Massachusetts
Department
Of
Public Health

Evaluation of Cancer Incidence in Census Tracts 3114 and 3116 in Lowell, Massachusetts

1982-2001

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Bureau of
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Program

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I. INTRODUCTION

At the request of concerned residents, the Lowell Department of Inspectional Services, and Senator Steven C. Panagiotakos, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health conducted an evaluation of cancer incidence for census tracts (CTs) 3114 and 3116 in Lowell (see Figure 1). This evaluation was initiated due to community concerns about a suspected increase in the incidence of cancer specifically in the area surrounding the former Lowell Landfill site, located in CT 3114 on Westford Street in the western part of the city, near the border of CT 3116 (see Figure 2).

This investigation provides a review of the pattern of nine cancer types in CTs 3114 and 3116 in Lowell and compares the incidence of these cancers with the cancer experience of the state of Massachusetts as a whole. Cancer incidence data for Lowell were obtained from the Massachusetts Cancer Registry (MCR) for the years 1982-2001. Two smaller time periods were evaluated, 1982-1991 and 1992-2001, to assess possible trends over time. The nine cancer types selected for this evaluation were based on potential associations with contaminants of concern at the former Lowell Landfill site and/or resident concern over suspected elevations of some cancer types.

In addition to calculating cancer incidence rates, a qualitative analysis of the geographic distribution of individuals diagnosed with each of the nine types of cancer was conducted by mapping their residence at time of diagnosis. This was done to determine whether the geographic pattern of cancer in this area of the city was unusual.

II. METHODS FOR ANALYZING CANCER INCIDENCE

A. Case Identification/Definition

Cancer incidence data (i.e., reports of new cancer diagnoses) for Lowell CTs 3114 and 3116 for the years 1982-2001 were obtained from the MCR, a division of the MDPH Bureau of Health Information, Statistics, Research and Evaluation (BHISRE). The MCR is a population-based surveillance system that began collecting information in 1982 on Massachusetts residents diagnosed with cancer in the state. All newly diagnosed cancer

cases among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111 s.111B).

Nine cancer types were evaluated in this investigation, including cancers of the bladder, brain, breast, kidney, liver, lung and bronchus, pancreas and stomach as well as leukemia. [Coding for cancer types 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 these cancer types.] These cancer types were selected for evaluation based on potential associations with contaminants of concern at the former Lowell Landfill site and/or resident concern over suspected elevations of some cancer types. All diagnoses reported to the MCR as primary cancers among residents of CT 3114 or CT 3116 for the nine cancer types were included in the analysis. Individuals diagnosed with cancer were selected for inclusion based on the address reported to the hospital or reporting medical facility at the time of diagnosis.

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). Therefore, each of the cancer types reviewed in this report was evaluated separately. 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.

It should be noted that duplicate records have been eliminated from the MCR data used in this report. Duplicate cases are additional reports of the same primary site cancer diagnosed in an individual by another health-care provider. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR after consulting with the reporting hospital/diagnostic facility and obtaining additional information regarding the histology and/or pathology of the case. However, reports of individuals with multiple primary site cancers were included as separate cases in this

report. In general, a diagnosis of a multiple primary cancer is defined by the MCR as a new cancer in a different location in the body or a new cancer of the same histology (cell type) as an earlier cancer, if diagnosed in the same primary site (original location in the body) more than 2 months after the initial diagnosis (MCR 2003).

B. Calculation of Standardized Incidence Ratios (SIRs)

To determine whether an elevation occurred among individuals diagnosed with cancer in CTs 3114 and 3116, 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 two time periods, 1982-1991 and 1992-2001, for each of the nine primary cancer types for each CT, in order to evaluate patterns or trends in cancer incidence over time.

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 the two CTs in Lowell (U.S. DOC 1980, 1990, and 2000). Midpoint population estimates were calculated for each time period evaluated (i.e., 1986 and 1996). 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.[1]

A CT is a geographic subdivision of a city or town designated by the United States Census Bureau. Because age group and gender-specific population information is necessary to calculate incidence rates, 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 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). Twenty-seven CTs are within the city of Lowell.

[1] 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.

SIRs were not calculated for some cancer types in the smaller time periods and/or CTs due to the small number of observed cases (less than five). It is standard BHISRE policy not to calculate rates with fewer than five observed diagnoses. However, the expected number of diagnoses was calculated during each time period and for each CT, and the observed and expected numbers of diagnoses were compared to determine whether excess numbers of cancer diagnoses were occurring.

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 diagnoses in an area to the expected number of diagnoses multiplied by 100. The population structure of each town is adjusted to the statewide incidence rate to calculate the number of expected cancer diagnoses. 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. Comparison of SIRs between towns or census tracts is not possible because each community has different population characteristics.

An SIR of 100 indicates that the number of cancer diagnoses observed in the population being evaluated is equal to the number of cancer diagnoses expected in the comparison or "normal" population. An SIR greater than 100 indicates that more cancer diagnoses occurred than were expected, and an SIR less than 100 indicates that fewer cancer diagnoses occurred than were expected. Accordingly, an SIR of 150 is interpreted as 50% more cancer diagnoses than the expected number; an SIR of 90 indicates 10% fewer cancer diagnoses 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 diagnoses indicates a 50% excess in cancer, but the excess is actually only two diagnoses. Conversely, an SIR of 150 based on 400 expected diagnoses and 600 observed diagnoses represents the same 50% excess in cancer, but because the SIR is based upon a greater number of diagnoses, the estimate is more stable. It is very unlikely that 200 excess diagnoses of cancer would occur by chance alone. As a result of the instability of incidence rates based on small numbers of diagnoses, SIRs were not calculated when fewer than five diagnoses 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 diagnoses 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 diagnoses.

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 diagnoses. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45–96), the number of cancer diagnoses 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 diagnoses 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 diagnoses, statistical significance was not assessed when fewer than five diagnoses were observed.

E. Determination of Geographic Distribution of Cancer Cases

In addition to calculating SIRs, the address at the time of diagnosis for each individual diagnosed with one of the nine cancer types in CTs 3114 and 3116 was mapped using a computerized geographic information system (GIS) (ESRI 2005). This allowed assignment of CT location for each individual diagnosed with cancer as well as an evaluation of the spatial distribution of the individuals at a smaller geographic level within CTs (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cancer diagnoses in CTs 3114 and 3116. This evaluation included consideration of the population density variability of each CT through the use of GIS-generated population density overlays. In instances where the address information from the MCR was incomplete, that is, did not include specific streets or street numbers, efforts were made to research those individuals diagnosed with cancer (e.g., by using telephone books issued within 2 years of an individual's diagnosis or searching files via the Registry of Motor Vehicles). 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 state and federal patient privacy and research laws not to reveal the name or any other identifying information of an individual diagnosed with cancer and reported to the MCR.]

III. RESULTS OF CANCER INCIDENCE ANALYSIS

The following sections present cancer incidence rates for the two CTs in Lowell during the 20-year time period 1982-2001. As shown in Figure 2, the former Lowell Landfill was located in CT 3114, near the border with CT 3116. To evaluate possible trends over

time, these data were analyzed by two smaller time periods, 1982-1991 and 1992-2002. Tables 1 and 2 summarize cancer incidence data for CT 3114 for the two time periods, 1982-1991 and 1992-2001, while Tables 3 and 4 summarize cancer incidence data for CT 3116 for each time period. SIRs were not calculated for some cancer types, in these smaller time periods and/or CTs, due to the small number of observed cases (less than five). As previously mentioned, the expected number of diagnoses was calculated during each time period and for each CT, and the observed and expected numbers of diagnoses were compared to determine whether excess numbers of cancer diagnoses were occurring.

Risk factor summaries for each type of cancer evaluated are included in Appendix B.

