Assessment of Cancer Incidence in Rockland, MA
1982-1994

TABLE OF CONTENTS

Assessment of Cancer Incidence in Rockland, MA
1982-1994

TABLE OF CONTENTS

I. INTRODUCTION

At the request of concerned residents in Rockland, Massachusetts and Senator Michael Morrissey, the Community Assessment Unit (CAU), a program of the Bureau of Environmental Heath Assessment (BEHA) within the Massachusetts Department of Public Health (MDPH), evaluated cancer incidence in the town of Rockland. This evaluation was initiated based on concerns over possible exposures to environmental contamination present at the South Weymouth Naval Air Station (SWNAS), which is a federal facility that was operated by the U.S. Navy from 1941 to 1997 (ATSDR 1999). Part of the SWNAS property lies in the northern part of Rockland, while other parts of the property are located either in or adjacent to three additional communities: Weymouth, Abington, and Hingham (Figure 1). To best determine whether the pattern of cancer appears unusual relative to possible base-related opportunities for environmental exposures, it is necessary to evaluate the entire four-town area. For this reason the CAU is conducting a larger review of cancer incidence data for the towns of Weymouth, Abington, and Hingham in a future report related to the SWNAS. Results from this analysis of Rockland cancer incidence data will be incorporated into the future report, such that the patterns of cancer incidence in all four communities combined relative to the SWNAS can be evaluated. In response to a request from Senator Morrissey's office, however, the MDPH has completed an examination of cancer incidence in Rockland first and that is the primary focus of this report.

II. OBJECTIVES

This evaluation is a preliminary investigation that analyzes descriptive health outcome data for selected cancer types in Rockland. Eleven cancer types were evaluated in this investigation: cancers of the bladder, bone, brain, breast, liver, lung, and pancreas; Hodgkin's disease; leukemia; melanoma; and non-Hodgkin's lymphoma (NHL). These cancer types were selected for evaluation based on results of a previous review of cancer rates in Rockland and the three other towns surrounding the SWNAS (see Appendix A), as well as community concerns over suspected elevations in specific cancer types. The investigation provides a comparison of

the incidence of cancer in Rockland with the incidence of cancer in the state. Using the state of Massachusetts as a comparison population provides stability and replication of incidence rates. The primary focus of this investigation was to provide a profile of the occurrence of the selected cancer types in terms of temporal (or time) and geographic distribution of these cancers in Rockland. In addition, available information related to factors associated with the development of these cancers, including risk factor information, was also evaluated.

Descriptive analyses can be useful in determining whether a common etiology (or cause) is likely and can serve to identify areas where further public health investigations or actions may be warranted. Such actions may include follow-up environmental investigation(s) or public health intervention activities when an excess of well-established risk factors has been identified. Descriptive analyses have inherent limitations in that a causal relationship between cancer and possible risk factors cannot be established. Nevertheless, a descriptive evaluation of data helps to identify patterns about risk factors (e.g., behavioral, opportunities for environmental exposures) in a geographic context. In this report, the analysis of the eleven cancer types is presented and discussed in the context of the available information. As previously mentioned, the findings of this report will be incorporated into a larger investigation of cancer and environmental contamination at the SWNAS.

The specific objectives of this investigation were as follows:

- To examine the incidence rates of selected cancer types in Rockland by smaller geographic areas within the town (i.e., census tracts) to determine whether certain areas have higher or lower cancer rates;

- To evaluate the geographic distribution and spatial pattern of selected cancer types in Rockland by mapping individual cases to determine whether an unusual geographic pattern of cases exists in certain areas of the town or in relation to areas of environmental

concern (e.g., landfills);

- To review available information for cancer cases in Rockland, as reported by the Massachusetts Cancer Registry, related to risk factors for developing cancer; and

- To interpret the results of this evaluation in the context of the available scientific and medical literature on the selected cancer types and to provide a clearer understanding of the pattern of disease in order to decide whether further public health action should be taken.

III. METHODS FOR ANALYZING CANCER INCIDENCE

A. Case Identification/Definition

Cancer incidence data for the years 1982-1994 were obtained for the town of Rockland from the Massachusetts Cancer Registry (MCR) of the MDPH, Bureau of Health Statistics, Research and Evaluation. Massachusetts has a population-based cancer registry. Regardless of which hospital an individual is diagnosed at, their actual residence is recorded in the MCR files. In 1982, the MCR began collecting information on Massachusetts residents diagnosed with cancer. All newly diagnosed cancer cases are required by law to be reported to the MCR within six months of the date of diagnosis (M.G.L. c.111s.111B). The 13-year period 1982-1994, constitutes the period for which the most recent and complete cancer incidence data were available at the time of this analysis.

The observed number of cancer cases in this evaluation was derived from cases reported to the MCR as primary site cancer cases diagnosed in Rockland residents during 1982 through 1994. Cases were selected for inclusion based on the address reported to the hospital or reporting facility at the time of diagnosis.

The term cancer is used to describe a variety of diseases associated with abnormal cell and tissue growth. Primary site (location in the body where the disease originated) and histology (tissue or cell type) classify the different cancer types. Epidemiological studies have revealed that different types of cancer are individual diseases with separate causes, risk factors, characteristics, and patterns of survival (Schottenfeld and Fraumeni 1996). For this reason environmental epidemiologic research generally focuses on temporal or spatial patterns of primary site cancers.

Primary site cancers are cancers that develop not as the result of the transfer of disease from one site or organ to another (metastasis), but as original cancers and locations of development. Metastatic cancers however, are due to the spread of a primary site cancer to another location in the body and are not considered to be separate cancers. Therefore, only primary site cancers were included in this evaluation.

In addition, this report presents data for primary brain cancer only. Primary brain tumors are those tumors that arise from the brain and its coverings. Because of differences in risk factors associated with primary brain cancer and cancers originating from other sites in the central nervous system (the majority of which develop due to congenital disorders), for purposes of this report, only primary brain tumors have been analyzed. The annual MCR reports compile tumors originating in the central nervous system as one group. This is for simplicity in reporting statewide surveillance. This group of tumors includes malignant brain tumors, spinal cord tumors, nerve sheath tumors and benign tumors of the brain and spinal cord. As with other metastatic disease, metastatic brain tumors, which result from other types of primary cancers spreading to the brain, were not included in this analysis (Black 1991).

Occasionally, the MCR database may contain duplicate cases. The data discussed in this report have been controlled for duplicate cases by excluding them from the analyses. However, reports of multiple primary site cancer cases were included. Duplicate cases are additional reports of the same primary site cancer case. A multiple primary cancer case is defined by the MCR as a new tumor of the same histology (cell type) as an earlier cancer, if diagnosed in the

same primary site (original location in the body) more than two months after the initial diagnosis (MCR 1996). MCR staff made the decision that a case was a duplicate and should be excluded from the analyses after consulting with the hospital or reporting facilities and obtaining additional information regarding the histology and/or pathology of the case. One duplicate case of breast cancer was reported to the MCR for the town of Rockland.

B. Calculation of Standardized Incidence Ratios (SIRs)

To determine whether elevated numbers of cancer cases have occurred in Rockland or its census tracts, cancer incidence data were analyzed by age and gender to compare the observed number of cancer cases in each census tract to the number that would be expected based on the statewide cancer experience. Standardized incidence ratios (SIRs) were calculated for the period 1982-1994 for each of the eleven cancer types for the two census tracts and the town as a whole. SIRs were also calculated for the two time periods 1982-1986 and 1987-1994, in order to evaluate temporal trends in cancer incidence.

The census tract (CT) is the smallest geographic area for which cancer rates can be accurately calculated. Specifically, a CT is a smaller statistical subdivision of a city or town. CTs usually contain between 2,500 and 8,000 persons and are designed to be homogenous with respect to population characteristics (US DOC 1990).

An SIR is an estimate of the occurrence of cancer in a population relative to what might be expected if the population had the same cancer experience as some larger comparison population designated as “normal” or average. Usually, the state as a whole is selected to be the comparison population. Using the state of Massachusetts as a comparison population provides a stable population base for the calculation of incidence rates. As a result of the instability of incidence rates based on small numbers of cases, SIRs were not calculated when fewer than five cases were observed.

Specifically, an SIR is the ratio of the observed number of cancer cases to the expected number of cases multiplied by 100. An SIR of 100 indicates that the number of cancer cases observed in the population evaluated is equal to the number of cancer cases expected in the comparison or "normal" population. An SIR greater than 100 indicates that more cancer cases occurred than expected and an SIR less than 100 indicates that fewer cancer cases occurred than expected. Accordingly, an SIR of 150 is interpreted as 50 percent more cases than the expected number; an SIR of 90 indicates 10 percent fewer cases than expected.

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

In order to calculate incidence rates, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated based on 1980 and 1990 U.S. census data for each CT in Rockland (U.S. DOC. 1980, 1990). Midpoint population estimates were calculated for each time period evaluated. 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.

According to the 1980 U.S. Census, the town of Rockland comprised two census tracts that divided the town into northern and southern regions along North Avenue and Webster Street (U.S. DOC. 1980) (Figure 2). CT 5021 is located in the southern portion of the town extending south from North Avenue and Webster Street. CT 5022 is located in the northern portion of the

town and contains a portion of the SWNAS. The towns of Weymouth and Hingham border CT 5022 to the north. During the 1990 U.S. Census, the Census Bureau further divided CT 5021, the southern tract, into two smaller census tracts, CT 5021.01 and CT 5021.02 (Figure 3). In this report, BEHA evaluated cancer incidence for CT 5021 in two ways. First, CT 5021.01 and 5021.02 were combined to calculate SIRs for CT 5021 across the entire 1982-1994 time period. Secondly, SIRs were also calculated for CT 5021.01 and CT 5021.02 separately for the later time period (1987-1994). Therefore, for the later time period 1987-1994, SIRs were evaluated for three smaller geographic areas (CT 5021.01, CT 5021.02 and CT 5022).

C. Calculation of the 95 Percent Confidence Interval (95%CI)

In addition to calculating SIRs, the statistical significance of each SIR was also assessed. A 95 percent confidence interval (95% CI) was calculated for each SIR to determine if the observed number of cases is significantly different from the expected number or if the difference may be due solely to chance (Rothman and Boice 1982). A 95% CI is a method of assessing the magnitude and stability of an SIR. 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 is the result of random fluctuation in the number of observed cancer cases.

For example, if a confidence interval does not include 100 and the interval is above 100 (e.g., 105-130), then statistically this means there is a significant excess in the number of cancer cases. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45-96), then statistically this means that the number of cancer cases is significantly lower than expected. If the confidence interval range includes 100, then the true SIR may be 100, and it cannot be concluded with sufficient confidence that the observed number of cases is not the result of chance and reflects a real cancer increase or decrease. Again, due to the instability of

incidence rates based on small numbers of cases, statistical significance was not assessed when fewer than five cases were observed.

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval (e.g., 103-115) allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval (e.g., 85-450) leaves considerable doubt about the true SIR, which could be much lower than or much higher than the calculated SIR. This would indicate an unstable statistic.

D. Evaluation of Risk Factor Information

Available information reported to the MCR at the time of diagnosis and related to risk factors for cancer was reviewed for each individual diagnosed with one of the eleven cancer types evaluated in this report. Information available from the MCR includes age at diagnosis, histology (cancer cell type), stage of disease, smoking status, and occupation. However, information about personal risk factors (e.g., family history, hormonal events, diet.) that may also influence the development of cancer is not collected by the MCR or any other readily accessible source and therefore could not be evaluated in this investigation.

