similar to those seen in the general population. A statistically significant elevation in breast cancer observed in Walpole CT 4112 may be partially attributed to a higher education level among residents of this census tract, however, a correlation to higher socioeconomic status using data on median income level was less clear. The MDPH will continue to monitor the incidence of brain/CNS and breast cancer incidence in Walpole through city/town cancer incidence reports published by the MCR.
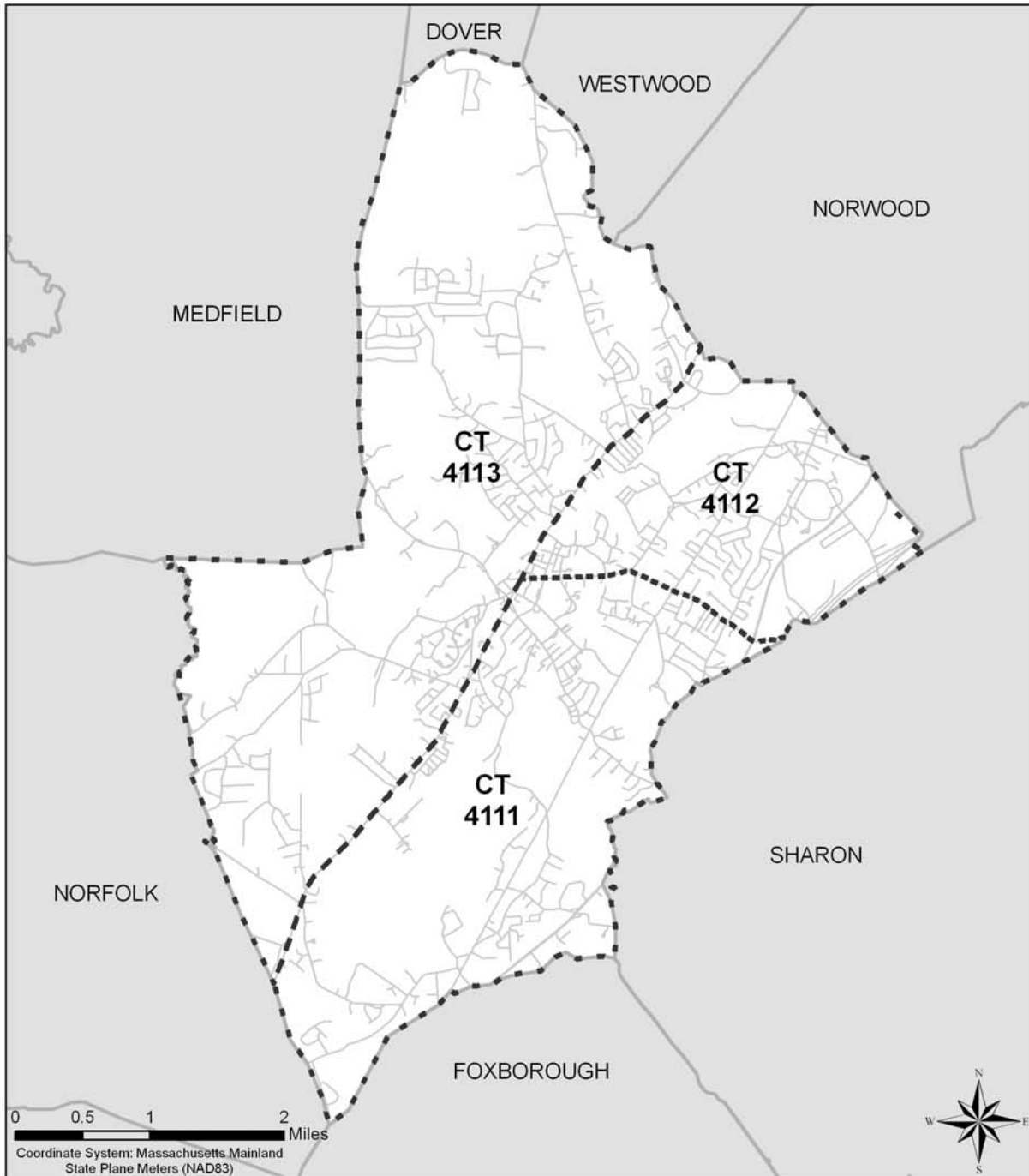
VI.   References

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). 2005. The Risk Factors for Breast Cancer. Available at:
http://www.cancer.org

Environmental Systems Research Institute (ESRI). 2004. ArcGIS, ver. 9.1. Copyright
Environmental Systems Research Institute, 1999-2002. Redlands, California.