A. Cancer Incidence in CT 3114, 1982-1991

In general, with some exceptions noted below, the incidence rates of the nine cancer types evaluated in Lowell CT 3114 were approximately at or near the rates expected during 1982-1991 (see Table 1). A slight elevation among females diagnosed with breast cancer was noted, 29 females were diagnosed while 26 would be expected. This elevation is not statistically significant. Additionally, more individuals were diagnosed with cancer of the pancreas in CT 3114 than expected. This elevation was due to an increase of diagnoses among males. Five males were diagnosed with cancer of the pancreas while approximately two would have been expected. This elevation is of borderline statistical significance. For both breast and pancreatic cancer, the differences between the numbers of observed diagnoses compared to the number expected could be due to chance or natural random variation in incidence rates.

B. Cancer Incidence in CT 3114, 1992-2001

During 1992-2001, cancer incidence occurred in CT 3114 near or below expected rates for eight of the nine cancer types (Table 2). The one exception was among males with cancer of the lung and bronchus; 17 males were diagnosed with this cancer type between 1992 and 2001 while approximately 14 males would have been expected. This elevation is not statistically significant.

C. Cancer Incidence in CT 3116, 1982-1991

For the nine types of cancer evaluated, the incidence of cancer in CT 3116 occurred near or below expected rates with one exception (Table 3). Males and females combined were diagnosed with bladder cancer slightly more often than expected. Between 1982 and 1991, fourteen individuals were diagnosed with bladder cancer while about ten individuals would have been expected to have a diagnosis of bladder cancer; this difference, however, is not statistically significant.

D. Cancer Incidence in CT 3116, 1992-2001

Cancer incidence occurred in CT 3116 during 1992-2001 near or below expected rates for eight of the nine cancer types evaluated (Table 4). The exception was bladder cancer which occurred more frequently among males. Eleven males were diagnosed with bladder while approximately six would have been expected; this difference, however, is not statistically significant.

IV. GEOGRAPHIC AND TEMPORAL DISTRIBUTION OF CANCER INCIDENCE IN LOWELL CTS 3114 AND 3116

In addition to determining census tract-specific incidence rates for each of the nine cancer types, a qualitative evaluation of the point pattern of cancer diagnoses was conducted. Place of residence at the time of diagnosis was mapped for each individual diagnosed with the cancer types evaluated in this report, to assess any possible geographic concentrations of diagnoses in relation to each other or in relation to the former Lowell Landfill. In addition, year of diagnosis for each individual was reviewed to determine if a temporal pattern existed among individuals diagnosed with cancer in either CT. As previously mentioned, cancer is one word that describes many different diseases. Therefore, for the purposes of this evaluation, the year of diagnosis for each individual in combination with the geographic distribution of each cancer type was evaluated to determine whether an atypical pattern of any one type of cancer was occurring.

In general, review of the geographic and temporal distribution of individuals diagnosed with cancer for the years 1982-1991 and 1992-2001 in CTs 3114 and 3116 in Lowell did not reveal any unusual spatial patterns or concentrations at the neighborhood level that

suggests a common factor (environmental or non-environmental) played a primary role in cancer diagnoses among residents of these neighborhoods in Lowell. In those instances where further evaluation was necessary, place of residence at diagnosis was found to correlate strongly with the population density patterns of the census tracts.

V. DISCUSSION AND CONCLUSIONS

According to American Cancer Society statistics, cancer is the second leading cause of death in Massachusetts and the United States. Not only will one out of three women and one out of two men 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 neighborhood or town. 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/or 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.

Descriptive epidemiological analyses such as this can be useful in evaluating cancer patterns in a geographic context, assessing if a common cause or etiology is possible, and serving to identify areas where further public health investigations or actions may be warranted. This descriptive analysis of cancer incidence data alone cannot be used to establish a causal link between a particular risk factor (either environmental or non-environmental) and the development of cancer. In addition, this analysis cannot determine the cause of any one individual's cancer diagnosis. The purpose of this

evaluation is to report on the patterns of cancer in two of the census tracts in Lowell and to determine whether such patterns are unusual.

Based on the information reviewed in this report, there does not seem to be an atypical pattern of cancer in CTs 3114 and 3116 in Lowell. As mentioned previously, the nine cancer types evaluated were chosen based on potential associations with contaminants of concern at the former Lowell Landfill site and/or resident concern over suspected elevations of some cancer types. Although there were elevations in some cancer types during certain time periods, in general, the incidence of cancer occurred about as expected when compared to the state as a whole. When elevations did occur, they were not statistically significant, meaning that they could be due to chance and represent natural variability in rates.

In each of the time periods evaluated, the incidence of bladder cancer among male residents of CT 3116 was more than expected, although the incidence was not statistically significantly elevated in either time period. Age and gender are important risk factors in the development of bladder cancer. The risk of bladder cancer increases with age and the average age of diagnosis is 68-69 years. Furthermore, according to the American Cancer Society (ACS), males are more likely to develop bladder cancer than females. During 1982 – 2001, the observed age and gender distribution of individuals diagnosed with bladder cancer in CT 3116 was consistent with this trend, as the average age of diagnosis among CT 3116 residents was approximately 69 years and 78% of individuals diagnosed were males. Cigarette smoking is the most well-established risk factor for the development of bladder cancer. Smoking history was reviewed for each individual diagnosed with this cancer type in CT 3116. Of the 27 individuals diagnosed with bladder cancer during 1982 – 2001, 52% of those with known smoking history were current/former smokers (n = 13). Smoking status was unknown for two individuals. Statewide, 67% of Massachusetts residents diagnosed with bladder cancer were current or former smokers at the time of their diagnosis.

There was a borderline statistically significant elevation in the incidence of pancreatic cancer among males in CT 3114 during the earlier time period, 1982-1991. Risk factors

for this cancer type include age, gender, tobacco use, diet, diabetes mellitus and family history. A review of available information on smoking status for the five males diagnosed with pancreatic cancer in this CT indicated that three of the five males were current smokers at the time of diagnosis. Smoking status was reported as unknown for the remaining two individuals diagnosed with pancreatic cancer. According to the American Cancer Society, men are 20% more likely to develop pancreatic cancer than females (ACS 2006). In Lowell CT 3114, one of the six individuals diagnosed with pancreatic cancer from 1982-1991 was a female, the remainder were males. Information for other risk factors, such as family history and diet, are not contained in the MCR data. In addition, the elevation in pancreatic cancer incidence among males in CT 3114 did not persist over time. For the second time period evaluated, 1992-2001, the incidence of pancreatic cancer among males occurred about as expected in this CT (2 diagnoses observed vs. 1.8 expected).

Due to the proximity of the former Lowell Landfill to two census tracts in Chelmsford, cancer incidence rates for the nine cancer types were also examined for census tracts 3172.03 and 3173.00 in Chelmsford. For both time periods, 1982-1991 and 1992-2001, the numbers of observed diagnoses were close to the number expected; no statistically significant elevations in incidence rates were seen for any of the nine cancer types.

In general, the analysis of the geographic distribution of place of residence for individuals diagnosed with cancer in CTs 3114 and 3116 did not reveal any atypical spatial patterns that would suggest a common factor (environmental or non-environmental) is related to the incidence of cancer in the two CTs during the 20-year time period 1982-2001. Moreover, no unusual concentrations of individuals diagnosed with cancer were observed in the vicinity of the former Lowell Landfill site.

Based on the results of this investigation, the MDPH does not recommend any further evaluation of cancer incidence in Lowell CTs 3114 and 3116.