Staging of breast cancer categorizes the extent of the disease and its spread at the time of diagnosis. The analysis of breast cancer in this report defines stage in four categories: localized, regional, distant, or unknown. Localized breast cancer represents a diagnosis that the tumor is invasive, but the cancer is confined to the breast. Regional indicates that the tumor has spread beyond the organ of origin (breast). A regional stage tumor may have spread to adjacent tissues or organs, lymph nodes, or both. Distant indicates that the cancer has metastasized or spread to organs other than those adjacent to the organ of origin, or to distant lymph nodes of both (MCR 1996). Some of the cases reported to the MCR are reported with an unknown stage. An unknown stage indicates that at the time of reporting, the tumor had not been staged.

E. Determination of Geographic Distribution

The geographic distribution of cancer cases was determined using available address information from the MCR indicating residence at the time of diagnosis. This information was mapped for each individual using a computerized geographic information system (MapInfo 1996). This allowed for the assignment of census tract location for each case as well as an evaluation of the spatial distribution of cases at a smaller geographic level (i.e., census tracts or neighborhoods). The geographic distribution was assessed using a qualitative evaluation of the point pattern of cases within the town and within the two census tracts. In instances where the address information was incomplete (i.e., did not include specific streets or street numbers), efforts were made to research those cases using telephone books and residential lists issued within two years of an individual's diagnosis. Address locations were also confirmed by site visits to the area. Due to Massachusetts laws protecting patient confidentiality, maps of the location of individual cancer cases are not provided in this report.

F. Environmental Concerns

Community environmental concerns regarding opportunities for exposures from the SWNAS and a potential relationship to cancer incidence in the areas surrounding the SWNAS cannot be fully evaluated at this time and therefore are not included in this report. To adequately address potential environmental exposures from the SWNAS, an evaluation of the patterns of cancer in each of the four communities surrounding the former air station (i.e., Abington, Hingham, Rockland and Weymouth) is necessary. This report evaluates the incidence of cancer in the town of Rockland only. Environmental sampling data collected as part of the SWNAS remedial effort will be reviewed and evaluated for its potential relationship to cancer incidence in this area in a future report that evaluates cancer incidence in Rockland and the three other communities adjacent to the SWNAS. This report does, however, review the pattern of cancer incidence relative to other areas of environmental concern in Rockland. These include the

location of known hazardous waste sites in Rockland listed by the Massachusetts Department of Environmental Protection (MDEP), the Suburban Auto site, French's Stream, and landfills located in the town. This information is discussed in section V. later in this report.

IV. RESULTS OF CANCER INCIDENCE ANALYSIS

The following sections present cancer incidence rates for Rockland as a whole and each of its census tracts for the eleven cancer types evaluated. Analysis by census tract or smaller geographic area helps in understanding whether the elevated incidence of a certain cancer type observed townwide may be explained by an increase of cases in a particular geographic area of the town. Cancer incidence data are shown in Tables 1A through 5. Figures 2 and 3 show the location of the Rockland census tracts.

A. Cancer Incidence in Rockland as a Whole

1. 1982-1994

During the 13-year period 1982-1994, cancer incidence overall in Rockland occurred approximately at the rates expected (see Table 1A). No statistically significant difference between the observed versus expected numbers of cases occurred for any of the cancer types evaluated during this time period overall or for males, or females, when evaluated separately.

Although not statistically significantly elevated, three cancer types occurred slightly more often than expected in the town overall. These included cancers of the lung, brain and liver. Lung cancer occurred slightly more often than expected, with 132 cases observed among males and females combined and approximately 120 cases expected (SIR=110). The increase of approximately 10 cases was evenly distributed among males and females. Among males in Rockland, 78 cases of lung cancer were observed versus 72.3 expected (SIR=108). Among

females, 54 cases of lung cancer occurred versus 47.4 cases expected (SIR=114). Neither elevation was statistically significant.

Among males and females combined, 14 cases of brain cancer were observed versus 12.5 cases expected. Brain cancer occurred at approximately the rate expected among males (7 cases observed versus 6.8 cases expected), while among females, approximately one additional case was observed compared to what was expected based on statewide rates (7 cases observed versus 5.8 cases expected).

For liver cancer, six cases occurred among males and females combined versus about four cases expected. Three cases occurred among males (versus 2.7 expected), and three cases occurred among females (versus 1.2 expected).

For the other eight cancer types evaluated, the incidence in the town among males and females combined occurred less often than expected based on statewide rates of these cancers. Overall, 28 cases of bladder cancer occurred versus 32.2 expected. Among males, 24 cases of bladder cancer occurred versus 23 expected, while among females 4 cases occurred versus 9.3 expected.

One case of bone cancer occurred among males in Rockland during the period 1982-1994. Breast cancer among females occurred less often than expected (125 observed versus 135.4 expected). Among males, two cases of breast cancer occurred versus approximately one case expected.

Seven cases of Hodgkin's disease occurred townwide during 1982-1994 (SIR=90). This was slightly fewer than expected (7.7 cases). Three cases occurred among males (versus 4.1 expected), and four cases among females (versus 3.6 expected). For leukemia, 12 cases occurred

among males and females combined during 1982-1994, while about 16 cases were expected based on the statewide experience. Among males, six cases of leukemia occurred versus 8.9 cases expected, while among females six cases occurred versus 7.2 cases expected.

Thirteen cases of melanoma occurred town-wide among males and females combined, while 20.6 cases were expected (SIR=63). Eight cases occurred among males (versus 10.7 expected), and five cases occurred among females (versus about 10 expected). For NHL, 24 cases occurred among males and females combined versus 28.4 expected cases. Among males, 16 cases occurred versus 14.5 cases expected, while among females, 8 cases occurred versus 13.9 expected.

For pancreatic cancer, during 1982-1994 for Rockland as a whole, 11 cases occurred among males and females combined, while about 17 cases were expected. Among males, four cases were observed versus 8 cases expected, and among females, 7 cases were observed versus 9.2 cases expected.

2. Temporal Evaluation 1982-1986 and 1987-1994

Review of cancer incidence in Rockland during two smaller time periods (1982-1986 and 1987-1994) showed predominantly that most cancer types occurred approximately equal to or sometimes less frequently than expected (see Table 1B). With the exception of melanoma during the later time period 1987-1994, no difference between observed and expected numbers of cases was statistically significant. Melanoma in the town overall during 1987-1994 occurred statistically significantly less often than expected (6 observed versus 13.6 expected; SIR=44). During the earlier time period, melanoma occurred at the expected rate among males and females combined (SIR=100).

Lung cancer occurred about as expected during the earlier time period (40 cases observed versus about 41 expected among males and females combined). During the later time period, lung cancer was elevated (92 cases observed versus about 79 cases expected), with the elevation primarily attributed to an elevation among females (43 cases observed versus 32.7 cases expected). Among males, 49 cases of lung cancer occurred during the later time period, with 46.0 cases expected. Neither elevation was statistically significant.

Brain cancer also occurred about as expected during the earlier time period (4 cases observed versus 4.2 cases expected) and slightly more often than expected during the later time period (10 cases observed versus 8.3 cases expected). When examined by gender, brain cancer occurred about as expected for males as well as for females during the earlier time period. During the later time period, 5 cases of brain cancer occurred among males versus 4.5 cases expected, while 5 cases occurred among females versus 3.8 cases expected. Neither difference was statistically significant.

Liver cancer occurred about as expected during the earlier time period (1 case observed versus 1.2 cases expected) and slightly more often than expected during the later time period (5 cases observed versus 2.7 cases expected). For the later time period, there was one excess case among males and also among females.

Bladder cancer occurred slightly more often than expected during the earlier time period (14 cases observed versus 12.4 expected), but less often than expected during the later time period (14 cases observed versus 19.7 cases expected). Among males, 10 cases occurred during the earlier time period versus 8.8 cases expected, while among females, 4 cases occurred versus 3.6 cases expected. During the later time period, no cases of bladder cancer occurred among females (versus 5.6 expected cases), and bladder cancer occurred as expected among males (14 cases observed versus 14.1 cases expected).

Female breast cancer occurred slightly more often than expected during the earlier time period (48 cases observed versus 44.4 cases expected). During the later time period, breast cancer among females occurred less often than expected (77 cases observed versus about 91 cases expected). One breast cancer case occurred among males in each of the two time periods.

Hodgkin's disease occurred slightly more often than expected among males and females combined during the earlier time period (4 observed versus 2.9 expected), and slightly less often than expected during the later time period (3 observed versus 4.9 expected).

Leukemia occurred less often than expected during both time periods among males and females combined. When examined by gender, 4 cases of leukemia among males were observed during the earlier time period versus 3.3 expected, and 2 cases were observed during the later time period versus 5.6 cases expected. Among females, no leukemia cases were observed during the earlier time period (versus 2.7 expected), while during the later time period, 6 cases occurred versus 4.4 cases expected.

NHL occurred about as expected during the earlier time period and less often than expected during the later time period. During the earlier time period, 9 cases occurred versus 8.9 cases expected. Among males, 6 cases were observed versus 4.5 cases expected, while among females, 3 cases were observed versus 4.4 cases expected. During the later time period, 15 cases among males and females combined occurred versus 19.5 expected cases. NHL occurred about as expected among males during the later time period (10 observed versus 10.1 expected), while among females, 5 cases were observed with 9.4 cases expected.

Finally, for pancreatic cancer, fewer cases occurred than expected among males and females combined during both time periods. In the earlier time period, two cases occurred (one each for males and females), while 6.5 cases were expected for males and females combined. During the later time period, 9 cases occurred, while 10.7 cases were expected. When examined

by gender, 3 pancreatic cancer cases occurred among males versus 5 cases expected, while among females, 6 cases occurred versus 5.7 cases expected.

B. Cancer Incidence in Rockland Census Tracts

1. Census Tract 5021

During the 13-year period 1982-1994, most of the cancer types evaluated occurred about as expected among males and females combined in CT 5021, located in the southern portion of Rockland. Similar to the pattern observed for the town as a whole, no statistically significant elevations in any of the cancer types occurred in this census tract (see Table 2A).

Lung, brain and liver cancer occurred slightly more often than expected. The remaining eight cancer types occurred as expected or less often than expected based on statewide rates. Lung cancer was elevated overall in CT 5021 with 80 cases observed versus 68.4 cases expected (SIR=117). The excess was attributed to an increase of about 7 cases among males (48 cases observed versus 41.7 expected) and approximately 5 cases among females (32 cases observed versus 26.6 cases expected). Neither elevation was statistically significant. When evaluated by smaller time period, lung cancer occurred about as expected for males and females combined in this census tract during the earlier time period 1982-1986 (25 observed versus 24.5 expected) and more often than expected during the later time period (55 observed versus 43.8 expected). Refer to Table 2B for a summary of these data. The elevation during the later time period can be attributed primarily to an elevation among females (26 observed versus 17.9 expected). However, the elevation was not statistically significant. Among males, three additional lung cancer cases were observed during the later time period (29 observed versus approximately 26 expected).