Henderson BE, Pike MC, Bernstein L, Ross RK. 1996. Breast Cancer, chapter 47 in Cancer
Epidemiology and Prevention. 2nd ed. Schottenfeld D and Fraumeni JF Jr.,eds. Oxford
University Press. pp: 1022-1035.

Madigan MP, Ziegler RG, Benichou J, et al. 1995. Proportion of breast cancer cases in the
United States explained by well-established risk factors. J Natl Cancer Inst 87(22): 1681-5.

Massachusetts Cancer Registry (MCR). 1996. Massachusetts Cancer Registry Abstracting and
Coding Manual for Hospitals. 2nd ed. Massachusetts Department of Public Health, Bureau of
Health Statistics, Research and Evaluation, Boston, MA.

MCR 2002. Cancer in Massachusetts Women 1989-1998. Boston: Massachusetts Department
of Public Health, Center for Health Statistics, Research, and Evaluation.

MCR 2005. Cancer Incidence in Massachusetts 1998-2002: City and Town Supplement. Boston:
Massachusetts Department of Public Health, Center for Health Statistics, Research, and
Evaluation.

National Cancer Institute (NCI). 2003. Menopausal Hormone Use: Questions and Answers.
Available at: http://www.cancer.gov/cancertopics/

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.

Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L, Mariotto A, Feuer EJ,
Edwards BK (eds). SEER Cancer Statistics Review, 1975-2002, National Cancer Institute.
Bethesda, MD, http://seer.cancer.gov/csr/1975_2002/, based on November 2004 SEER data
submission, posted to the SEER web site 2005.

Segnan N. Socioeconomic status and cancer screening. International Agency for Research on
Cancer 1997;138:369-376.

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

U.S. Department of Commerce (DOC) 2000b. Census 2000 Summary File 3 (SF 3) Sample Data. Available at: http://factfinder.census.gov/

Figure 1
Location of 2000 Census Tracts (CT)
Walpole, Massachusetts

DOVER

WESTWOOD

NORWOOD

MEDFIELD

CT
4113

CT
4112

CT
4111

NORFOLK

SHARON

FOXBOROUGH

0   0.5   1   2
Miles

Coordinate System: Massachusetts Mainland
State Plane Meters (NAD83)

Center for
CEH
Environmental Health

Geographic data supplied by: Massachusetts Executive
Office of Environmental Affairs, MassGIS; Geographic
Data Technology, Inc.; U.S. Bureau of the Census.
Approximate location of Bird land fill derived from
GZA GeoEnviromental, Inc., 1997.

Legend

☐ Walpole
☐ Town Boundary
☐ 2000 Census Tract (CT) Boundary
    Major or Minor Road