VI. REFERENCES

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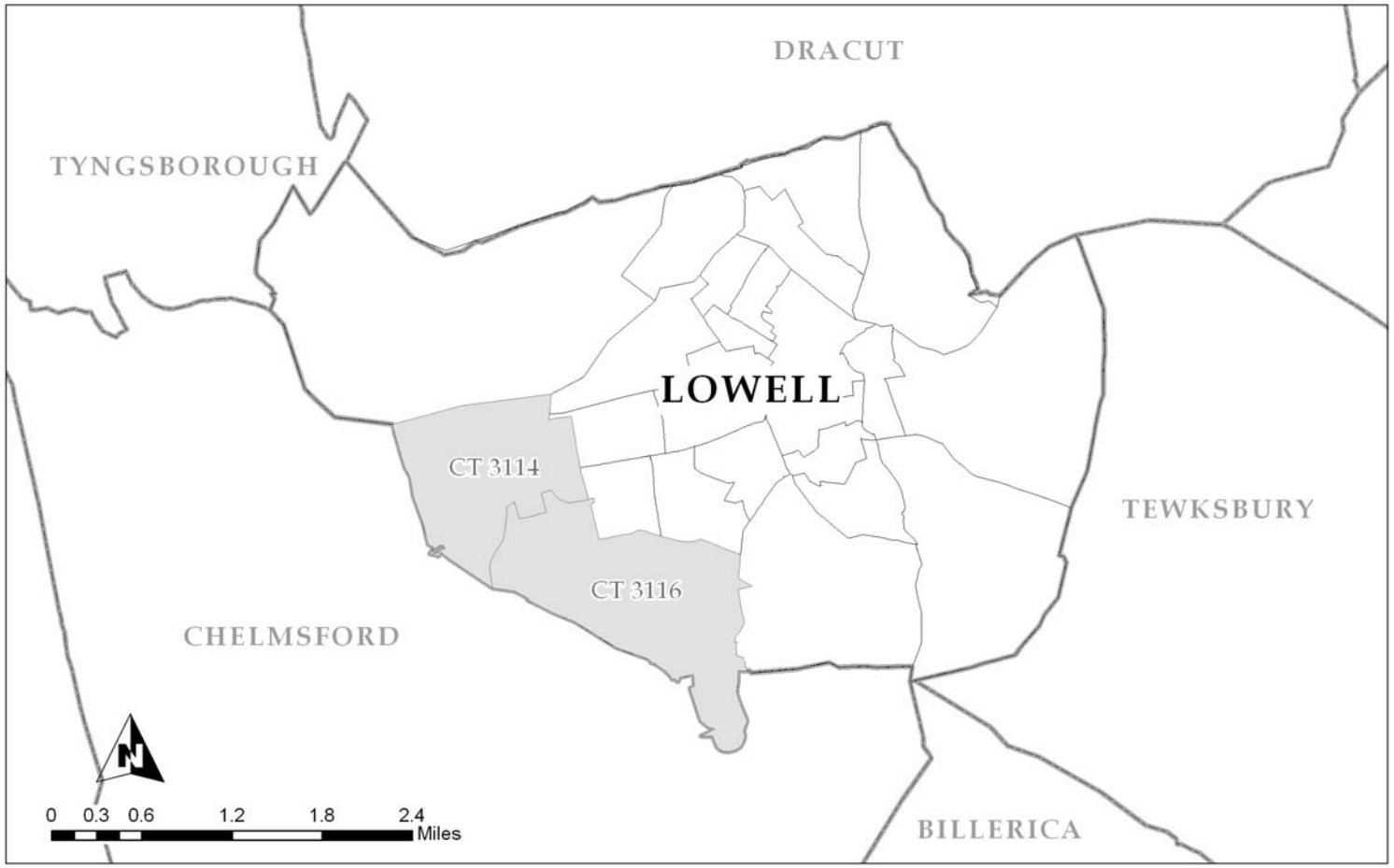
Figures

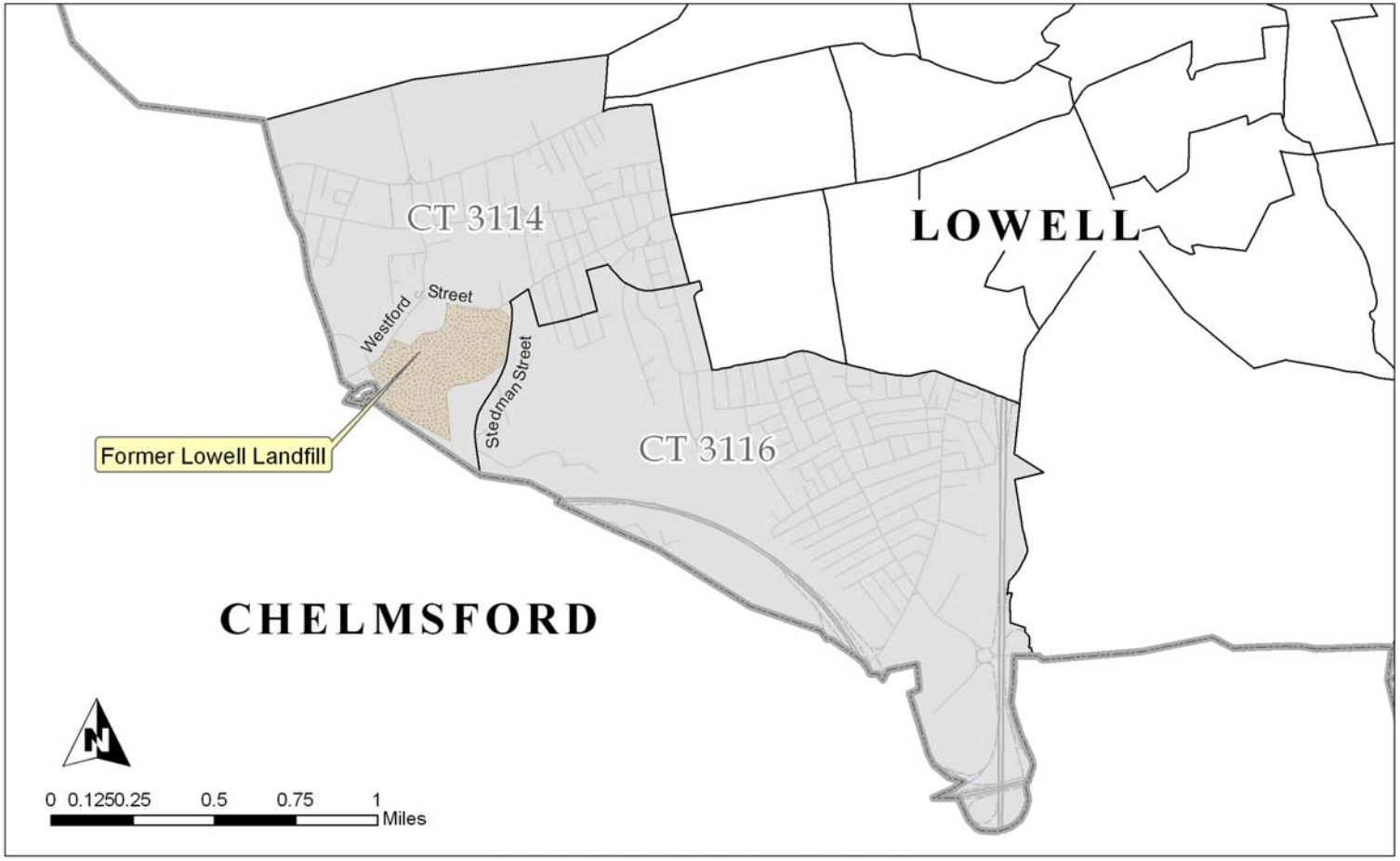
Figure 1
Location of Census Tracts 3114 and 3116
Lowell, Massachusetts



Source data supplied by the Massachusetts Executive Office of Environmental Affairs, MassGIS, Geographic Data Technology, Inc., and U.S. Bureau of the Census

Area of Study
Town Boundary
Census Tract Boundary

Figure 2
Location of the former Lowell Landfill site
Lowell, Massachusetts



Bureau of BEH Environmental Health

Source data supplied by the Massachusetts Executive Office of Environmental Affairs, MassGIS, Geographic Data Technology, Inc., and U.S. Bureau of the Census