Brain cancer occurred slightly more often than expected in CT 5021 during 1982-1994 (9

observed versus 7.6 expected). Among males, 5 cases occurred versus 4.2 expected, and among females, 4 cases occurred versus 3.4 expected. When examined by smaller time periods, brain cancer also occurred about as often as expected (3 observed versus 2.7 expected during 1982-1986 and 6 cases observed versus 5 expected cases during 1987-1994).

Liver cancer occurred more often than expected in this census tract during 1982-1994 (5 observed verses 2.2 expected), but the elevation was not statistically significant. The excess was attributed to slightly more than one case than expected occurring among males as well as females. When examined by smaller timer period, one liver cancer case occurred in the earlier time period (versus 0.7 expected), and 4 cases occurred during the later time period (versus 1.5 expected). The excess in the later time period could be attributed primarily to two additional cases that occurred among males. Among females, one liver cancer case was observed, where less than one case was expected.

Bladder cancer occurred slightly less often than expected during the 1982-1994 period (16 observed versus approximately 18 expected). During the earlier time period, about one excess case of bladder cancer occurred among males and females combined (8 observed versus 7.2 expected), while during the later time period, bladder cancer occurred less often than expected (8 observed versus 10.7 expected). One bone cancer case occurred in this census tract, versus 1.4 expected cases.

Breast cancer among females occurred as expected in this census tract during 1982-1994 (77 observed versus 77.2 expected). During the earlier time period, breast cancer among females occurred more often than expected (31 observed versus 26.3 expected), but this elevation was not statistically significant. During the later time period, breast cancer among females occurred slightly less often than expected (46 observed versus 50.9 expected).

Hodgkin's disease occurred as expected among males and females combined during

1982-1994 (5 observed versus 5 expected). When examined by smaller time periods, Hodgkin's disease occurred slightly more often than expected during the earlier time period, due to an increase among females (3 observed versus 0.9 expected). In the later time period, Hodgkin's disease occurred less often than expected (1 observed versus 3.1 expected), and no cases were observed among females.

Leukemia, NHL, melanoma, and pancreatic cancer also occurred about as often or less frequently than expected for males and females combined in CT 5021 during the 1982-1994 period. When incidence rates for males and females were examined separately, about one additional case of NHL occurred among males (10 observed versus 8.7 expected), but NHL occurred slightly less often than expected among females (5 observed versus 7.6 expected). Leukemia, melanoma, and pancreatic cancer occurred about as often or slightly less frequently than expected for the 1982-1994 period for males and for females.

When examined by smaller time periods, leukemia occurred less often than expected during the earlier time period among males and females combined (2 observed versus 3.7 expected) and approximately at the rate expected during the later time period (6 observed versus 5.8 expected). During the later time period, leukemia occurred slightly less often than expected among males (2 observed versus 3.3 expected) and slightly more often than expected among females (4 cases observed versus 2.5 expected).

Melanoma occurred more often than expected among males and females combined during the earlier time period (6 observed versus 4.3 expected). The excess occurred among females (4 observed versus 2.1 expected). During the later time period, 4 cases of melanoma were observed versus 8.1 expected. No cases occurred among females, where 3.8 cases were expected.

Pancreatic cancer occurred slightly less often than expected during the earlier time period

(2 observed versus 3.7 expected) and about as expected during the later time period (6 observed versus 5.8 expected). When examined by gender, pancreatic cancer occurred about as expected during both time periods for males and for females.

2. Census Tract 5022

Census tract 5022 is located in the northern portion of Rockland and includes part of the SWNAS. Overall in this census tract, almost all cancer types evaluated occurred about as expected among males and females combined during the 1982-1994 period. Refer to Table 3A for a summary of these data. The incidence of bladder cancer occurred less often than expected. Twelve cases of bladder cancer were observed during the 13-year period where 14.3 cases were expected (SIR=84). When examined by gender, bladder cancer occurred about as expected among males (10 cases observed versus 9.9 expected), while about two fewer cases than expected occurred among females (2 observed versus 4.4 expected). When examined by smaller time periods, bladder cancer occurred about as expected during both time periods for males (see Table 3B). For females, bladder cancer occurred about as expected during the earlier time period and no cases of bladder cancer occurred among females during the later time period. No bone cancer occurred in this census tract during 1982-1994.

A total of five brain cancer cases occurred in CT 5022 during 1982-1994 versus 4.9 cases expected. Among males, two cases occurred versus 2.6 expected, while among females, three cases occurred versus 2.3 expected. One case of brain cancer occurred in CT 5022 during the earlier time period versus 1.5 expected cases, while four cases were observed during the later time period versus 3.4 cases expected. Brain cancer occurred about as expected during the later time period among males and among females.

A total of 48 breast cancer cases occurred among females versus 58.2 cases expected (SIR=83). During the earlier time period, 17 cases occurred versus 18.1 expected, and during the

later time period, 31 cases occurred versus 40 expected cases. One case of breast cancer among males occurred in this census tract during the 1982-1994 period.

Two cases of Hodgkin's disease occurred among males and females combined (versus 2.7 cases expected). One case each occurred among males and among females. Both cases occurred during the later time period 1987-1994, when approximately 1.8 cases were expected based on statewide rates of Hodgkin's disease.

Four cases of leukemia occurred in CT 5022 during 1982-1994 versus 6.7 cases expected. Slightly fewer cases occurred among males (2 observed versus 3.6 expected) and among females (2 observed versus 3.1 expected) for this cancer type. When examined by smaller time periods, leukemia occurred about as expected during the earlier time period among males and females combined (2 observed versus 2.4 expected), and less often than expected during the later time period (2 observed versus 4.2 expected). No cases of leukemia occurred among females during 1982-1986, and no cases of leukemia occurred among males during 1987-1994.

Lung cancer occurred about as expected among males and females combined in CT 5022 during 1982-1994 (52 observed versus 51.5 expected). About one fewer case occurred among males (30 observed versus 30.7 expected) and about one more case was observed among females than expected (22 observed versus 20.8 expected). Lung cancer also occurred about as expected during each of the two smaller time periods for CT 5022. During the earlier time period, 15 cases of lung cancer occurred among males and females combined versus 16.4 expected, and during the later time period 37 cases occurred versus 35 cases expected.

The incidence of melanoma, NHL, and pancreatic cancer occurred less often than expected among males and females combined in CT 5022 during 1982-1994. Three cases of melanoma occurred versus 8.2 expected. Nine cases of NHL occurred versus 12 expected. Three cases of pancreatic cancer occurred versus 7.8 cases expected. No pancreatic cancer

occurred among males in this census tract. Among females, three cases of pancreatic cancer were observed versus 4.3 cases expected. When examined by smaller time periods, all three pancreatic cancer cases occurred during the later time period. Melanoma and NHL occurred about as or less often than expected in the two smaller time periods for males and females combined, for males, and for females.

3. Census Tracts 5021.01 and 5021.02

As noted earlier, the 1990 census data divided census tract 5021 into two smaller census tracts, 5021.01 and 5021.02. This section presents the results of cancer incidence data for these two smaller census tracts for the 1987-1994 time period only (Tables 4 and 5). In most instances, the number of cases for specific cancer types was less than five and thus SIRs could not be calculated.

a. Census Tract 5021.01

Cancer incidence in CT 5021.01 (the upper portion of CT 5021) generally occurred as expected during 1987-1994 (see Table 4). Three cancer types occurred slightly greater than expected in this area. These include cancer of the liver, lung and pancreas. Liver cancer was slightly greater than expected among males and females combined (3 observed versus 0.9 expected). Approximately one additional case occurred both among males and among females.

Lung cancer also occurred more often than expected among males and females combined (36 cases observed versus 26 cases expected). Among males, lung cancer was slightly elevated (18 observed versus 15.8 expected), and among females, 18 cases occurred versus 10.2 cases expected. The elevation among females was statistically significant (SIR=176, 95% CI=104-278).

Pancreatic cancer was slightly elevated among males and females combined (5 observed versus 3.4 expected). Among males, 2 cases were observed and 1.7 cases were expected, while

among females, 3 cases were observed and 1.7 cases were expected.

The other eight cancer types evaluated all occurred at approximately the rate expected in CT 5021.01 during 1987-1994. Specifically, four cases of bladder cancer occurred among males and females combined, while 6.5 cases were expected. The rate of brain cancer was equal to the expected rate where three brain cancer cases occurred and three were expected. One case of Hodgkin's disease occurred versus 1.9 cases expected. Three cases of leukemia occurred versus 3.5 cases expected. Three cases of melanoma occurred versus 4.8 cases expected. In addition, three cases of NHL occurred, and 6.6 cases were expected among males and females combined. No cases of bone cancer occurred in this area.

b. Census Tract 5021.02

Cancer incidence data for CT 5021.02 (the lower portion of CT 5021) are presented in Table 5. As with CT 5021.01, cancer incidence in 5021.02 occurred about as expected based on the statewide experience for the majority of cancer types evaluated. Five cancer types had either one or no cancer cases observed among males and females combined for the 1987-1994 time period. These included bone cancer, Hodgkin's disease, liver cancer, melanoma, and pancreatic cancer. Refer to Table 5 for a summary of this data.

Cancers of the bladder and breast occurred slightly less often than expected. Four cases of bladder cancer occurred among males and females combined, with 4.2 cases expected. All four cases occurred among males. Breast cancer among females occurred slightly less often than expected (18 cases observed versus 21.6 cases expected).

Cancers of the brain, leukemia and lung occurred slightly more often than expected based on statewide rates of cancer. Three cases of brain cancer occurred among males and females combined, while two cases were expected. For leukemia, a total of three cases were observed among males and females combined, and 2.3 were expected. All three leukemia cases occurred

among females, where one case was expected. Nineteen cases of lung cancer occurred among males and females combined versus 17.8 cases expected. Among males, 11 cases were observed versus 10.1 expected. Among females, 8 cases of lung cancer were observed in this census tract versus 7.6 expected. In addition, five cases of NHL occurred among males and females combined in this census tract, and 4.5 cases were expected. Three cases occurred among males and two cases occurred among females.

C. Evaluation of Cancer Risk Factor Information

As previously mentioned, cancer is a term that describes a variety of diseases. As such, epidemiological studies have generally shown that different cancer types have different causes, patterns of incidence, risk factors, latency periods (i.e., period between exposure and development of disease), characteristics, and trends in survival. Available risk factor information from the MCR was evaluated for all cancer types included in this report.