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**Figure 2
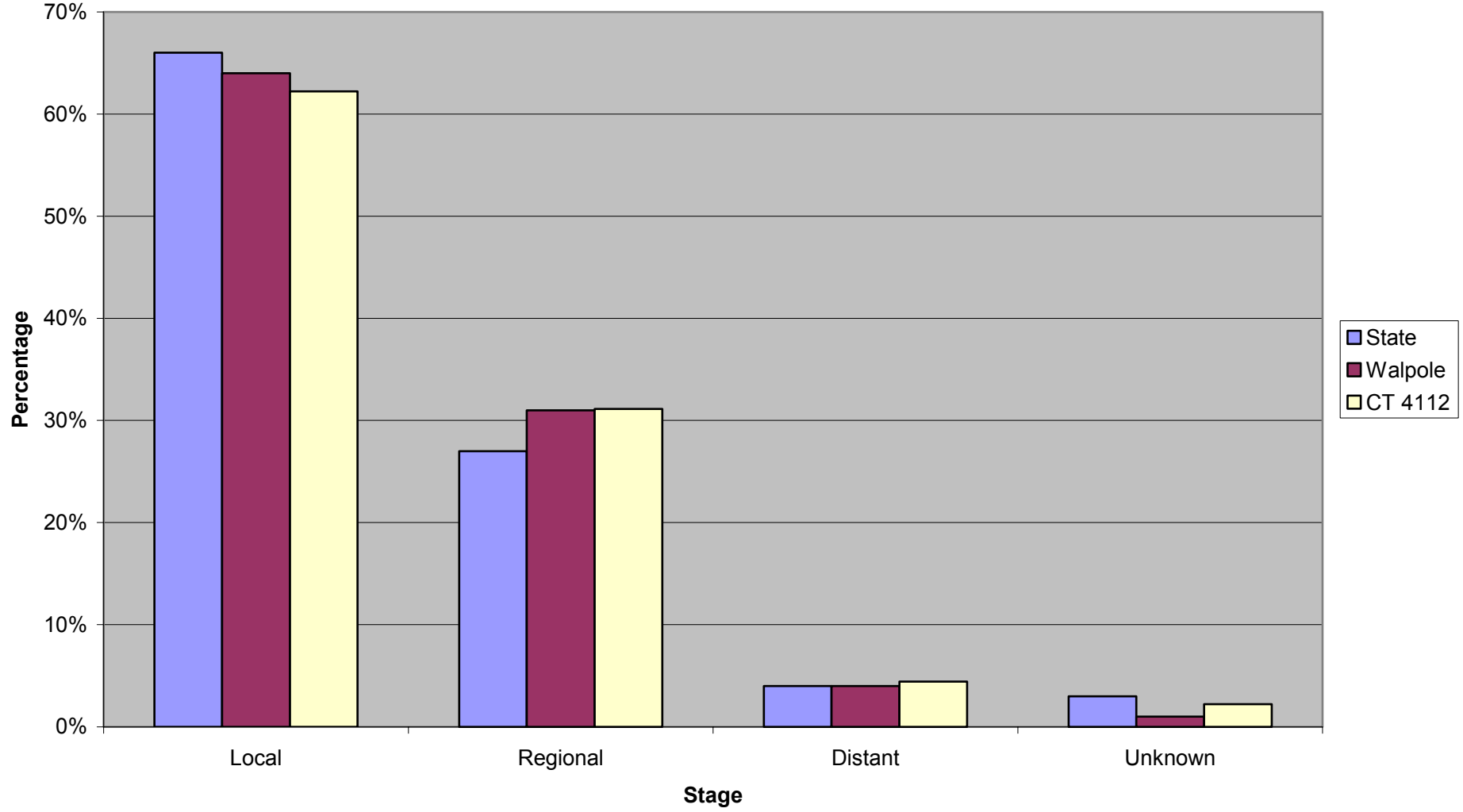Breast Cancer Stage at Diagnosis 1998-2002
Walpole, MA**

TABLE 1
Brain/CNS Cancer Incidence
Walpole, MA
1998-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	7	3.0	234	94 -- 483	3	1.7	NC	NC -- NC	4	1.3	NC	NC -- NC
4112	1	2.3	NC	NC -- NC	0	1.2	NC	NC -- NC	1	1.0	NC	NC -- NC
4113	6	3.8	157	57 -- 341	5	2.1	239	77 -- 557	1	1.7	NC	NC -- NC
City Total[†]	14	9.1	154	84 -- 258	8	5.0	160	69 -- 316	6	4.1	146	53 -- 318

[†] Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.

Expected number of cases presented are rounded to the nearest tenth.

SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases   95% CI = 95% Confidence Interval

Exp = Expected number of cases   NC = Not calculated

SIR = Standardized Incidence Ratio   * = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

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TABLE 2
Breast Cancer Incidence
Walpole, MA
1998-2002

Census Tract	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
4111	22	31.7	69	43 --	105	0	0.3	NC	NC -- NC		22	31.5	70	44 --	106
4112	45	24.5	184	* 134 --	246	0	0.2	NC	NC -- NC		45	24.3	185	* 135 --	248
4113	43	41.6	103	75 --	139	0	0.3	NC	NC -- NC		43	41.2	104	75 --	140
City Total†	113	97.8	116	95 --	139	0	0.8	NC	NC -- NC		113	97.0	116	96 --	140

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.