Area of Study | Town Boundary | Census Tract Boundary

Tables

TABLE 1
Cancer Incidence
Census Tract 3114
Lowell, Massachusetts
1982-1991

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	5	6.8	74	24 -- 173	4	4.9	NC	NC -- NC	1	1.9	NC	NC -- NC
Brain	2	2.8	NC	NC -- NC	2	1.5	NC	NC -- NC	0	1.3	NC	NC -- NC
Breast	29	26.2	111	74 -- 159	0	0.2	NC	NC -- NC	29	26.0	111	75 -- 160
Kidney/Renal Pelvis	5	3.5	142	46 -- 331	3	2.1	NC	NC -- NC	2	1.4	NC	NC -- NC
Leukemia	2	3.5	NC	NC -- NC	1	2.0	NC	NC -- NC	1	1.5	NC	NC -- NC
Liver	1	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung and Bronchus	20	22.9	87	53 -- 135	15	14.2	105	59 -- 174	5	8.7	58	19 -- 135
Pancreas	6	3.5	173	63 -- 377	5	1.6	308	99 -- 720	1	1.8	NC	NC -- NC
Stomach	4	3.6	NC	NC -- NC	2	2.1	NC	NC -- NC	2	1.4	NC	NC -- NC

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
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

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

TABLE 2
Cancer Incidence
Census Tract 3114
Lowell, Massachusetts
1992-2001

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Bladder	8	5.8	137	59	-- 270	5	4.1	122	39	-- 284	3	1.7	NC	NC	-- NC
Brain	0	3.2	NC	NC	-- NC	0	1.8	NC	NC	-- NC	0	1.4	NC	NC	-- NC
Breast	33	31.3	105	73	-- 148	0	0.2	NC	NC	-- NC	33	31.1	106	73	-- 149
Kidney/Renal Pelvis	4	4.7	NC	NC	-- NC	3	2.8	NC	NC	-- NC	1	1.9	NC	NC	-- NC
Leukemia	1	4.6	NC	NC	-- NC	0	2.5	NC	NC	-- NC	1	2.1	NC	NC	-- NC
Liver	2	1.3	NC	NC	-- NC	1	1.0	NC	NC	-- NC	1	0.4	NC	NC	-- NC
Lung and Bronchus	30	25.8	116	79	-- 166	17	13.7	125	72	-- 199	13	12.1	107	57	-- 183
Pancreas	4	3.9	NC	NC	-- NC	2	1.8	NC	NC	-- NC	2	2.1	NC	NC	-- NC
Stomach	3	3.2	NC	NC	-- NC	2	1.9	NC	NC	-- NC	1	1.3	NC	NC	-- NC

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
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

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

TABLE 3
Cancer Incidence
Census Tract 3116
Lowell, Massachusetts
1982-1991

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	14	9.7	144	79 -- 242	10	7.0	144	69 -- 264	4	2.8	NC	NC -- NC
Brain	3	3.4	NC	NC -- NC	1	1.8	NC	NC -- NC	2	1.6	NC	NC -- NC
Breast	35	37.7	93	65 -- 129	0	0.2	NC	NC -- NC	35	37.5	93	65 -- 130
Kidney/Renal Pelvis	6	5.0	120	44 -- 260	5	3.0	168	54 -- 392	1	2.0	NC	NC -- NC
Leukemia	4	4.4	NC	NC -- NC	2	2.4	NC	NC -- NC	2	2.0	NC	NC -- NC
Liver	0	1.1	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung and Bronchus	29	34.8	83	56 -- 120	19	21.5	88	53 -- 138	10	13.3	75	36 -- 138
Pancreas	7	5.1	137	55 -- 283	2	2.4	NC	NC -- NC	5	2.7	182	59 -- 425
Stomach	3	5.1	NC	NC -- NC	2	3.0	NC	NC -- NC	1	2.1	NC	NC -- NC

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
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

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

TABLE 4
Cancer Incidence
Census Tract 3116
Lowell, Massachusetts
1992-2001

Cancer Type	Total						Males						Females					
	Obs	Exp	SIR	95% CI			Obs	Exp	SIR	95% CI			Obs	Exp	SIR	95% CI		
Bladder	13	8.0	163	87	--	278	11	5.7	194	96	--	346	2	2.3	NC	NC	--	NC
Brain	2	3.5	NC	NC	--	NC	1	1.9	NC	NC	--	NC	1	1.6	NC	NC	--	NC
Breast	36	39.7	91	63	--	126	0	0.3	NC	NC	--	NC	36	39.4	91	64	--	126
Kidney/Renal Pelvis	7	6.1	114	46	--	235	3	3.7	NC	NC	--	NC	4	2.5	NC	NC	--	NC
Leukemia	7	5.4	131	52	--	269	4	2.9	NC	NC	--	NC	3	2.4	NC	NC	--	NC
Liver	2	1.7	NC	NC	--	NC	1	1.3	NC	NC	--	NC	1	0.5	NC	NC	--	NC
Lung and Bronchus	34	36.3	94	65	--	131	18	19.4	93	55	--	146	16	16.8	95	54	--	154
Pancreas	4	5.3	NC	NC	--	NC	2	2.5	NC	NC	--	NC	2	2.9	NC	NC	--	NC
Stomach	2	4.2	NC	NC	--	NC	1	2.5	NC	NC	--	NC	1	1.6	NC	NC	--	NC

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
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

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

APPENDIX A

Cancer Incidence Coding Definitions

APPENDIX A
ICD CODES USED FOR THIS REPORT

Cancer Site / Type	ICD-O-3[1]	
	Primary Site Codes	**Histology Type Codes[2]**
Bladder	C67.0 - C67.9	all except 9590 - 9989
Brain	C71.0 - C71.9	all except 9590 - 9989
Breast	C50.0 - C50.9	all except 9590 - 9989
Kidney/Renal Pelvis	C64.9, C65.9	all except 9590 - 9989
Leukemia	C00.0 - C80.9	includes 9733, 9742, 9800-9820, 9826, 9831-9948, 9963-9964
	C42.0, C42.1, C42.4	includes 9823, 9827
Liver and Intrahepatic Bile Ducts	C22.0, C22.1	all except 9590 - 9989
Lung and Bronchus	C34.0 - C34.9	all except 9590 - 9989
Pancreas	C25.0 - C25.9	all except 9590 - 9989
Stomach	C16.0 - C16.9	all except 9590 - 9989

[1] *International Classification of Diseases for Oncology, 3d Ed.* (2) (includes codes added since publication)

[2] Only invasive cancers (those with invasive behaviors) are included in this report.

APPENDIX B

Risk Factor Information for Selected Cancer Types

Risk Factor Summary for Bladder Cancer

The American Cancer Society estimates that bladder cancer will affect 61,420 people in the U.S. in 2006, accounting for 6% of all cancers diagnosed in the United States among men and 2% among women. In Massachusetts, bladder cancer accounts for approximately 5% of all cancers diagnosed among males and females combined (ACS 2006a). Males are four times more likely to develop bladder cancer than females and whites are two times more likely to develop this disease than blacks. The risk of bladder cancer increases with age and nearly 90% of people with this cancer are over the age of 55 at the time of diagnosis (ACS 2006b).

The greatest risk factor for bladder cancer is cigarette smoking. Smokers are more than twice as likely to develop bladder cancer compared to nonsmokers (ACS 2006a). The risk of developing bladder cancer increases with the number of packs smoked per day and with duration of smoking. Further, the risk of bladder cancer may be higher in women than in men who smoke comparable numbers of cigarettes (Castelao et al. 2001). Approximately 25-60% of all bladder cancers can be attributed to tobacco use (Johansson and Cohen 1997). Smoking cessation has been found to reduce the risk of developing bladder cancer by 30% to 60% (Silverman et al. 1996).