1. *Bladder Cancer*

a) Age Distribution

Bladder cancer is a disease that mainly affects white men over the age of 65 (Silverman et al 1996). Approximately two-thirds of all bladder cancer cases occur in individuals age 65 or older. In Massachusetts, bladder cancer was the fourth most common cancer diagnosed among men (MCR 1997). In Rockland, 24 of the 28 bladder cancer cases during 1982-1994 occurred among males, with 61% of all bladder cancer cases in the town over age 65 at diagnosis. In CT 5021, 7 of the 16 cases were diagnosed at age 65 or older (average age of 59 years), while in CT 5022, 10 of the 12 cases were age 65 or older (average age of 72 years). Thus, the age distribution in CT 5021 was slightly younger than in CT 5022.

b) Smoking Status

Smokers have approximately two to three times the risk of developing bladder cancer compared to persons who have never smoked. Sixty-four percent of bladder cancer cases in Rockland were reported as current or former smokers at the time of diagnosis, and an additional 18% had an unknown smoking status (see Figure 4A and 4B). Among those with a known smoking status (i.e., those who reported smoking status as current, former or never smoked), 18 of 23 bladder cancer cases (78%) reported being current or former smokers (see Figure 5). This percentage for Rockland is higher than in the state of Massachusetts (66%).

c) Occupation

Studies have suggested a variety of occupations as being associated with the development of bladder cancer (Silverman et al. 1996). Working in occupations with opportunity for exposure to dyestuffs, rubber, and aromatic amine manufacturing is associated with a high risk of developing bladder cancer due to exposure to the chemicals 2-napthylamine, benzidine or both. Working in other occupations such as leather tanning, painting, motor vehicle operations, and employment in the aluminum industry have been associated to a lesser extent with the development of bladder cancer. However, specific occupational exposures related to this cancer type have not been confirmed and the study findings are not always consistent. Of the 18 cases for which occupation information was available, four reported an occupation that has been suggested to increase the risk of developing this type of cancer. It is therefore possible that occupational exposures may have contributed to the development of bladder cancer in these individuals. However, occupation information was reported as unknown or retired for ten individuals (approximately 36% of bladder cancer cases) and therefore it is difficult to draw any definitive conclusions about the possible contribution of occupational exposures in general among bladder cancer cases in Rockland.

2. Brain Cancer

a) Age Distribution

After a peak among children less than 10 years of age, brain tumor incidence declines and then increases at age 25. After age 75, the incidence of brain tumors levels off (Preston-Martin and Mack 1996). In Rockland, the distribution of brain cancer cases was fairly evenly distributed throughout each of the age groups in the town and in both census tracts. Brain cancer in Rockland primarily occurred among adults. The average age at diagnosis among cases for the town as a whole during 1982-1994 was 57 years. In CT 5021, eight of the nine cases were greater than age 25 at diagnosis, while one childhood brain cancer was reported. In CT 5022, all cases were over 25 years of age.

b) Histology (Cell Type)

In adults, the most common type of brain tumor is glioma, which is a general classification of malignant (or cancerous) tumors that includes a variety of types. Two of the more common types of gliomas are astrocytomas and glioblastoma multiformes. Among the 13 Rockland adults diagnosed with brain cancer during 1982-1994, 12 individuals had a type of glioma, with eight of these individuals having either astrocytoma or glioblastoma multiforme. This pattern of brain cancer histology is consistent with the medical literature.

c) Occupation

The epidemiological literature has provided conflicting results on possible associations between brain cancer and occupational exposures. Specifically, studies on worker exposure to vinyl chloride and chemicals in the petrochemical industry have revealed conflicting results . regarding the association between these chemicals and the development of brain tumors. Studies investigating the possible association between occupational exposure of parents (in particular, paper or pulp-mill, aircraft, rubber, and electric workers) and the onset of brain tumors in their

children have also provided inconclusive results.

When available information from the MCR was examined for Rockland residents diagnosed with brain cancer, occupation for 9 of the 14 cases was reported as "retired," "unknown," or "housewife". Of the remaining five individuals, none reported occupation in any of the above-mentioned industries. Due to the lack of available occupational information, it is impossible to determine the role that occupation may have played in brain cancer incidence in Rockland

3. Breast Cancer

The risk of developing breast cancer can be influenced by a number of factors. Epidemiological studies have determined few well-established risk factors for this cancer type. The most well established risk factors for breast cancer are related to genetics and specific reproductive events in women such as age at first pregnancy, number of births, and age at menopause (Kessler 1992). Other factors such as a woman's age and demographic characteristics (e.g., socioeconomic status) are known to increase breast cancer risk. More recent research on breast cancer has included evaluation of the possible contributions of occupation or environmental factors in breast cancer development.

a) Age

Breast cancer has the highest incidence rate of all cancers among women ages 35 and above, with higher incidence rates in older age groups (Devesa et al. 1995). Breast cancer incidence and age have been shown to be related; incidence increases from age 35 to 45, increases at a slower rate from age 45 to 50, and at a steeper rate in post-menopausal women after age 50 (Kessler 1992). In Rockland, female breast cancer incidence tended to increase with increasing age. Approximately 79% of the individuals diagnosed with breast cancer were greater than 50 years old. The greatest number of cases was observed in the age group 45-64 years of age.

b) Stage of Diagnosis

The stage of cancer as reported to the MCR at the time of diagnosis was reviewed for breast cancer cases in Rockland. Staging categorizes the extent of disease and its spread at the time of diagnosis. Breast cancer survival correlates strongly with a diagnosis of early stage cancer, especially cancer limited to the breast (i.e., local stage or stage I) (ACS 1999). An evaluation of staging information can help to determine whether breast cancer patients in a given area are being diagnosed more frequently at an early or late stage of disease. This information can then be used to identify cancer patterns within communities where further public health intervention may be warranted.

This analysis defines stage in four categories: local, regional, distant or unknown. Local stage breast cancer represents a diagnosis that the tumor is invasive but the cancer is defined to the breast. Regional stage indicates that the tumor has spread beyond the organ of origin (breast). This may include spread to adjacent tissues or organs, lymph nodes, or both. Distant stage indicates that the cancer has spread or metastasized to organs other than those adjacent to the organ of origin, spread to distant lymph nodes, or both (MCR 1996). Some of the cases reported to the MCR are reported with an unknown stage. This indicates that at the time of reporting, the tumor had not been staged.

The greatest percentage of breast cancer cases that occurred in Rockland during 1982-1994 was diagnosed at a local stage (55%). This was slightly less than the percentage of local stage breast cancers observed for the state of Massachusetts (59%). Approximately 30% of breast cancer cases in Rockland were diagnosed at a regional stage; about 7% at a distant stage; and stage at diagnosis was unknown for 8% of breast cancer cases in the town (Figure 6). These figures are fairly similar to those for the state as a whole.

When examined by smaller time periods, local stage diagnoses in Rockland occurred less often during the earlier time period than during the later time period (49% during 1982-1986 versus 59% during 1987-1994). Similar increases in local stage at diagnosis were observed for

the state as a whole (53% during 1982-1986 versus 62% during 1987-1994).

4. Hodgkin's Disease

a) Age

Epidemiologic studies have shown two peaks in the age distribution for Hodgkin's disease. The first peak occurs in young adults, usually between the ages of 15 and 39, and the second peak occurs in adults age 55 years and above. In Rockland as a whole, a total of seven individuals were diagnosed with Hodgkin's disease during 1982-1994. Of these, three were age 15 to 39 and three individuals were aged 55 or older.

b) Occupation

Occupational exposures to woodworkers and workers in the chemical industry are suggested in several epidemiological studies to be associated with the development of Hodgkin's disease. Three of the seven Hodgkin's disease cases among Rockland residents did not have occupational information reported to the MCR. Of the other four individuals, no occupation reported to the MCR was associated with the chemical industry or woodworking.

5. Leukemia

Leukemia is the most common type of cancer diagnosed among children. However, leukemia also occurs among adults, usually of older ages. There are four main types of leukemia with notable differences in the age distribution by type (NCI 1996). Acute lymphocytic leukemia (ALL) is most common among children. Chronic lymphocytic leukemia (CLL) rarely occurs before age 30, after which the incidence increases rapidly with increasing age. The majority of CLL cases (i.e., 90%) occur in people over 50 years old. Acute myelocytic leukemia (AML) displays the highest incidence among young and middle-aged adults. Chronic myelocytic leukemia (CML) can occur at any age, but is most often observed in individuals from

age 30-50 years old. Rates for all types of leukemias are higher in males than in females.

a) Age Distribution

In Rockland, leukemia cases occurred equally among males and females for each of the age groups evaluated. The age distribution of leukemia did not show a pattern that was unexpected when compared to epidemiologic studies of this cancer type (nine of the twelve cases were 40 years or older). The median age of diagnosis among Rockland leukemia cases during 1982-1994 was 55 years. One childhood case was observed (i.e., less than age 19).

b) Histology (Cell Type)

As noted above, there are four major leukemia types. ALL is most common among children and was the type observed for the one case of childhood leukemia observed in Rockland. The majority of CLL cases occur in people over 50 years of age. In Rockland, those individuals diagnosed with CLL were over 50 years of age. AML displays the highest incidence among young and middle-aged adults. In Rockland, the majority of individuals with AML were between the ages of 20 and 60.

6. Liver Cancer

a) Histology

Hepatocellular carcinoma is the most common primary cancer of the liver. Among Rockland residents diagnosed with liver cancer during 1982-1994, four of the six cases had this histology type.

b) Age

The risk of developing liver cancer increases with age but the disease can occur in

persons of any age. Among Rockland residents, all liver cancer cases occurred among individuals aged 55 years or older. The mean age at diagnosis was 73 years.

c) Occupation

Risk factors related to the development of liver cancer include long-term exposure to vinyl chloride. Occupational exposure to vinyl chloride (used to make a variety of plastic consumer products and home furnishings) has been associated with the development of liver cancer. Occupations reported to the MCR for individuals diagnosed with liver cancer in Rockland did not reveal information sufficient to determine whether occupational exposures may have played a role for any of these individuals.

7. Lung Cancer

Although lung cancer was elevated in Rockland as a whole during 1982-1994, the elevation was not statistically significant. The increase in this cancer type can be attributed primarily to lung cancer among residents in CT 5021 (located in the southern portion of the town), particularly during the 1987-1994 period.

a) Age Distribution

The incidence of lung cancer increases sharply with age, peaking at about 60 to 70 years of age. Only two percent of lung cancers occur before the age of 40 (Blot and Fraumeni 1996). Lung cancer cases in Rockland displayed an age distribution that was as expected based on the epidemiologic literature. Nearly 80% of the individuals in Rockland were diagnosed in their 50s, 60s, or 70s. Among males, the median age at date of diagnosis was 64 years, while for females, it was 66 years. Consistent with the literature, less than two percent of Rockland residents were diagnosed with lung cancer at less than 40 years of age.

For CT 5021, where lung cancer was elevated, the median age at diagnosis was 64 years for males and females combined and 62 years for females. Eighty-seven percent of lung cancer cases in CT 5021 were in their 50s, 60s, or 70s.

b) Smoking Status

Lung cancer is strongly associated with a history of smoking. Therefore, smoking status information collected at the time of diagnosis for individuals in Rockland diagnosed with lung cancer was reviewed. The majority of lung cancer cases in Rockland were current or former smokers at the time of diagnosis. For individuals with a known smoking status (i.e., current or former smoker, or never smoked), 110 of 113 individuals (i.e., 97%) diagnosed with lung cancer during 1982-1994 were current or former smokers. As shown in Figure 4A, 85% of lung cancer cases were current or former smokers at the time of diagnosis (an additional 13% had an unknown smoking status). The percent of individuals with lung cancer who were current or former smokers was 5% higher in Rockland than in the state of Massachusetts (97% in Rockland versus 92 % in Massachusetts. The distribution of known smoking status for lung cancer cases in Rockland and Massachusetts is presented in Figure 7.