Expected number of cases presented are rounded to the nearest tenth.

SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases   95% CI = 95% Confidence Interval

Exp = Expected number of cases   NC = Not calculated

SIR = Standardized Incidence Ratio   * = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

Table 3
Education Level by Census Tract
Walpole, MA

Census Tract	2000 Population (25 years and older)	Population with Bachelor's or Higher *	Percent of Population w/ Advanced Degree
4111	5108	1975	39%
4112	4110	1766	43%
4113	6691	2597	39%

*Includes population 25 years and over with bachelor's,

masters, professional and doctorate degrees

Data Source:Census 2000 Summary File 3 (SF 3) - Sample Data

Table 4
Median Household Income in 1999
Walpole, MA

Census Tract	Median Household Income
4111	$68,768
4112	$75,463
4113	$77,521
Massachusetts	$50,502

Data Source:Census 2000 Summary File 3 (SF 3) - Sample Data

APPENDIX A
Explanation of a Standardized Incidence Ratio (SIR)
And 95% Confidence Interval

In order to evaluate cancer incidence a statistic known as a standardized incidence ratio (SIR) was calculated for each cancer type. 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 some 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. 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.

Specifically, an SIR is the ratio of the observed number of cancer cases to the expected number of cases multiplied by 100. An SIR of 100 indicates that the number of cancer cases observed in the population 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 expected and an SIR less than 100 indicates that fewer cancer cases occurred than expected. Accordingly, an SIR of 150 is interpreted as 50% more cases than the expected number; an SIR of 90 indicates 10% fewer 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 4 expected cases and 6 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.

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, a 95% confidence interval (CI) was calculated for each SIR. A 95% CI assesses the magnitude and stability of an SIR. Specifically, a 95% CI is the range of estimated SIR values that has 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 5% percent chance that the observed difference 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), then there is 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), then the number of cancer cases is statistically significantly lower than expected. If the confidence interval range includes 100, then the true SIR may be 100, and it cannot be concluded with sufficient confidence that the observed number of cases is not the result of chance and reflects a real cancer increase or decrease. Statistical significance is not assessed when fewer than five cases are observed.

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 (e.g., 103--115) allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval (e.g., 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.

APPENDIX B

Risk Factor Information for Selected Cancer Types

Brain and Central Nervous System Cancer

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 25% of all reported primary brain tumors and the majority of spinal cord tumors. The majority of meningiomas (about 85%) are benign and can be cured by surgery. In addition to these main types, there are a number of rare brain tumors, including medulloblastomas, which develop from the neurons 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, 2006a). The American Cancer Society estimates that 18,820 Americans (10,730 men and 8,090 women) will be diagnosed with primary brain cancer (including cancers of the central nervous system, or spinal cord) and approximately 12,820 people (7,260 men and 5,560 women) will die from this disease in 2006 (ACS, 2006).

Brain and spinal cord cancers account for over 20% of malignant tumors diagnosed among children aged 0-14 (ACS, 2006b). About half of all childhood brain tumors are astrocytomas and 25% are primitive neuroectodermal tumors (PNET), which spread along the spinal cord and the meninges (ACS, 2006b). 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[)]. 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, 2006a). 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 radiation exposure and increased risk of brain tumors has been

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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, 2006a). 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 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).

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 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

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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, 2006a). 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, 2006b). 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, 2006b). 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).

References

American Cancer Society. 2006. Cancer Facts & Figures 2006. Atlanta: American Cancer Society, Inc.

American Cancer Society. 2006a. Detailed Guide: Brain/CNS Tumors in Adults. Available at: http://www.cancer.org. Cited March 31, 2006.

American Cancer Society. 2006b Detailed Guide:.Brain/Central Nervous System (CNS) Tumors in Children. Available at: http://www.cancer.org. Cited March 31, 2006.