Studies have also revealed a number of occupations that are associated with bladder cancer. In fact, exposures to chemicals in the workplace account for an estimated 20-25% of all bladder cancers diagnosed among men in the U.S. (Johansson and Cohen 1997). Occupational exposure to aromatic amines, such as benzidine and beta-naphthylamine, increases the risk of bladder cancer (ACS 2006b). These chemicals were common in the dye industry in the past. A higher risk of bladder cancer has also been observed among aromatic amine manufacturing workers as well as among workers in the rubber, leather, textiles, printing, and paint products industries (ACS 2006a; Silverman et al. 1996). The development of new chemicals, changed worker exposures, and the elimination of many known bladder carcinogens in the workplace have caused shifts in those occupations considered to be high risk. For example, risks among dye, rubber, and leather workers have declined over time, while other occupations such as motor vehicle operation (e.g., drivers of trucks, buses, and taxis) and the aluminum industry have emerged as potential high-risk occupations (Silverman et al. 1996). However, specific occupational exposures in these occupations have not been confirmed and study findings are not consistent. Further, the risk of bladder cancer from occupational exposures may be increased among smokers (ACS 2006b).

Dietary factors such as consumption of fried foods as well as foods high in fat and cholesterol have been found to be associated with increased bladder cancer risk (Silverman et al. 1996). Use of some anti-cancer drugs (e.g., cyclophosphamide and chlornaphazine), use of phenacetin, and infection with *Shistosoma haematobium* (a parasite found in Africa) are thought to be associated with the development of bladder cancer. However, not all epidemiological studies have produced convincing findings (Silverman et al. 1996).

Other risk factors for bladder cancer include a personal history of bladder cancer, certain rare birth defects involving the bladder, and exposure to ionizing radiation (ACS 2006a; Silverman et al. 1996). Long term exposure to chlorinated by-products in drinking water

has also been suggested to increase the risk of developing bladder cancer, particularly among men (Villanueva 2003).

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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 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 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 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).

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Risk Factor Information for Breast Cancer

Breast cancer is the most frequently diagnosed cancer among women in both the United States and in Massachusetts. According to the American Cancer Society, female breast cancer incidence in Massachusetts is the fourth highest among all states (ACS 2006). The breast cancer incidence rate has been rising in the United States since the 1980s. However, the rate of increase slowed in the 1990s compared to the 1980s. Most recently, breast cancer incidence has only increased in women over 50 years of age (ACS 2006a). A similar trend occurred in Massachusetts and there was even a significant decrease in incidence (2.5%) between 1998 and 2002 (MCR 2005).

In the year 2006, approximately 212,920 women in the U.S. will be diagnosed with breast cancer (ACS 2006). 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 et al. 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 three-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 5 to 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. Women who inherit BRCA1 or BRCA2 gene mutations have up to an 80% chance of developing breast cancer at some point in their lifetimes (ACS 2006).

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 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; ACS 2006). Women who experience menopause at a later age (after the age of 55) have a slightly elevated risk for developing the disease (ACS 2006). 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 and Leake 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 2006; 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. 1997; ACS 2006). 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 2006). Despite this association, the effects of alcohol on estrogen metabolism have not been fully investigated (Swanson et al. 1997).

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 and non-Hodgkin's Lymphoma) (ACS 2006). 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 suggest 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). In particular, 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 occupational exposures 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 et al. 1999; Aschengrau et al. 1998; Lewis-Michl et al. 1996). This 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, 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.

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Kidney cancer involves a number of tumor types located in various areas of the kidney and renal system. Renal cell cancer (which affects the main area of the kidney) accounts for over 90% of all malignant kidney tumors (ACS 2006). The American Cancer Society estimates that there will be approximately 38,890 cases of kidney and upper urinary tract cancer, resulting in more than 12,840 deaths in 2006 (ACS 2006). Kidney cancer is twice as common in males as it is in females and the incidence most often occurs in individuals between 55 and 84 years of age (ACS 2006). The gender distribution of this disease may be attributed to the fact that men are more likely to smoke and are more likely to be exposed to potentially carcinogenic chemicals at work.

Since 1970, U.S. incidence rates for renal cell cancer have risen between 2% and 4% annually among the four major race and gender groups (i.e., white males, white females, black males, and black females) (Chow et al. 1999; McLaughlin et al. 1996). Rapid increases in incidence among blacks as compared to among whites have resulted in an excess of the disease among blacks; age-adjusted incidence rates between 1975 and 1995 for white men, white women, black men, and black women were 9.6, 4.4, 11.1, and 4.9 per 100,000 person-years, respectively (Chow et al. 1999). Rising incidence rates may be partially due to the increased availability of screening for kidney cancer.

The etiology of kidney cancer is not fully understood. However, a number of environmental, cellular, and genetic factors have been studied as possible causal factors in the development of renal cell carcinoma. Cigarette smoking is the most important known risk factor for renal cell cancer. Smoking increases the risk of developing renal cell cancer by about 40% (ACS 2006). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed (Yuan et al. 1998).

Virtually every study that has examined body weight and renal cell cancer has observed a positive association. Some studies suggest that obesity is a factor in 20% of people who develop kidney cancer (ACS 2006). A diet high in protein (meat, animal fats, milk products, margarine and oils) has been implicated in epidemiological studies as a risk factor for renal cell carcinoma (McLaughlin et al. 1996). Consumption of adequate amounts of fruits and vegetables lowers the risk of renal cell cancer. In addition, use of diuretics and antihypertensive medications are associated with increased risk of renal cell carcinoma. However, hypertension has also been linked to kidney cancer and it is not clear whether the disease or the medications used to treat them is the cause (ACS 2000). Long-term use of pain relievers such as phenacetin (and possibly acetaminophen and aspirin) increases the risk for cancer of the renal pelvis and renal cell carcinoma (McLaughlin et al. 1996).

Certain medical conditions that affect the kidneys have also been shown to increase kidney cancer risk. There is an increased incidence of renal carcinoma in patients with end-stage renal disease who develop acquired cystic disease of the kidney. This phenomenon is seen among patients on long-term dialysis for renal failure (Linehan et al. 1997). In addition, an association has been established between the incidence of von Hippel-Lindau disease and

certain other inherited conditions in families and renal cell carcinoma, suggesting that genetic and hereditary risk factors may be important in the development of kidney cancer (ACS 2006; McLaughlin et al. 1996).

Environmental and occupational factors have also been associated with the development of kidney cancer. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. Exposure to cadmium is associated with an increased incidence of kidney cancer, particularly in men who smoke (ACS 2006; Linehan et al. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS 2006). Although occupational exposure to petroleum, tar, and pitch products has been implicated in the development of kidney cancer, most studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between gasoline exposure and renal cancer (Linehan et al. 1997; McLaughlin et al. 1996).

Wilms' tumor is the most common type of kidney cancer affecting children and accounts for approximately 5% to 6% of all kidney cancers and about 6% of all childhood cancers. This cancer is more common among African Americans than other races and among females than males. Wilms' tumor most often occurs in children under the age of 7 years. The causes of Wilms' tumor are not known, but certain birth defect syndromes and other genetic risk factors (such as family history or genetic mutations) are connected with this cancer. However, most children who develop Wilms' tumor do not have any known birth defects or inherited gene changes. No environmental risk factors, either before or after a child's birth, have been shown to be associated with the development of Wilms' tumor (ACS 2006a).

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Risk Factor Information for Leukemia

Leukemia is the general term that includes a group of different cancers that occur in the blood forming organs and result in the formation of abnormal amounts and types of white blood cells in the blood and bone marrow. Individuals with leukemia generally maintain abnormally high amounts of leukocytes or white blood cells in their blood. This condition results in an individual's inability to maintain certain body functions, particularly a person's ability to combat infection.