Lung cancer incidence was elevated among females in Rockland, particularly during the later portion of the 13-year period evaluated and was statistically elevated among females in CT 5021.01 during 1987-1994. For those individuals with a known smoking status, approximately 88% of Rockland women diagnosed with lung cancer during 1982-1986 reported being a current or former smoker (Figure 8). This compares with 87% of Massachusetts women diagnosed with lung cancer during the same time period who reported being a current or former smoker (Figure 8). Among females diagnosed with lung cancer during 1987-1994, 39 of 40 Rockland females reported being a current or former smoker (98%). In comparison to the state of Massachusetts, the distribution of current or former smokers among females was about 9% higher in Rockland than the state (98% in Rockland versus 89% in Massachusetts during the same 1987-1994 period). These data are displayed in Figure 9.

c) Histology (Cell Type)

There are four major histologic types of lung cancers: squamous cell carcinoma, small cell carcinoma, adenocarcinomas, and large cell carcinoma (Higginson et al. 1992; Isselbacher et al. 1994). The incidence of adenocarcinomas has been rising in recent years, while squamous cell carcinoma incidence has been falling (Isselbacher et al. 1994; Thun et al. 1997). Adenocarcinoma is now the most common type of lung cancer in the United States (Thun et. al. 1997). While about 90% of patients with lung cancer of all types have a history of smoking, the non-smoking patient who develops lung cancer usually has adenocarcinoma (Isselbacher et al. 1994). It is important to note however that exposure to second hand smoke may play a role in development of adenocarcinoma in non-smokers.

For Rockland, adenocarcinoma was the most common histologic type of lung cancer reported to the MCR (about 43% of those lung cancers with specific histology classification), followed by squamous cell carcinoma (about 25%). For females, the majority of histology types from Rockland as a whole were adenocarcinoma (about 60%). During 1982-1994, a total of three individuals in Rockland who developed lung cancer reported having never smoked. Consistent with the epidemiological literature, all three individuals had adenocarcinoma histology type.

d) Occupation

Occupational exposures to certain chemicals have also been associated with the development of certain cancers. Industrial workers often have more intense and prolonged exposures to chemicals than does the general population. Occupation as reported to the MCR at the time of diagnosis was reviewed for lung cancer cases among Rockland residents. This information was reviewed to determine whether occupational factors were likely to have contributed to the development of lung cancer in Rockland. While the occupational information reported to the MCR is generally limited to job title and does not include specific job-duty information that could further define exposure potential it is sometimes helpful in determining

the likelihood that occupational exposures may have contributed to disease.

Occupations such as underground miners, chemical workers, paper and pulp workers, carpenters, butchers and meat packers, and shipyard workers are some of the occupations associated with increased risk of developing lung cancer (Blot and Fraumeni 1996). This is a result of exposure to a number of chemical compounds that may be found in these workplaces such as arsenic, asbestos, chloromethyl ethers, chromium, vinyl chloride, and ionizing radiation. In addition, occupational exposure to such compounds in conjunction with cigarette smoking can dramatically increase the risk of developing lung cancer (Blot and Fraumeni 1996). Review of occupational information for lung cancer in the town of Rockland revealed that 36% of cases had occupations reported as retired or unknown. About 10% of lung cancer cases in Rockland reported an occupation that has been found to be associated with the development of this cancer.

8. Melanoma

The incidence of malignant melanoma increases with age and rises steeply until the age of 50. Melanoma does occur frequently among younger individuals but the incidence is highest primarily among middle-aged individuals (Armstrong and English 1996). Melanoma distributes differently by location on the body according to gender. Among females, melanoma occurs more frequently on the lower limbs and among males this cancer occurs more frequently on the trunk (Armstrong and English 1996).

a) Age

The majority of melanoma cases diagnosed in Rockland during 1982-1994 were greater than or equal to 45 years of age (i.e., 62%). The minimum age observed among the cases identified in this town was 20 years and the maximum was 75 years. The median age was 49 years.

b) Anatomic Site

Of the five melanoma cases diagnosed among Rockland females during 1982-1994, the location where the cancer initially occurred was unspecified for two individuals. For the other three cases, one each occurred on the face, lower limb or hip, and upper limb and shoulder. Of the eight melanoma cases diagnosed among males during the same time period, two occurred on the trunk, four on the scalp and neck, and one each on the face and lower limb or neck.

9. NHL

a) Age Distribution

NHL occurs at all ages. However, similar to a number of cancer types, the incidence of this cancer generally increases with age (Scherr and Mueller 1996). In Rockland, NHL occurred as expected with respect to age, with all age groups showing a similar number of NHL cases compared to what would be expected. The average age at diagnosis in Rockland during 1982-1994 was 60 years for males and females combined.

b) Occupation

Some occupations have been associated with an increased risk of developing NHL, specifically occupations related to chemicals or agriculture. Farmers, herbicide and pesticide applicators and grain workers appear to have the most increased risk for development of NHL (Zahm et al. 1990, 1993; Tatham et al, 1997). Studies conducted among agricultural workers have demonstrated increases in NHL among those using herbicides for more than 20 days per year and individuals who mix or apply herbicides. A greater incidence of NHL appears to be related specifically to exposure to the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) and organophosphate insecticides (Wigle et al. 1990; Zahm et al 1990, Zahm et al. 1993). Further studies of exposure to these chemicals and NHL incidence have shown that the increased risk is attributed to a specific impurity, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, present in these herbicides. However, reports of accidental industrial exposures to TCDD have not demonstrated an increased risk of NHL (Scherr and Mueller 1996). An elevated risk for NHL

development has also been noted among fence workers, orchard workers and meat workers. Review of occupational information for NHL cases diagnosed in Rockland did not reveal any occupations that have been suggested as associated with NHL.

10. Pancreatic Cancer

The etiology (or cause) of pancreatic cancer remains largely unknown. The only established risk factor for developing pancreatic cancer is cigarette smoking, which the American Cancer Society estimates is responsible for about 30% of pancreatic cancer cases (ACS 1998). Many other risk factors have been suggested, but investigations have provided inconsistent results.

a) Age Distribution

The most reliable and important known predictor of pancreatic cancer is age. In the first three decades of life, this cancer is extremely uncommon. The median age at diagnosis in the United States is 71 years, and the majority of cases occur between age 65 and 79 (Anderson et al. 1996). Individuals over age 80 experience an increased risk of developing pancreatic cancer approximately 40 times greater than those individuals at age 40.

The median age of the 11 pancreatic cancer cases occurring in Rockland during 1982-1994 was 65 years. Consistent with the literature, more (6 of 11 cases) Rockland pancreatic cancer cases were diagnosed between the ages of 65 and 79, and no individual was diagnosed with pancreatic cancer before their 50s.

b) Smoking Status

As shown in Figures 4A and 4B, the percentage of pancreatic cancer cases in Rockland that were current or former smokers was less than that of the state (i.e., 27% versus 43%). However, the percent of pancreatic cancer cases with an unknown smoking status was fairly

large for Rockland as compared to the state (64% versus 23%). Only 4 of the 11 pancreatic cancer cases in Rockland had a known smoking status reported to the MCR. Three of the four pancreatic cancer cases reported being current or former smokers.

c) Occupation

Excess pancreatic cancer deaths have been observed in some occupations where exposure to chemicals or petroleum has occurred. These include manufacturers of photographic film, chemists, leather tanners and automobile mechanics (Anderson et al. 1996). Although some studies have demonstrated an increased number of deaths due to pancreatic cancer among individuals employed in these occupations, further analytical studies have not confirmed these associations (NCI 1996). In Rockland, occupations for 8 of the 11 cases of pancreatic cancer were reported as unknown, retired, or at home. None of the remaining three individuals had an occupation reported in one of the occupations suspected of contributing to pancreatic cancer.

D. Analysis of Geographic Distribution

Place of residence at the time of diagnosis was geocoded and mapped for each of the eleven cancer types to assess any possible geographic pattern of cases. In addition to quantitatively determining census-tract-specific incidence ratios for each cancer type, a qualitative evaluation was conducted to determine whether any specific cancer type appeared to be concentrated in some area(s) for the town as a whole or within either census tract in Rockland.

Review of these data showed that there were no apparent spatial patterns of any specific cancer type at smaller geographic levels (e.g., neighborhoods within the census tracts of Rockland) that was not likely related to the presence of such factors as a more densely populated area of the census tract. For example, lung cancer appeared more concentrated geographically in the northern portion of CT 5021, but this area is the center of town and more densely populated. As a result, it is reasonable to expect more cases in this area of the census tract.

V. COMMUNITY ENVIRONMENTAL CONCERNS

Community environmental concerns include the South Weymouth Naval Air Station (SWNAS), the Suburban Auto site, located at 200 VFW Drive, and possible concentrations of cancer cases along French's Stream (running generally north-south through the length of Rockland). In addition, residents have expressed concern regarding a suspected increase in cancer near two landfills in the town. The geographic pattern of cancer incidence in relation to these areas in Rockland is discussed below.

A. South Weymouth Naval Air Station (SWNAS)

As mentioned earlier in this report, primary environmental concerns in Rockland have focused on the SWNAS and the pattern of cancer as it relates to this area. The SWNAS is either located in or adjacent to the four towns: Abington, Hingham, Rockland and Weymouth. In order to provide an accurate evaluation of cancer incidence in relation to the SWNAS, it is necessary to examine the pattern across all four communities adjacent to the site. A follow-up report evaluating cancer in the three other towns adjacent to the SWNAS (i.e., Abington, Hingham, and Weymouth) as well as potential environmental exposures at this site is expected to be complete later this year. The follow-up report will also include a more complete evaluation of the pattern of cancer in Rockland in relation to the SWNAS. Therefore, it is not possible to reach conclusions about the pattern of cancer in Rockland in relation to potential exposures from the site at this time.

Although conclusions about the true pattern of cancer incidence and potential exposures from this site are not able to be determined, the general pattern of cancer in this area of Rockland during the period 1982-1994 was reviewed. This information showed that in the census tract adjacent to the SWNAS (i.e., CT 5022), each of the cancer types evaluated occurred at or below the expected rate. The majority of cancer types evaluated in Rockland (eight of the eleven types reviewed) occurred less often than expected. For the three cancer types in which some elevations were noted (brain, liver and lung cancer), the elevations occurred in CT 5021, the census tract located in the southern portion of Rockland (i.e., not the census tract that contains

the SWNAS).

Additionally, concern has been raised about suspected increased cancer incidence in the Rockland neighborhoods in CT 5022 nearest to the SWNAS. These concerns focus on the pattern of cancer in relation to runway locations and predominant wind patterns (i.e., neighborhoods located at the southeast portion of the SWNAS in the vicinity of Union Street) as well as the potential for off-site migration of contaminants from the SWNAS. Concerns have been raised about potential contamination in French's Stream, located on the western side of the SWNAS, and Old Swamp River, located on the eastern side of the SWNAS.

A review of the pattern of cancer incidence in neighborhoods in CT 5022 nearest the SWNAS did not demonstrate a geographic concentration of any specific cancer type. The predominant wind direction measured at the SWNAS is from the south-southwest, towards the general direction of Weymouth and Hingham (NRCC 2000). Further, the cancers that were observed among residents in these areas were various types and did not occur in any discernable pattern spatially or with respect to year of diagnosis, which would indicate a temporal trend.

The CAU also reviewed the pattern of cancer incidence in Rockland in relation to concerns over suspected exposures from French's stream. French's stream originates at the southern portion of the SWNAS (CT 5022) and flows south along the Rockland and Abington town border. The stream continues to flow in a southeasterly direction through the lower half of the town of Rockland towards Hanover. The stream has been known to overflow its banks during periods of heavy rain. In 1986, a spill of 6,000 gallons of jet fuel from the SWNAS discharged to French's Stream (Stone & Webster 1996). Limited historical sampling data is available for French's Stream (ATSDR 1999). However, when the pattern of cancer incidence in Rockland was reviewed in relation to French's stream, there was no specific geographic pattern of any one cancer type observed in neighborhoods bordering French's stream in either CT 5022 or 5021.