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.

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health
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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.

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.

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Breast Cancer

Breast cancer is the most frequently diagnosed cancer among women in both the United States and in Massachusetts. According to the North American Association of Central Cancer Registries, female breast cancer incidence in Massachusetts is the fifth highest among all states (Chen et al, 2000). Although during the 1980s breast cancer in the U.S. increased by about 4% per year, the incidence has leveled off to about 110.6 cases per 100,000 (ACS 2000). A similar trend occurred in Massachusetts and there was even a slight decrease in incidence (1%) between 1993 and 1997 (MCR 2000).

In the year 2005, approximately 211,240 women in the U.S. will be diagnosed with breast cancer (ACS 2005). Worldwide, female breast cancer incidence has increased, mainly among women in older age groups whose proportion of the population continues to increase as well (van Dijck, 1997). A woman's risk for developing breast cancer can change over time due to many factors, some of which are dependent upon the well-established risk factors for breast cancer. These include increased age, an early age at menarche (menstruation) and/or late age at menopause, late age at first full-term pregnancy, family history of breast cancer, and high levels of estrogen. Other risk factors that may contribute to a woman's risk include benign breast disease and lifestyle factors such as diet, body weight, lack of physical activity, consumption of alcohol, and exposure to cigarette smoke. Data on whether one's risk may be affected by exposure to environmental chemicals or radiation remains inconclusive. However, studies are continuing to investigate these factors and their relationship to breast cancer.

Family history of breast cancer does affect one's risk for developing the disease. Epidemiological studies have found that females who have a first-degree relative with premenopausal breast cancer experience a 3-fold greater risk. However, no increase in risk has been found for females with a first degree relative with postmenopausal breast cancer. If women have a first-degree relative with bilateral breast cancer (cancer in both breasts) at any age then their risk increases five-fold. Moreover, if a woman has a mother, sister or daughter with bilateral premenopausal breast cancer, their risk increases nine fold (Broeders and Verbeek, 1997). In addition, twins have a higher risk of breast cancer compared to non-twins (Weiss et al, 1997).

A personal history of benign breast disease is also associated with development of invasive breast cancer. Chronic cystic or fibrocystic disease is the most commonly diagnosed benign breast disease. Women with cystic breast disease experience a 2-3 fold increase in risk for breast cancer (Henderson et al, 1996).

According to recent studies, approximately 10% of breast cancers can be attributed to inherited mutations in breast cancer related genes. Most of these mutations occur in the BRCA1 and BRCA2 genes. Approximately 50% to 60% of women who inherit BRCA1 or BRCA2 gene mutations will develop breast cancer by the age of 70 (ACS 2001).

Cumulative exposure of the breast tissue to estrogen and progesterone hormones may be one of the greatest contributors to risk for breast cancer (Henderson et al, 1996). Researchers suspect

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that early exposures to a high level of estrogen, even during fetal development, may add to one's risk of developing breast cancer later in life. Other studies have found that factors associated with increased levels of estrogen (i.e., neonatal jaundice, severe prematurity, and being a fraternal twin) may contribute to an elevated risk of developing breast cancer (Ekbom et al, 1997). Conversely, studies have revealed that women whose mothers experienced toxemia during pregnancy (a condition associated with low levels of estrogen) had a significantly reduced risk of developing breast cancer. Use of estrogen replacement therapy is another factor associated with increased hormone levels and it has been found to confer a modest (less than two-fold) elevation in risk when used for 10-15 years or longer (Kelsey, 1993). Similarly, more recent use of oral contraceptives or use for 12 years or longer seems to confer a modest increase in risk for bilateral breast cancer in premenopausal women (Ursin et al, 1998).