In 2006, leukemia is expected to affect approximately 35,070 individuals in the United States (20,000 males and 15,070 females) in the United States, resulting in 22,280 deaths. Acute cases of leukemia are slightly more common that chronic, 15,860 and 14,520 respectively. In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2006, representing more than 2% of all cancer diagnoses. There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also a few rare types, such as hairy cell leukemia. In adults, the most common types are AML (approximately 11,700 cases) and CLL (approximately 9,560 cases). Incidences of ALL have increased approximately 1.8% per year since 1988 while incidences of CLL have decreased approximately 1.9% each year since 1988. Leukemia is the most common type of childhood cancer, accounting for about 30% of all cancers diagnosed in children. The majority (74%) of these cases are of the ALL type (ACS 2006a).

While ALL occurs predominantly among children (peaking between ages 2 and 3 years), an elevation in incidence is also seen among older individuals, and 1300 (one-third) of total cases of ALL will occur in adults. ALL risk is lowest for adults aged 25 through 50 and then begins to pick up (ACS 2006b). The increase in incidence among older individuals begins at approximately 40-50 years of age, peaking at about age 85 (Linet and Cartwright 1996). ALL is more common among whites than African Americans and among males than females (Weinstein and Tarbell 1997). Exposure to high-dose radiation (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents) is a known environmental risk factor associated with the development of ALL (ACS 2006b). Significant radiation exposure (e.g., diagnostic x-rays) within the first few months of development may carry up to a 5-fold increased risk of developing ALL (ACS 2006b). However, few studies report an increased risk of leukemia associated with residing in proximity to nuclear plants or occupational exposure to low-dose radiation (Linet and Cartwright 1996; Scheinberg et al. 1997). There is conflicting evidence about whether exposure to electromagnetic fields (EMF) plays a role in the development of ALL, however, most studies to date have found little or no risk (ACS 2006b).

Few other risk factors for ALL have been identified. There is evidence that genetics may play an important role in the development of this leukemia type. Studies indicate that siblings of twins who develop leukemia are at an increased risk of developing the disease. Children with Down's syndrome are 10 to 20 times more likely to develop acute leukemia (Weinstein and Tarbell 1997). In addition, other genetic diseases, such as Li-Fraumeni syndrome and Klinefelter's syndrome, are associated with an increased risk of

developing leukemia. Patients receiving medication that suppresses the immune system (e.g., organ transplant patients) may be more likely to develop ALL (ACS 2006c). ALL has not been definitively linked to chemical exposure, however, childhood ALL may be associated with maternal occupational exposure to pesticides during pregnancy (Infante-Rivard et al. 1999). Certain rare types of adult ALL are caused by human T-cell leukemia/lymphoma virus-I (HTLV-I) (ACS 2006c). Some reports have linked other viruses with various types of leukemia, including Epstein-Barr virus and hepatitis B virus. Still others propose that leukemia may develop as a response to viral infection. However, no specific virus has been identified as related to ALL (Linet and Cartwright 1996). Reports also suggest an infectious etiology for some childhood ALL cases, although a specific viral agent has not been identified and findings from studies exploring contact among children in day-care do not support this hypothesis (Greaves MF 1997; Kinlen and Balkwill 2001; Rosenbaum et al. 2000).

Although AML can occur in children (usually during the first two years of life), AML is the most common leukemia among adults, with an average age at diagnosis of 65 years (ACS 2006d). This type of leukemia is more common among males than among females but affects African Americans and whites at similar rates (Scheinberg et al. 1997). High-dose radiation exposure (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents), long-term occupational exposure to benzene (a chemical in gasoline and cigarette smoke), and exposure to certain chemotherapy drugs, especially alkylating agents (e.g., mechlorethamine, cyclophosphamide), have been associated with an increased risk of developing AML among both children and adults (ACS 2006d). The development of childhood AML is suspected to be related to parental exposure to pesticides and other chemicals, although findings are inconsistent (Linet and Cartwright 1996). Studies have suggested a link between electromagnetic field (EMF) exposure (e.g., from power lines) and leukemia (Minder and Pfluger 2001; Schuz et al. 2001). However, there is conflicting evidence regarding EMF exposure and leukemia and it is clear that most cases are not related to EMF (Kleinerman et al. 2000).

Other possible risk factors related to the development of AML include cigarette smoking and genetic disorders. It is estimated that approximately one-fifth of cases of AML are caused by smoking (Scheinberg et al. 1997). Also, a small number of AML cases can be attributed to rare inherited disorders, such as Down's syndrome (ACS 2006d). Recently, scientists have suggested that a mutation in a gene responsible for the deactivation of certain toxic metabolites may have the ability to increase the risk of acute myeloid leukemia in adults. However, further research is necessary in order to confirm the findings of this study (Smith et al. 2001).

CLL is chiefly an adult disease; the average age at diagnosis is about 70 years (ACS 2006e). Twice as many men as women are affected by this type of leukemia (Deisseroth et al. 1997). While genetics and diseases of the immune system have been suggested as playing a role in the development of CLL, high-dose radiation and benzene exposure have not (ACS 1999; Weinstein and Tarbell 1997). It is thought that individuals with a

family history of CLL are two to four times as likely to develop the disease. Some studies have identified an increased risk of developing CLL (as well as ALL, AML, and CML) among farmers due to long-term exposure to herbicides and/or pesticides (Linet and Cartwright 1996). Although viruses have been implicated in the etiology of other leukemias, there is no evidence that viruses cause CLL (Deisseroth et al. 1997).

Of all the leukemias, CML is among the least understood. While this disease can occur at any age, CML is extremely rare in children (about 2% of leukemias in children) and the average age of diagnosis is 40 to 50 years (ACS 2006f). Incidence rates are higher in males than in females, but unlike the other leukemia types, rates are higher in blacks than in whites in the U.S. (Linet and Cartwright 1996). High-dose radiation exposure may increase the risk of developing CML (ACS 2006f). Finally, CML has been associated with chromosome abnormalities such as the Philadelphia chromosome (Weinstein and Tarbell 1997).

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An estimated 18,510 people in the U.S. (12,600 men and 5,910 women) will be diagnosed with liver and intrahepatic bile duct cancer in 2006, accounting for approximately 1% of all new cancers (ACS 2006). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver and accounts for about 75% of all cases. Rarer forms of malignant liver cancer include the fibrolamellar subtype of HCC, cholangiocarcinoma, and angiosarcomain adults and hepatoblastoma in children. Cholangriocarcinomas account for approximately 10% to 20% of all primary liver cancers and people with gallstones, gall bladder inflammation, chronic ulcerative colitis (long-standing inflammation of the large bowel) or chronic infection with certain types of parasitic worms are at an increased risk for developing this cancer. Hepatoblastoma is a rare cancer that forms usually in children under age 4 and has a 90% survival rate with early detection (ACS 2006a).

In some developing countries, HCC is most common type of cancer diagnosed particularly in East Asia and Africa. Incidence in the United States had been increasing up to 1999. Recently, the rate has become more stable (ACS 2006a). Rates of HCC in the U.S. had increased by 70% during the 1980s and 1990s (Yu et al. 2000). Similar trends were observed in Canada and Western Europe. The primary reason for the higher rates observed during those years was the increase in hepatitis C virus infection, an important factor related to liver cancer (El-Serag 2001; El-Serag and Mason 2000).

Men are at least three times more likely to develop HCC than women. Much of this is likely due to differences in lifestyle factors which increase a person's risk for developing liver cancer (ACS 2006a). Although 85% of individuals diagnosed with liver cancer are between 45 and 85 years of age, the disease can occur in persons of any age (ACS 2006a).

Several important risk factors for liver cancer have been identified. Chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk factors for developing liver cancer (ACS 2006a). It is estimated that 80% of HCC cases worldwide can be attributed to HBV infection (Yu et al. 2000). In the United States, HBV accounts for less than a quarter of the cases and infection with HCV plays a much larger role in the incidence of this cancer. HBV and HCV can be spread through intravenous drug use (e.g., the sharing of contaminated needles), unprotected sexual intercourse, and transfusion of and contact with unscreened blood and blood products. In addition, mothers who are infected with these viruses can pass them on to their children at birth or in early infancy (ACS 2006a).