B. Suburban Auto

The Suburban Auto site is located at 200 VFW Drive in CT 5022 (see Figure 10, location #10) . The site is a former auto salvage and repair location that is currently used as a metal scrap yard. In 1989, a tire fire occurred at the site. After the fire was extinguished, a sheen was observed on water leaving the site and draining into a nearby wetland and on-site stream (Coler & Colantonio 1999). As a result of the fire, approximately 300 cubic yards of contaminated soils from the fire area were stockpiled on the southern portion of the site. The stockpiled soils were subsequently removed to a landfill in 1997 (Coler & Colantonio 1999). A Phase I environmental investigation of the site was completed in the fall of 1999. Sampling conducted to date at the site has revealed that metals, particularly lead, volatile organic chemicals (VOCs), polychlorinated biphenyls (PCBs) and petroleum-based hydrocarbons have been detected in the site soils. Some metals and VOCs have also been detected in groundwater samples from the site (Coler & Colantonio 1999).

Review of the geographic distribution of cancer cases in CT 5022 and particularly the area near the Suburban Auto site did not indicate an unusual pattern. The cancer types that were observed in this area and the neighborhoods surrounding the site were various types that occurred over the entire 13-year period evaluated. No one cancer type displayed a geographic concentration. However, additional environmental investigations are planned at the Suburban Auto site. The Bureau of Environmental Health Assessment is currently evaluating environmental information specific to this site and possible health impacts, including non-cancer related health outcomes in a separate report.

C. Potentially Hazardous Waste Sites (21E Sites)

The most recent information regarding potentially hazardous waste sites located in the town of Rockland was obtained from the Massachusetts Department of Environmental Protection (MDEP), Bureau of Waste Site Cleanup (BWSC), Sites Transition List (MDEP 1998). The MDEP is responsible for the monitoring, assessment, and clean up of releases of oil and hazardous materials at disposal sites (subsequently named 21E Sites) in Massachusetts. These

sites are regulated under Massachusetts General Laws, Chapter 21E and the Massachusetts Contingency Plan (MCP) 310 CMR 40.0000 (MDEP 1997).

A total of 28 sites in Rockland were identified by the MDEP as Confirmed disposal sites or Locations to be Investigated (LTBI). The status of one of these sites was subsequently modified to indicate that the location was not a disposal site or did not currently require further remedial response actions. Of the remaining 27 sites, 10 are in some phase of confirmation status, 8 are listed as LTBI, 4 have waiver status, and 5 were undetermined. Refer to Figure 10 for the location of MDEP 21E sites listed prior to October, 1993.

Confirmed status is a location confirmed by the MDEP to be a disposal site, and for which remedial response actions have not yet been completed. LTBIs are locations the MDEP considers reasonably likely to be disposal sites, but are as yet unconfirmed. Waiver status sites are locations confirmed by the MDEP to be non-priority disposal sites, and where an interested party has been authorized to proceed with response action without MDEP oversight.

MDEP 21E sites are also classified by the type of contamination present at the site. Sites may be listed as having petroleum contamination (i.e., diesel, gasoline, fuel oils), hazardous contamination (i.e., metals, organics, volatile organics, pesticides), or both (a combination of petroleum and hazardous materials). Of the 27 sites, nine are listed as having petroleum contamination, 10 are listed as having hazardous contamination, four are listed as having both, and information was not available for the four remaining sites.

The geographic distribution of each primary site cancer was examined relative to the locations of all of these sites. No unusual patterns or concentrations of cancer cases with respect to type of cancer, age or other factors were observed in Rockland in relation to the 21E sites evaluated.

D. Landfills

Additional concerns were expressed about two landfills in Rockland, one of which is located at the southeastern corner in CT 5021 and the other in the central part of CT 5022. Refer to Figure 11 for the location of the two landfills. Information about these landfills was obtained from conversations with MDEP staff, as well as file reviews at MDEP. The Beech Street landfill (in CT 5021) was a municipal solid waste landfill and received wastes from 1976 to 1995 (MDEP 1999a). No dumping of hazardous waste has been documented at this landfill, but traces of solvents have been detected on the older portion of the site. In 1995 it was closed due to changes made in state landfill regulations and was subsequently capped in 1997 (MDEP 1999b). Currently, the site is the location of a waste transfer station (a facility that handles but does not dispose of solid waste) and a compost site (a registered yard waste composting site).

The landfill in CT 5022 is located on Pleasant Street and VFW Drive. The landfill was formerly a municipal solid waste landfill that was shut down and assigned inactive status in 1976 (MDEP 1999b). No information was available in the MDEP files on when the landfill began operations. Inactive landfills are landfills that are no longer in operation and were not closed properly. Files about this landfill were not located at MDEP, and thus little information about the landfill is available (MDEP 1999c).

Cancer incidence data were reviewed to assess possible geographic patterns in relation to these areas. This evaluation included particular emphasis to both lung and liver cancer patterns in CT 5021 (and CT 5021.01) due to the observed elevations in this area of Rockland during 1987-1994. Careful review did not reveal any unusual geographic pattern of cancer cases in relation to cancer type, age or other factors.

VI. DISCUSSION

A. Discussion of Cancer Incidence and Related Risk Factors

In the town of Rockland as a whole, eight of the eleven cancer types evaluated occurred less often than expected. With the exception of lung cancer among females in CT 5021.01, no cancer type was elevated at a level that was statistically significant in comparison to statewide rates of cancer during any of the time periods evaluated or smaller geographic areas (i.e., census tracts) evaluated. Melanoma incidence occurred significantly less often than expected in Rockland during the time period 1987-1994. Both males and females in the town experienced melanoma less often than expected but the observed decrease was more pronounced among Rockland females where one case occurred and approximately seven cases were expected.

With respect to the pattern of cancer incidence in the town of Rockland, review of available information related to risk factors for the individual cases demonstrated that most cancer types in Rockland displayed a pattern typical of the expected pattern based on current epidemiologic studies of cancer. In general, the risk of developing cancer increases with increasing age and the majority of cancers in Rockland during the 13-year time period were diagnosed among individuals in older age groups. Two cases of childhood cancer (i.e., less than age 19) were diagnosed in Rockland during the time period evaluated. One individual lived in CT 5021 and one individual lived in CT 5022.

With the exception of pancreatic cancer, for which smoking status was unknown for the majority of individuals, cancers associated with a history of smoking showed a greater percentage of current or former smokers in Rockland than observed in the state. This was particularly true among female lung cancer cases during the more recent time period evaluated (1987-1994), where the percentage of individuals with a history of smoking was nearly 10% greater than the state.

Occupational information available from the MCR for individuals diagnosed with cancer

is fairly limited in that only job title is reported and for a large number of individuals occupation is reported as retired or unknown. However, even though the limited information available does not allow for a clear determination of how likely occupational exposures may have contributed to cancer incidence in the town, the information did indicate that some individuals in Rockland were employed in occupations associated with certain cancers (e.g., bladder and lung cancer).

Although slightly elevated in the town overall, the incidence of lung cancer was predominantly elevated in CT 5021, the southern census tract. During the time period 1987-1994, a statistically significant elevation was observed among females in CT 5021.01. Although some occupational and environmental exposures have been suggested as associated with lung cancer, this cancer type is primarily caused by a history of smoking. In Rockland, the majority of individuals diagnosed with lung cancer (approximately 97%) reported a history of smoking at the time of diagnosis. This percentage was approximately 5% greater than observed in individuals diagnosed with lung cancer in the rest of the state. Further, the elevation in lung cancer was principally among females in the more recent time period (i.e., 1987-1994). This gender pattern is not unexpected as rates of lung cancer have increased among women in both Massachusetts and the United States.

The increasing trend of lung cancer rates in females is largely due to the smoking habits among women in general. As women began smoking in larger numbers and at a later time period than men, given the long latency (or development) period for lung cancer, the incidence correspondingly has increased among women during later time periods. In Rockland, females diagnosed with lung cancer during the later time period 1987-1994 were primarily current or former smokers at diagnosis (approximately 98%). Of note, is that this percentage was slightly higher than the percentage of current or former smokers among male lung cancer cases in the town (98% among females vs. 96% among males). Further, the percentage of female lung cancer cases with a reported history of smoking was nearly 10% greater than female lung cancer cases in the state (98% in Rockland vs. 89% in Massachusetts). During the earlier time period 1982-1986, the percentage of current or former smokers among female lung cancer cases in Rockland and the state was about equal.

A number of occupational exposures have been associated with an increased risk of developing lung cancer, particularly exposures related to arsenic, asbestos, chloromethyl ethers, chromium, vinyl chloride and ionizing radiation. Although 36% of individuals in Rockland diagnosed with lung cancer had a reported occupation as unknown or retired, 10% of the individuals worked in an occupation that is related to an increased incidence of this cancer. This information indicates that occupation likely played a role in at least some of the incidence of lung cancer in Rockland. Further, given the large percentage of individual lung cancer cases in Rockland who were current or former smokers, it appears likely that a combination of smoking and possibly occupation played a role in the incidence of lung cancer in the town.

Although no other cancer type was significantly elevated in the town, cancers of the brain and liver displayed slight increases in incidence above the statewide rates for Rockland overall. Further examination of the incidence of these cancer types showed no predominant pattern in the town by time period or census tract. Although the elevations did occur in the lower census tract, CT 5021, they were based on increases of less than three cases. As previously mentioned, cancer is not a single disease but rather a number of different types of diseases. Different types of cancer have different causes, patterns of incidence and risk factors related to their development. More specific discussion of the patterns of brain and liver cancer and their related risk factors is described below. Additional discussion of risk factor information for the eight other cancer types is included in Appendix B.

In Rockland overall, approximately one additional case of brain cancer occurred versus the expected number during the 13-year period 1982-1994. This case occurred in the later time period 1987-1994 in CT 5021, located in the southern section of Rockland. When brain cancer incidence was examined by gender, no particular pattern emerged in this census tract and the number of cases overall in this area of town was relatively small. A total of five cases occurred among males and four cases occurred among females. Brain cancer incidence in Rockland displayed a typical pattern for this cancer type where most of the individuals diagnosed were adults and the average age at diagnosis was 57 years. One case of childhood cancer occurred in

the town and although childhood cancer is relatively rare, brain cancer is one of the more common cancer types diagnosed among children. In CT 5022, the census tract located adjacent to the SWNAS, all of the individuals diagnosed were greater than age 25.

Although factors related to the development of brain cancer remain largely unknown, studies have suggested exposure to ionizing radiation has the potential to increase the risk of developing this cancer type. Some studies have also suggested certain occupational exposures may increase the risk of developing brain cancer including workers exposed to vinyl chloride (a chemical used in the manufacture of plastics and wood furnishings) as well as workers in the petrochemical industry. The occupational information for individuals in Rockland diagnosed with brain cancer was primarily reported as unknown or retired. Thus, the ability to assess whether occupational exposure(s) may have contributed to brain cancer incidence among some of these individuals was limited. However, review of information for those with a reported occupation revealed no individuals with an occupation in the petrochemical industry or employment where exposure to vinyl chloride seemed likely. Some epidemiologic studies suggest that exposure to pesticides may be related to an increased risk of developing brain cancer. However, this association has not been confirmed.