Cumulative lifetime exposure to estrogen may also be increased by certain reproductive events during one's life. Women who experience menarche at an early age (before age 12) have a 20% increase in risk compared to women who experience menarche at 14 years of age or older (Broeders and Verbeek, 1997; Harris et al, 1992). Women who experience menopause at a later age (after the age of 50) have a slightly elevated risk for developing the disease (ACS 2001). Furthermore, the increased cumulative exposure from the combined effect of early menarche and late menopause has been associated with elevated risk (Lipworth, 1995). In fact, women who have been actively menstruating for 40 or more years are thought to have twice the risk of developing breast cancer than women with 30 years or less of menstrual activity (Henderson et al, 1996). Other reproductive events have also shown a linear association with risk for breast cancer (Wohlfahrt, 2001). Specifically, women who gave birth for the first time before age 18 experience one-third the risk of women who have carried their first full-term pregnancy after age 30 (Boyle et al, 1988). The protective effect of earlier first full-term pregnancy appears to result from the reduced effect of circulating hormones on breast tissue after pregnancy (Kelsey, 1993).

Diet, and particularly fat intake, is another factor suggested to increase a woman's risk for breast cancer. Currently, a hypothesis exists that the type of fat in a woman's diet may be more important than her total fat intake (ACS, 1998; Wynder et al, 1997). Monounsaturated fats (olive oil and canola oil) are associated with lower risk while polyunsaturated (corn oil, tub margarine) and saturated fats (from animal sources) are linked to an elevated risk. However, when factoring in a woman's weight with her dietary intake, the effect on risk becomes less clear (ACS, 1998). Many studies indicate that a heavy body weight elevates the risk for breast cancer in postmenopausal women (Kelsey, 1993), probably due to fat tissue as the principal source of estrogen after menopause (McTiernan, 1997). Therefore, regular physical activity and a reduced body weight may decrease one's exposure to the hormones believed to play an important role in increasing breast cancer risk (Thune et al, 1997).

Aside from diet, regular alcohol consumption has also been associated with increased risk for breast cancer (Swanson et al, 1996; ACS, 2001). Women who consumed one alcoholic beverage per day experienced a slight increase in risk (approximately 10%) compared to non-drinkers, however those who consumed 2 to 5 drinks per day experienced a 1.5 times increased risk (Ellison et al., 2001; ACS, 2001). Despite this association, the effects of alcohol on estrogen metabolism have not been fully investigated (Swanson et al, 1996).

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To date, no specific environmental factor, other than ionizing radiation, has been identified as a cause of breast cancer. The role of cigarette smoking in the development of breast cancer is unclear. Some studies suggest a relationship between passive smoking and increased risk for breast cancer; however, confirming this relationship has been difficult due to the lack of consistent results from studies investigating first-hand smoke exposure (Laden and Hunter, 1998).

Studies on exposure to high doses of ionizing radiation demonstrate a strong association with breast cancer risk. These studies have been conducted in atomic bomb survivors from Japan as well as patients that have been subjected to radiotherapy in treatments for other conditions (i.e., Hodgkin's Disease, non-Hodgkin's Lymphoma, tuberculosis, post-partum mastitis, and cervical cancer) (ACS, 2001). However, it has not been shown that radiation exposures experienced by the general public or people living in areas of high radiation levels, from industrial accidents or nuclear activities, are related to an increase in breast cancer risk (Laden and Hunter, 1998). Investigations of electromagnetic field exposures in relation to breast cancer have been inconclusive as well.

Occupational exposures associated with increased risk for breast cancer have not been clearly identified. Experimental data suggests that exposure to certain organic solvents and other chemicals (e.g., benzene, trichloropropane, vinyl chloride, polycyclic aromatic hydrocarbons (PAHs)) causes the formation of breast tumors in animals and thus may contribute to such tumors in humans (Goldberg and Labreche, 1996). Particularly, a significantly elevated risk for breast cancer was found for young women employed in solvent-using industries (Hansen, 1999). Although risk for premenopausal breast cancer may be elevated in studies on the occupational exposure to a combination of chemicals, including benzene and PAHs, other studies on cigarette smoke (a source of both chemicals) and breast cancer have not shown an associated risk (Petralia et al, 1999). Hence, although study findings have yielded conflicting results, evidence does exist to warrant further investigation into the associations.