Cirrhosis is also a major risk factor for the development of liver cancer. Cirrhosis is a progressive disease that is the result of scar tissue formation on the liver, which can lead to cancer. Researchers estimate that 60% to 80% of HCC cases are associated with cirrhosis. However, it is unclear if cirrhosis itself causes liver cancer or if the underlying causes of cirrhosis contribute to the development of this disease (Garr et al. 1997). Most

liver cirrhosis in the U.S. occurs as a result of chronic alcohol abuse, but HBV and HCV are also major causes of cirrhosis (ACS 2006a). In addition, certain inherited metabolic diseases, such as hemochromatosis, which causes excess iron accumulation in the body, can lead to cirrhosis (ACS 2006a). Some studies have shown that people with hemochromatosis are at an increased risk of developing liver cancer (Fracanzani et al. 2001).

Epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, chronic consumption of alcoholic beverages has been associated with liver cancer (Wogan 2000). As noted above, it is unclear if alcohol itself causes HCC or if underlying cirrhosis is the cause (London and McGlynn 1996). However, it is clear that alcohol abuse can accelerate liver disease and may act as a co-carcinogen in the development of liver cancer (Ince and Wands 1999). Long-term exposure to aflatoxin can also cause liver cancer. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be exposed to aflatoxins if they consume contaminated peanuts and other foods that have been stored under hot, humid conditions (Wogan 2000). Vinyl chloride, a known human carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS 2006a; London and McGlynn 1996). These chemicals may also increase the risk of cholangiocarcinoma and HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now strictly regulated in the U.S. (ACS 2006a). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS 2006a; ATSDR 2001).

The use of oral contraceptives by women may also be a risk factor in the development of liver cancer. However, most of the studies linking oral contraceptives and HCC involved types of oral contraceptives that are no longer used. There is some indication that the increased risk may be confined to oral contraceptives containing mestranol. It is not known if the newer oral contraceptives, which contain different types and doses of estrogen and different combinations of estrogen with other hormones, significantly increase the risk of HCC (ACS 2006a; London and McGlynn 1996). Long-term anabolic steroid use may slightly increase the risk of HCC (ACS 2006a). Although many researchers believe that cigarette smoking plays a role in the development of liver cancer, the evidence for this is still inconclusive (Mizoue et al. 2000; London and McGlynn 1996).

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Risk Factor information for Lung Cancer

Lung cancer generally arises in the epithelial tissue of the lung. Several different histologic or cell types of lung cancer have been observed. The various types of lung cancer occur in different regions of the lung and each type is associated with slightly different risk factors (Blot and Fraumeni 1996). The most common type of lung cancer in the United States today is adenocarcinoma which accounts for about 40% of all lung cancers (ACS 2005). The greatest established risk factor for all types of lung cancer is cigarette smoking, followed by occupational and environmental exposures.

The incidence of lung cancer increases sharply with age peaking at about age 60 or 70. Lung cancer is very rare in people under the age of 40. The incidence is greater among men than women (probably because men are more likely to be smokers than women) and among blacks than whites (Blot and Fraumeni 1996). The American Cancer Society estimates that lung and bronchus cancer will be diagnosed in 174,470 people (92,700 cases in men and 81,770 in women) in the U.S. in 2006, accounting for about 12% of all new cancer diagnoses. For purposes of treatment, lung cancer is divided into two clinical groups: small cell lung cancer (13%) and non-small cell lung cancer (87%) (ACS 2006). Lung cancer is the leading cause of cancer death among both men and women; more people die of lung cancer than of colon, breast, and prostate cancers combined (ACS 2005). In Massachusetts, an estimated 4,070 individuals will be diagnosed with lung and bronchus cancer in 2006. Incidence rates for lung and bronchus cancer in Massachusetts from 1998 through 2002 were 86.5 per 100,000 and 60.4 per 100,000 for males and females, respectively (ACS 2006). Nationwide, the incidence rate declined significantly in men during the 1990s, most likely as a result of decreased smoking rates over the past 30 years. Rates for women are approaching a plateau, after a long period of increase. This is likely because decreasing smoking patterns among women have lagged behind those of men (ACS 2006). Trends in lung cancer incidence suggest that the disease has become increasingly associated with populations of lower socioeconomic status, since these individuals have higher rates of smoking than individuals of other groups (Blot and Fraumeni 1996).

Approximately 87% of all lung cancers are caused directly by smoking cigarettes and some of the rest are due to exposure to second hand smoke, or environmental tobacco smoke. The longer a person has been smoking and the higher the number of cigarettes smoked per day, the greater the risk of lung cancer. Smoking cessation decreases the elevated risk and ten years after smoking cessation the risk is reduced by one-third of what it would have been had smoking continued. However, former smokers still carry a greater risk than those who have never smoked. There is no evidence that smoking low tar or “light” cigarettes reduces the risk of lung cancer and mentholated cigarettes are thought to increase the risk of lung cancer. Additionally, breathing secondhand smoke also increases an individual’s risk of developing lung cancer. A nonsmoking spouse of a smoker has a 30% greater risk of developing lung cancer than the spouse of a nonsmoker (ACS 2005).

Workplace exposures have also been identified as playing important roles in the development of lung cancer. Occupational exposure to asbestos is an established risk factor for this disease; asbestos workers are about seven times more likely to die from lung cancer than the general population (ACS 2005). Underground miners exposed to radon and uranium are at an increased risk for developing lung cancer (Samet and Eradze 2000). Chemical workers, talc miners and millers, paper and pulp workers, carpenters, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers are some of the occupations associated with an increased risk of lung cancer (Blot and Fraumeni 1996; Pohlabeln et al. 2000). In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer (ACS 2005; Blot and Fraumeni 1996). Industrial sand workers exposed to crystalline silica are also at an increased risk for lung cancer (Rice et al. 2001; Steenland and Sanderson 2001). Occupational exposure to the compounds noted above in conjunction with cigarette smoking dramatically increases the risk of developing lung cancer (Blot and Fraumeni 1996).

As noted above, exposure to radon (a naturally occurring radioactive gas produced by the breakdown of radium and uranium) has been associated with increased risk of developing lung cancer among miners. Recently, a number of studies have demonstrated that exposure to elevated levels of residential radon may also increase lung cancer risk (Lubin and Boice 1997; Kreienbrock et al. 2001; Tomasek et al. 2001). Epidemiological evidence suggests that radon may be the second leading cause of lung cancer after smoking (Samet and Eradze 2000). However, actual lung cancer risk is determined by cumulative lifetime exposure to indoor radon. Therefore, normal patterns of residential mobility suggest that most people living in high-radon homes experience lifetime exposures equivalent to residing in homes with lower radon levels (Warner et al. 1996).

Some types of pneumonia may increase the risk of lung cancer due to scarred lung tissue (ACS 2002). In addition, people who have had lung cancer have a higher risk of developing another tumor. A family history of lung cancer also increases an individual's risk this is due to an abnormality on chromosome 6 (ACS 2005).

Air pollution may increase the risk of developing lung cancer in some cities. However, this risk is much lower than that due to cigarette smoking (ACS 2005).

Diet has also been implicated in the etiology of lung cancer, however, the exact relationship is unclear. Diets high in fruits and vegetables decrease lung cancer risk, but the reasons for this are unknown (Brownson et al. 1998). A study showed a positive association between total fat, monounsaturated fat, and saturated fat and lung cancer among males, however, this effect was not observed among women (Bandera et al. 1997).