As previously discussed, the incidence of liver cancer was also slightly greater than expected in the town of Rockland generally during 1982-1994. Among both males and females in Rockland combined, approximately two cases above the expected number occurred. Five of the six individuals diagnosed with liver cancer in Rockland were diagnosed in the later time period 1987-1994. The incidence was fairly evenly distributed among males and females but when evaluated at the census tract level, five of the six cases occurred in the southern census tract, CT 5021. Although the incidence of liver cancer was elevated in this census tract (SIR=224), the incidence rate was based on a relatively small number of cases (i.e., 5 cases observed while slightly more than two expected). The instability of this SIR is demonstrated in the width of the 95% confidence interval (95% CI=72-522).

In the United States, men are more likely to develop liver cancer than women and the

incidence of this cancer type is increasing in general. The distribution of this cancer type was fairly equal in Rockland where three cases were diagnosed among men and women each during 1987-1994. The major factors related to the development of liver cancer are chronic infections with hepatitis, chronic alcohol consumption, and cirrhosis of the liver. Approximately 70 to 90% of individuals diagnosed with liver cancer have cirrhosis of the liver. However, some studies have suggested that occupational exposure to vinyl chloride may increase the incidence of this cancer. This is generally a concern related to historical workplace exposures as workplaces are currently regulated against these types of exposures. The mean age of individuals in Rockland diagnosed with liver cancer was 73 years. The occupational information reviewed for Rockland did not indicate occupations known to be related to vinyl chloride exposure or exposures associated with liver cancer.

B. Discussion of Cancer Incidence and Environmental Concerns

With respect to the review of cancer incidence in Rockland and the possible relationship to areas of environmental concern, the geographic or spatial pattern of individual cancer cases was examined in relation to proximity to these sites. As the SWNAS abuts four towns including Rockland, the true pattern of cancer incidence in relation to possible exposures from this site could not be evaluated as part of this report. However, the general pattern of cancer incidence in Rockland during the period 1982-1994, showed that in CT 5022, the Rockland census tract adjacent to the SWNAS, each of the cancer types occurred at the expected rate. The majority of cancer types (eight of the eleven types reviewed) occurred less often than expected in the town. The three cancer types in which elevations did occur (brain, liver and lung cancer) were in CT 5021, the census tract located in the southern portion of Rockland. With the exception of lung cancer among females, the elevations in this census tract were based on increases of less than three cases.

Although concern has been raised about suspected increased cancer incidence in Rockland neighborhoods nearest to the SWNAS with respect to runway locations and

predominant wind patterns (i.e., neighborhoods in the vicinity of Union Street), the pattern of cancer incidence in these neighborhoods did not demonstrate a concentration of any one cancer type. The predominant wind direction measured at the SWNAS is from the south-southwest towards the general direction of Weymouth and Hingham (NRCC 2000). Further, the cancer types that were observed among residents in these areas were various and did not occur in any pattern spatially or with respect to year of diagnosis.

Cancer of the brain and liver occurred slightly more often than expected in Rockland and CT 5021. Although an increased incidence of these cancer types is associated with occupational exposure to vinyl chloride, the association is based on studies of workers exposed to high levels of this chemical, not studies of residential exposure. Based on review of the available information regarding hazardous waste sites and landfill areas in the town of Rockland, it is unlikely that exposures to vinyl chloride have occurred to the general Rockland population. That is, other than in a workplace setting, vinyl chloride enters the environment predominantly by releases to air. These releases occur mainly as a result of air emissions from manufacturing facilities (e.g., plastics) or as a result of releases from improper disposal at landfills or the breakdown of other manufactured substances such as trichloroethylene (TCE), trichloroethane (TCA), and tetrachloroethylene (PCE). Vinyl chloride is not normally found in suburban or rural ambient air in detectable amounts (ATSDR 1997). Although specific information about potential exposure to vinyl chloride from landfills in Rockland is not known, the geographic pattern of these cancer types in relation to the Beech Street landfill or known locations of hazardous waste sites in CT 5021 did not demonstrate any unusual pattern of cases.

Based on the information reviewed in this evaluation, the pattern of cancer incidence in the town of Rockland does not suggest that environmental exposures played a major role. From the available risk factor information evaluated, it appears that tobacco use and to a lesser extent, occupational exposures probably contributed to cancer incidence in the town. It is important to note however, that this evaluation can not determine the exact cause (environmental or otherwise) of any one individual's cancer diagnosis. Further, as previously mentioned, additional environmental sampling efforts and investigation are currently underway at the

SWNAS and the Suburban Auto site as well as an evaluation of cancer incidence in the towns of Weymouth, Hingham and Abington that are adjacent to the SWNAS. Therefore, a more comprehensive evaluation of the pattern of cancer incidence in relation to these concerns will be possible upon review of this additional information.

VII. CONCLUSIONS

- During the period 1982-1994, the majority of the eleven cancer types evaluated in Rockland occurred approximately as expected or in some cases slightly less often than what would have been expected based on the statewide incidence of these cancers.

- With the exception of lung cancer, there were no statistically significant elevations any of the eleven cancer types in Rockland or its census tracts for three time periods evaluated. The elevation in lung cancer incidence was the result of a statistically significant elevation in CT 5021.01 among females in the later time period, 1987-1994. Further, the rate of current and former female smokers diagnosed with lung cancer in Rockland was nearly 10% higher than that observed for the state as a whole.

- Melanoma among males and females combined during 1987-1994 occurred statistically significantly less often than expected (6 observed versus 13.6 expected; SIR=44). During the earlier time period, melanoma occurred as expected among males and females combined.

- There were no unusual geographic patterns of cancer cases observed in Rockland or its two census tracts that were not attributable to more densely populated areas. Further, no geographic pattern of cancer cases was observed in relation to MDEP 21E sites or other areas of environmental concern to local residents (e.g., French's Stream, landfills).

- While no unusual pattern of cancer in Rockland was observed near the SWNAS, it is not possible at this time to determine whether environmental exposure opportunities at the SWNAS have impacted the four abutting communities. This evaluation is being completed as part of a larger evaluation of cancer incidence that includes all (four towns (Rockland, Abington, Hingham, and Weymouth).

VIII. RECOMMENDATIONS

- The Massachusetts Department of Public Health should evaluate more recent cancer incidence data for Rockland as they become available, with particular attention to lung cancer in CT 5021.01.

- Based on the information regarding the incidence of lung cancer and smoking in Rockland, particularly among females, the findings of this investigation should be provided to local health officials and the Massachusetts Tobacco Control Program for consideration of appropriate follow-up and intervention strategies.

- In a future report, the MDPH will evaluate cancer incidence data for Rockland along with those of Abington, Hingham, and Weymouth for possible geographic patterns related to environmental exposure opportunities at the SWNAS.

- When available, the MDPH will review additional environmental sampling data for the Suburban Auto site in relation to the pattern of cancer incidence and childhood lead prevalence data in Rockland.

IX. REFERENCES

ACS. 1998. Cancer information database: what are the risk factors for cancer of the pancreas? American Cancer Society. http://www.cancer.org/cidSpecific cancers/pancreas/risk.html.

ACS. 1999. Cancer facts and figures: breast cancer. From American Cancer Society. http//www.cancer.org/statistics/eff98/selected cancers.html.

Anderson, D., J. Potter, and T. Mack. 1996. Pancreatic cancer. pp. 725-771. In: Cancer epidemiology and prevention. Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

Armstrong, B.K., and D.R. English. 1996. Cutaneous malignant melanoma. pp. 1282-1312. In: Cancer epidemiology and prevention. Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

ATSDR. 1997. Toxicological Profile for vinyl chloride (update), September. U.S. Department of Health and Human Services (USDHHS), Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR. 1999. Public health assessment for South Weymouth Naval Air Station. Agency for Toxic Substance and Disease Registry, U.S. Department of Health and Human Services, Atlanta, GA.

Black, P.M. 1991. Brain tumors (first of two parts). N. Eng. J. Med. 324(21): 1471-76.

Blot and Fraumeni 1996. Cancers of the lung and pleura. pp. 637-665. In: Cancer epidemiology and prevention. Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

Coler & Colantonio. 1999. Phase I Intial site investigation and Tier Classification Opinion Suburban Auto of Rockland, Inc. 163 Forest Street, Rockland, Massachusetts RTN:4-6043. September 3, 1999. Coler & Colantonio, Inc. Norwell, MA.

Devesa, S.S., W. Blot. 1995. Recent cancer trends in the United States. J. Ntl. Can. Inst. 87(3):5-12.

Higginson, J., C.S. Muir, and N. Munoz. 1992. Human cancer: epidemiology and environmental causes. Cambridge Monographs on Cancer Research, Cambridge University Press, Cambridge, Great Britain.

Isselbacher, et al. (eds.). 1994. Harrison's principles of internal medicine. Thirteenth Edition, McGraw-Hill, Inc, New York, NY.

Kessler, L.G. 1992. The relationship between age and incidence of breast cancer. Cancer Supp. 69(7):1896-1903.

MapInfo, Version 4.1, 1996. Copyright MapInfo Corporation, 1985-1996. Troy, New York.

MCR. 1996. Massachusetts Cancer Registry Abstracting and Coding Manual for Hospitals. Second Edition. Massachusetts Department of Public Health, Bureau of Health Statistics, Research and Evaluation, Boston, MA.

MCR. 1997. Cancer incidence and mortality in Massachusetts 1987-1994: Statewide report. August 1997. Massachusetts Department of Public Health, Bureau of Health Statistics, Research and Evaluation, Massachusetts Cancer Registry. Boston, MA.

MDEP 1997. The Massachusetts Contingency Plan 310 CMR 40.0000. Massachusetts Department of Environmental Protection, Bureau of Hazardous Waste Site Cleanup. Boston, MA. May 1997.

MDEP 1998. Bureau of Waste Site Cleanup Site lists. From http://www.magnet .state.ma.us /dep/bwsc/sitelist. Massachusetts Department of Environmental Protection, Bureau of Waste Site Cleanup. Boston, MA. October 1998.

MDEP 1999a. Massachusetts Department of Environmental Protection Solid Waste Facilities Database. September 1999.

MDEP 1999b. Personal communication with Robert Johnson, Southeast Regional Offices, Massachusetts Department of Environmental Protection. September 1999.

MDEP 1999c. Personal communication with Rob Roy, Southeast Regional Offices, Massachusetts Department of Environmental Protection. September 1999.

NCI. 1996. Cancer Rates and Risks. Fourth Edition. National Cancer Institute, National Institutes of Health, Publication No. 96-691.

NRCC. 2000. Climatological data for the South Weymouth Naval Air Station 1993-1996. Northeast Regional Climate Center, Cornell University. Ithaca, NY.

Preston-Martin, S. 1996. Epidemiology of primary CNS neoplasms. Neurol Clin. 14 (2):273-90.

Preston-Martin, S., and W. Mack. 1996. Neoplasms of the nervous system. pp. 1231-1281. In: Cancer epidemiology and prevention. Second Edition. D. Schottenfeld, and J.F. Fraumeni (eds.). Oxford University Press, New York, NY.

Rothman, K., and J. Boice. 1982. Epidemiological analysis with a programmable calculator. Epidemiology Resources, Inc., Boston, MA.