Other occupational and environmental exposures have been suggested to confer an increased risk for breast cancer in women, such as exposure to polychlorinated biphenyls (PCBs), chlorinated hydrocarbon pesticides (DDT and DDE), and other endocrine-disrupting chemicals. Because these compounds affect the body's estrogen production and metabolism, they can contribute to the development and growth of breast tumors (Davis et al, 1997; Holford et al, 2000; Laden and Hunter, 1998). However, studies on this association have yielded inconsistent results and follow-up studies are ongoing to further investigate any causal relationship (Safe, 2000).

When considering a possible relationship between any exposure and the development of cancer, it is important to consider the latency period. Latency refers to the time between exposure to a causative factor and the development of the disease outcome, in this case breast cancer. It has been reported that there is an 8 to 15 year latency period for breast cancer (Petralia, 1999; Aschengrau, 1998; Lewis-Michl, 1996). That means that if an environmental exposure were related to breast cancer, it may take 8 to 15 years after exposure to a causative factor for breast cancer to develop.

Socioeconomic differences in breast cancer incidence may be a result of current screening participation rates. Currently, women of higher socioeconomic status (SES) have higher screening rates, which may result in more of the cases being detected in these women. However, women of higher SES may also have an increased risk for developing the disease due to different reproductive patterns (i.e., parity, age at first full-term birth, and age at menarche). Although women of lower SES show lower incidence rates of breast cancer in number, their cancers tend to be diagnosed at a later stage (Segnan, 1997). Hence, rates for their cancers may appear lower due to the lack of screening participation rather than a decreased risk for the disease. Moreover, it is likely that SES is not in itself the associated risk factor for breast cancer. Rather, SES probably represents different patterns of reproductive choices, occupational backgrounds, environmental exposures, and lifestyle factors (i.e., diet, physical activity, cultural practices) (Henderson et al, 1996).

Despite the vast number of studies on the causation of breast cancer, known factors are estimated to account for less than half of breast cancers in the general population (Madigan et al, 1995). Researchers are continuing to examine potential risks for developing breast cancer, especially environmental factors.

References

American Cancer Society. 2005. Cancer Facts & Figures 2005. Atlanta: American Cancer Society, Inc.

American Cancer Society. 2001. The Risk Factors for Breast Cancer from: http://www3.cancer.org/cancerinfo/print_cont.asp?ct=5&st=pr&language=english

American Cancer Society, 2000. Cancer Facts and Figures 2000.

American Cancer Society. 1998. The Risk Factors for Breast Cancer from: http://cancer.org/bcn/info/brrisk.html

Aschengrau A, Paulu C, Ozonoff D. 1998. Tetrachloroethylene contaminated drinking water and risk of breast cancer. Environ Health Persp 106(4):947-953.

Boyle P, Leake R. Progress in understanding breast cancer: epidemiological and biological interactions. Breast Cancer Res 1988;11(2):91-112.

Broeders MJ, Verbeek AL. Breast cancer epidemiology and risk factors. Quarterly J Nuclear Med 1997;41(3)179-188.

Chen VW, Howe HL, Wu XC, Hotes JL, Correa CN (eds). Cancer in North America, 1993-1997. Volume 1: Incidence. Springfield, IL: North American Association of Central Cancer Registries, April 2000.

Davis DL, Axelrod D, Osborne M, Telang N, Bradlow HL, Sittner E. Avoidable causes of Breast Cancer: The Known, Unknown, and the Suspected. Ann NY Acad Sci 1997;833:112-28.
Ekbom A, Hsieh CC, Lipworth L, Adami HQ, Trichopoulos D. Intrauterine Environment and Breast Cancer Risk in Women: A Population-Based Study. J Natl Cancer Inst 1997;89(1):71-76.

Ellison RC, Zhang Y, McLennan CE, Rothman KJ. Exploring the relation of alcohol consumption to the risk of breast cancer. Am J Epi 2001; 154:740-7.

Goldberg MS, Labreche F. Occupational risk factors for female breast cancer: a review. Occupat Environ Med 1996;53(3):145-156.

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health
March, 2005

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Hansen J. Breast Cancer Risk Among Relatively Young Women Employed in Solvent-Using Industries. Am J Industr Med 1999;36(1):43-47.