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The American Cancer Society estimates that approximately 33,730 people in the U.S. (17,150 men and 16,580 women) will develop pancreatic cancer in 2006. This disease accounts for approximately 2% of all new cases of cancer in both men and women, but between 5% and 6% of all cancer deaths (ACS 2006a). This discrepancy has been attributed to detection of pancreatic cancer at an advanced stage and the short median survival time for this cancer of approximately three months. Between 1920 and 1965, mortality from this disease increased nearly 200% from 2.9 to 8.2 per 100,000 people. These increases are believed to be due, in part, to improved diagnosis during this time period (Anderson et al. 1996). However, over the past 25 years, incidence rates have declined slowly but consistently in men and a slight decline in rates among women has been observed since the mid-1980s. Further, since the 1970s, men have experienced a slight decrease in mortality from pancreatic cancer, although rates among women have not dropped (ACS 2006a). The risk of developing pancreatic cancer increases with age and the majority of cases occur between age 60 and 80. Men are approximately 20% more likely to develop pancreatic cancer than are women (ACS 2006b).

Very little is known about what causes pancreatic cancer and how to prevent it. However, a number of risk factors have been identified. Besides age, the most consistent and only established risk factor for pancreatic cancer is cigarette smoking. According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS 2006b). Studies have estimated that the risk of pancreatic cancer is two to six times greater in heavy smokers than in non-smokers (Anderson et al. 1996).

Certain medical conditions, such as chronic pancreatitis, diabetes mellitus, and cirrhosis, have been associated with pancreatic cancer, but the reasons for these associations are largely unknown. More recently, a possible role for the bacteria *Helicobacter pylori,* which causes ulcers and some gastric cancers, has been suggested in the development of pancreatic cancer (ACS 2006b; Stolzenberg-Solomon et al. 2001). Some researchers also believe that excess stomach acid may increase the risk of pancreatic cancer (ACS 2006b).

There is also some evidence to suggest that certain dietary factors may be related to the development of pancreatic cancer. Increased risks of pancreatic cancer may be associated with animal protein and fat consumption as evidenced by higher rates of this cancer in countries whose populations eat a diet high in fat (ACS 2006a). Decreased risks for the disease are usually associated with fruit and vegetable consumption (ACS 2006). Obesity is also a risk factor for pancreatic cancer, and very overweight people are 20% more likely to develop pancreatic cancer (ACS 2006b). Although older studies suggested that coffee and alcohol consumption may be risk factors, more recent studies do not support this association (Michaud et al. 2001).

Numerous occupations have been investigated for their potential role in the development of pancreatic cancer, but studies have not produced consistent results. Heavy exposure to

certain pesticides (including DDT and its derivatives) may increase the risk of pancreatic cancer (ACS 2006b; Ji et al. 2001; Porta et al. 1999). Exposure to certain dyes and chemicals related to gasoline, in addition to asbestos and ionizing radiation, has also been associated with the development of pancreatic cancer in some studies. However, other studies have found no link between these agents and pancreatic cancer (ACS 2006b; Anderson et al. 1996). A recent evaluation of data from several studies has implicated organic solvents (e.g., chlorinated hydrocarbons and polycyclic aromatic hydrocarbons), nickel compounds, and chromium compounds in the development of pancreatic cancer, but further studies are needed to corroborate this claim (Ojajarvi et al. 2000). Although occupational exposures may have played a role in the incidence of this cancer in the past, currently most newly diagnosed patients with pancreatic cancer do not have evidence of a specific chemical exposure or relevant occupational history (Evans et al. 1997).

Finally, pancreatic cancer seems to run in some families. According to the American Cancer Society, an inherited tendency to develop pancreatic cancer may account for as many as 10% of cases. Also, inherited DNA mutations that increase risk of developing pancreatic cancer can also increase the risk of developing other cancers. For example, some people with inherited BRCA2 mutations (which increases risk of breast cancer), an inherited tendency for melanoma (skin cancer), or an inherited tendency for colorectal cancer are also at an increased risk of developing pancreatic cancer (ACS 2006b). Pancreatic cancer has been observed in both familial clusterings among siblings as well as in individuals of consecutive generations (Anderson et al. 1996).

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According to the American Cancer Society, approximately 21,260 Americans (13,000 men and 8,260 women) will be diagnosed with stomach cancer in 2007 (ACS 2007). Approximately 90-95% of these cases will suffer from an adenocarcinoma, a cancer which develops within the epithelial cells of the stomach's innermost lining, the mucosa. Less common types of stomach cancer include lymphoma of mucosa-associated lymphoid tissue, gastrointestinal stromal tumors, and carcinoid tumors. The majority of stomach cancers tend to occur in people over the age of 50, with most diagnoses happening after the age of 70. This type of cancer is more common in men than women, and is found more frequently among Asian, Pacific Islander, Hispanic, and African populations than in non-Hispanic white Americans (Shibata / Parsonnet 1996).

Stomach, or gastric, cancer is an increasingly rare form of cancer in the United States. It was once the leading cause of cancer deaths in the United States, yet since the mid-twentieth century, its prevalence has been drastically reduced. It is currently the seventh-leading cause of cancer deaths in the U.S. (NCI 2007). This reduction can be attributed to many factors, including increased refrigeration of foods and decreased consumption of salted and smoked meats. Some physicians feel that it can also be attributed to the widespread use of antibiotics to kill infections, such as *h. pylori*, which may increase one's risk for developing stomach cancer (NCI 2007). Stomach cancer is a much larger problem globally, particularly in underdeveloped nations. It is the second-leading cause of cancer deaths worldwide, with approximately 700,000 deaths in 2002 (ACS 2007).

While the exact cause of stomach cancer is unknown, many risk factors for the disease have been identified. Risk factors for stomach cancer include *h. pylori* infection, which can lead to chronic atrophic gastritis, a possible pre-cancerous change in the lining of the stomach (ACS 2007). *H. pylori* infection can also lead to the formation of peptic ulcers. The majority of people who carry the *h. pylori* bacterium do not develop cancer, but it has been confirmed as increasing one's risk for stomach cancer. This risk may be increased when someone is taking medicines known as histamine antagonists and proton-pump inhibitors (PPIs) to inhibit acid production in the stomach, which may, in turn, allow for increased bacterial growth. Many researchers now suggest eradication of *h. pylori* before beginning these medicines (Shibata / Parsonnet 1996).

Dietary factors may also affect one's risk for developing stomach cancer. Increased risk is associated with higher levels of consumption of smoked and salted fish and meats and pickled vegetables. Diets high in whole grains, fruits, and vegetables which contain vitamins A and C have been shown to reduce the risk of stomach cancer. Recent studies have also found that certain chemicals in barbequed and grilled muscle meats may increase cancer risk. These chemicals, known as heterocyclic amines (HCAs) are formed when muscle meats (beef, pork, fowl, and fish) are cooked at high temperatures for an extended period of time (to a medium-well or well-done temperature). Frying, boiling, and grilling cause the formation of most HCAs, but this effect can be somewhat negated by microwaving meats before cooking them.

Other notable causes for increased risk for stomach cancer include tobacco use. Smoking increases risk for cancers of the upper portion of the stomach closest to the esophagus,

and the rate of stomach cancer is approximately doubled for smokers over nonsmokers (ACS 2007). Obesity has also emerged as a factor contributing to cancer in this area of the stomach.

Medical and familial history may also contribute to one's risk for developing stomach cancer. People who have had previous stomach surgery, such as a gastric bypass or removal of an ulcer, have an increased risk for stomach cancer due to a higher concentration of bacteria in the stomach and potential for reflux of bile from the small intestine, as well as a change in the pH balance of the stomach. According to the American Cancer Society, people with Type A blood have a higher risk for stomach cancer, for unknown reasons (ACS 2007). Pernicious anemia is also noted as a risk factor for stomach cancer. Inherited genetic disorders, such as hereditary nonpolyposis colorectal cancer and familial adenomatous polyposis cause a slightly increased risk for stomach cancer in individuals affected by the inherited gene mutations. Also, people with several first-degree relatives with stomach cancer are more likely to develop the disease.

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