Harris JR, Lippman ME, Veronesi U, Willett W. Breast Cancer (First of Three Parts). N Engl J Med 1992;327(5):319-328.

Henderson BE, Pike MC, Bernstein L, Ross RK. 1996. Breast Cancer, chapter 47 in Cancer Epidemiology and Prevention. 2nd ed. Schottenfeld D and Fraumeni JF Jr.,eds. Oxford University Press. pp: 1022-1035.

Holford TR, Zheng T, Mayne ST, Zahm SH, Tessari JD, Boyle P. Joint effects of nine polychlorinated biphenyl (PCB) congeners on breast cancer risk. Int J Epidemiol 2000;29(6):975-982.

Kelsey JL. Breast Cancer Epidemiology. Epidemiol Reviews 1993;15:7-16.

Laden F, Hunter DJ. Environmental Risk Factors and Female Breast Cancer. Ann Rev of Public Health 1998;19:101-123.

Lewis-Michl EL, Melius JM, Kallenbach LR, Ju CL, Talbot TO, Orr MF, and Lauridsen PE. 1996. Breast cancer risk and residence near industry or traffic in Nassau and Suffolk counties, Long Island, New York. Arch Environ Health 51(4):255-265.

Lipworth L. Epidemiology of breast cancer. Eur J Cancer Prev 1995;4:7-30.

Massachusetts Cancer Registry 2000. *Cancer Incidence and Mortality in Massachusetts 1993-1997: Statewide Report*. March 2000. Massachusetts Department of Public Health, Bureau of Health Statistics, Research and Evaluation, Massachusetts Cancer Registry. Boston, MA.

Madigan MP, Ziegler RG, Benichou J, Byrne C, Hoover RN. Proportion of Breast Cancer Cases in the United States Explained by Well-Established Risk Factors. J Natl Cancer Inst 1995;87(22):1681-5.

McTiernan A. Exercise and Breast Cancer—Time To Get Moving? The N Engl J Med 1997;336(18):1311-1312.

Petralia SA, Vena JE, Freudenheim JL, Dosemeci M, Michalek A, Goldberg MA, Brasure J, Graham S. Risk of premenopausal breast cancer in association with occupational exposure to polycyclic aromatic hydrocarbons and benzene. Scandin J Work Envir Health 1999;25(3):215-221.

Safe SH. Endocrine Disruptors and Human Health—Is There a Problem? An Update. Environ Health Perspec 2000;108(6):487-493.

Segnan N. Socioeconomic status and cancer screening. International Agency for Research on Cancer 1997;138:369-376.

Swanson CA, Coates RJ, Malone KE, Gammon MD, Schoenberg JB, Brogan DJ, McAdams M, Potischman N, Hoover RN, Brinton LA. Alcohol Consumption and Breast Cancer Risk among Women under Age 45 Years. Epidemiology 1997;8(3):231-237.

Thune I, Brenn T, Lund E, Gaard M. Physical Activity and the Risk of Breast Cancer. N Engl J Med 1997;336(18):1269-1275.

Ursin G, Ross RK, Sullivan-Haley J, Hanisch R, Henderson B, and Bernstein L. Use of oral contraceptives and risk of breast cancer in young women. Breast Cancer Res 1998;50(2):175-184.

van Dijck JAAM, Broeders MJM, Verbeek ALM. Mammographic Screening in Older Women, Is It Worthwhile? Drugs and Aging 1997;10(2):69-79.

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health
March, 2005

30

Weiss HA, Potischman NA, Brinton L, Brogan D, Coates RJ, Gammon MD, Malone KE, Schoenberg JB. Prenatal and Perinatal Factors for Breast Cancer in Young Women. Epidemiology 1997;8(2):181-187.

Wohlfahrt J, Melbye M. Age at Any Birth is Associated with Breast Cancer Risk. Epidemiology 2001;12(1):68-73.

Wynder E, Cohen LA, Muscat JE, Winters B, Dwyer JT, Blackburn G. Breast Cancer: Weighing the Evidence for a Promoting Role of Dietary Fat. J Natl Cancer Inst 1997;89(11)766-775.

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health
March, 2005

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