Scherr, P.A., and N.E. Mueller. 1996. Non-Hodgkin's lymphomas. pp. 920-945. In: Cancer epidemiology and prevention, Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

Schottenfeld, D. and Fraumeni, J.F., Jr. (eds). 1996. Cancer Epidemiology and Prevention. 2nd ed. Oxford University Press, New York, NY.

Silverman, D., A. Morrison, and S. Devesa. 1996. Bladder cancer. pp. 1156-1179. In: Cancer epidemiology and prevention, Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

Stone & Webster Environmental Technology and Services. 1996. Final Phase I environmental baseline survey Naval Air Station South Weymouth, MA, November 18, 1996. Stone & Webster Environmental Technology and Services, Boston, MA.

Tatham, L., et al. 1997. Occupational risk factors for subgroups of non-Hodgkin's lymphoma. Epidemiology. 8(5):551-558.

Thun, J.J., C.A. Lally, J.T. Flannery, E.E. Calle, W.D. Flanders, and C.W. Heath, Jr. 1997. Cigarette smoking and changes in the histopathology of lung cancer. J. Ntl. Can. Inst. 89(21):1580-1586.

U.S. DOC. 1980. Census of Population: General Population Characteristics, Massachusetts. U.S. Department of Commerce, Washington, DC: US Gov't Printing Office.

U.S. DOC, 1990. Census of Population: General Population Characteristics, Massachusetts. U.S. Department of Commerce, Washington, DC: US Gov't Printing Office.

Wigle, D.T., R.M. Semenciew, K. Wilkins, D. Riedel, L. Ritter, H.I. Morrison, and Y. Mao. 1990. Mortality study of Canadian male farm operators: non-Hodgkin's lymphoma mortality

and agricultural practices in Saskatchewan. J. Ntl. Can. Inst. 82(7):575-582.

Zahm, S.H., D.D. Weisenburg, P.A. Babbitt, R.C. Saal, J.B. Vaught, K.P. Cantor, and A. Blair. 1990. A case-control study of non-Hodgkin's lymphoma and the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) in eastern Nebraska. Epidemiology. Vol. 1, No. 5, pp. 349-356.

Zahm, S.H., D.D. Weisenburg, P.A. Babbitt, R.C. Saal, J.B. Vaught, and A. Blair. 1993. The role of agricultural pesticide use in the development of non-Hodgkin's lymphoma in women. Arch. Environ. Health. 48(5):353-58.

XI. FIGURES

XII. TABLES

XIII. APPENDIX A: PHASE I REVIEW OF CANCER INCIDENCE IN ABINGTON, ROCKLAND, HINGHAM AND WEYMOUTH 1982-1992

XIV. APPENDIX B: DISCUSSION OF CANCER RISK FACTORS

A. Bladder Cancer

Bladder cancer accounts for six percent of all cancers diagnosed in the United States among men and two percent among women. White males have the highest prevalence of bladder cancer among all racial groups. A male to female ratio of approximately four to one has been observed among whites, while a slightly lower male to female ratio of three to one has been observed among most other racial groups. The occurrence of bladder cancer rises with increasing age. Approximately two-thirds of all bladder cancer cases occur in persons age 65 and over (Silverman et al. 1996). In Rockland, 24 of the 28 bladder cancer cases during 1982-1994 occurred among males, with 61% of all bladder cancer cases in the town over age 65 at diagnosis.

In the United States, it has been estimated that 48% of bladder cancer diagnosed among men and 32% among women is attributed to cigarette smoking (Hartge et al. 1987). Smokers experience approximately two to three times the risk of developing bladder cancer over persons who have never smoked. The risk of developing bladder cancer is increased with greater smoking intensity and duration. Sixty-four percent of bladder cancer cases in Rockland were reported as current or former smokers at the time of diagnosis, and an additional 18% had an unknown smoking status. Among those with a known smoking status, 18 of 23 bladder cancer cases (78%) reported being current or former smokers. This percentage for Rockland is higher than in the state of Massachusetts (66%). Smoking cessation has been found to reduce the risk of developing bladder cancer by 30 to 60%. This reduction in risk has been thought by some to occur in the first two to four years, whereas other research supports a reduction in cancer risk many years after smoking cessation (Silverman et al. 1996).

Studies have revealed a variety of occupations as being associated with the development of bladder cancer (Silverman et al. 1996). Working in occupations like dye-stuffs, rubber, and aromatic amine manufacturing is associated with a high risk of developing bladder cancer due to exposure to the chemicals 2-napthylamine, benzidine or both. Working in other occupations

such as leather tanning, painting, motor vehicle operations, and the aluminum industry have been associated to a lesser extent with the development of bladder cancer. However, specific occupational exposures have not been confirmed and the study findings are not always consistent.

Also, dietary factors such as consumption of fired foods as well as foods high in fat and cholesterol have been found to have an association with increased bladder cancer risk (Silverman et al. 1996). While other factors like some anti-cancer drugs (cyclophosphamide and chloraphazine), bladder infection, and infection with Shistosome Haematobium (a parasite found in Africa) are thought to be associated with the development of bladder cancer, not all epidemiological studies have produced convincing findings (Silverman et al. 1996).

B. Bone Cancer

While every year 60 new primary bone cancer cases are diagnosed in Massachusetts, the occurrence of these neoplasms is rare (Suit et al. 1996). Less than one percent of all newly diagnosed cancer cases are cancers of the bone (Miller et al. 1996).

Bone cancer is subdivided into three main types, osteosarcoma, chondrosarcoma, and Ewing's sarcoma (Miller et al. 1996). While osteosarcoma and Ewing's sarcoma are both frequently observed in adolescence, osteosarcoma also occurs in individuals over the age of 40. This rise in incidence after middle age is usually associated with the presence of Paget's disease. Chondrosarcoma incidence increases with age; about 50% of cases occur later in life (i.e., after age 40) with less than 4% occurring in adolescence (Miller et al. 1996; Malawer et al. 1997). Among the major bone cancer types, males experience a higher incidence of bone cancer than females among both white and black individuals. In the United States and Africa, the occurrence of Ewing's sarcoma among black individuals is almost non-existent. While the incidence of Ewing's sarcoma among the Asian population also is very low, it is higher than that observed among blacks (Miller et al. 1996).

Very little is known about factors associated with the development of bone cancer. Ionizing radiation has been identified as one of the only environmental factors known to play a role in the development of certain types of this cancer (i.e., osteosarcoma and chondrosarcoma). The exposure to high-dose radiation for short periods of time (usually during the treatment of cancer) and ongoing exposure to internally deposited radionuclides (used to treat bone disease or for diagnostic radiography) have been associated with an excess risk of bone cancer. However, although the use of high dose radiation has been identified as a risk factor, it is estimated to account for less than one percent of these cases (Miller et al. 1996).

Limited information is available regarding bone cancer and occupational risk factors (Hoppin et al. 1999). In the past, occupational exposure to radium was found to increase the risk of developing bone cancer. Prior to 1930, a study revealed women employed as radium dial painters in the United States were found to be ingesting radium orally by licking their paintbrushes to produce finer tips for finer lines. (Miller et al. 1996). Some studies have revealed that certain woodworking occupations (e.g., carpenters, furniture workers) are associated with increased bone cancer mortality, but findings are inconsistent (Hoppin et al. 1999).

Metal implants (i.e., hip replacement) are also thought to play a causal role in the development of bone cancer. This is thought to be due to the use of metals such as chromium (a known human carcinogen) and cadmium (a suspected human carcinogen), or the use of bone cement. While an association between bone cancer and metal implants is suggested, no definitive links in humans have been identified (Miller et al. 1996).

Some pre-existing conditions have been identified as being associated with the development of certain bone cancers. Chondrosarcoma has been identified among individuals with such conditions as Ollier's syndrome or Maffucci's syndrome in combination with Ollier's syndrome. Paget's disease is also associated with an increased risk of developing osteosarcoma after middle age. While this association has been made, less than one percent of individuals with

Paget's disease are diagnosed with a bone cancer. An elevated risk of developing bone cancer has also been associated with a family history of Li-Fraumeni syndrome (Miller et al. 1996).

C. Brain Cancer

Brain tumors can be either malignant (cancerous) or benign (noncancerous). Primary brain tumors (i.e., brain cancer) mainly comprise two types: gliomas and malignant meningiomas. Gliomas are a general classification of malignant tumors that include a variety of types. Meningiomas are tumors that 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, these tumors account for about 50% of all reported primary brain and spinal cord tumors. About 85% of meningiomas are benign. In addition, the brain is a site where both primary and secondary malignant tumors (i.e., brain cancer) can arise. Secondary brain tumors generally originate elsewhere in the body and then metastasize, or spread, to the brain (ACS 1999a).

Brain tumor incidence first declines after a peak in childhood (under ten years of age), increases from age 25 to 75, and then levels off after age 75 (Preston-Martin and Mack 1996). As previously mentioned, the average age at diagnosis for Rockland as a whole during 1982-1994 was 57 years. In CT 5021, 8 of 9 cases were greater than age 25 at diagnosis, while one childhood case was reported. In CT 5022, all cases were over 25 years of age. Males are almost twice as likely than females to develop a brain tumor of any type (i.e., cell type) (Bondy and Wrensch 1996). In general, the highest rates of brain and nervous system cancer tend to occur in whites. The incidence of gliomas is less among black men and women, but for meningiomas, the reverse is true, where higher rates have been observed among blacks than whites (Preston-Martin 1996).

Astrocytomas and medulloblastoma, which are types of gliomas, are the most common malignant brain tumors in children (Black 1991). In adults, the most frequent types of tumors are astrocytic tumors (mainly astrocytomas and glioblastoma multiforme), meningiomas, and

benign tumors of the ear canal and pituitary gland. Among 13 Rockland adults diagnosed with brain cancer during 1982-1994, 12 individuals had a type of glioma, with 8 of these having either astrocytoma or glioblastoma multiforme. Among adults, astrocytic tumors tend to bear a graver prognosis than those seen in children (NCI 1996).

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

The most established risk factor for brain tumors 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 (Preston-Martin 1996). Decades ago, before the risks of radiation were realized, children with health conditions such as ringworm of the scalp were treated with low-dose radiation therapy which greatly increased their risk of developing brain tumors later in life (ACS 1999a). Meningiomas are the most common type of tumors that occur from this type of exposure (Preston-Martin and Mack 1996). Results from a number of studies have shown that between 7 and 17% of patients with glioblastoma and glioma had received previous therapeutic radiation treatment for cancer (Bondy and Wrensch 1996).

Among adults, the risk of developing meningiomas or nerve sheath tumors 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 child brain tumors (Preston-Martin and Mack 1996).

Head trauma is most strongly associated with the development of meningiomas compared with any other type 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

(Preston-Martin and Mack 1996). Gliomas are the most common type of childhood brain tumors and have been positively associated with trauma at birth (i.e., Cesarean section, prolonged labor) (Black 1991).

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 is a common inherited cause of brain or spinal cord tumors and occurs in about 1 out of 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 1999a). Of those afflicted with the disease, about 5 to 10% will develop a central nervous system tumor (Preston-Martin and Mack 1996).

The association between the development of brain cancer and N-nitroso compounds (NOCs), among the most potent of carcinogens, has been heavily researched and studied. NOCs have been found in tobacco smoke, cosmetics, automobile interiors, and cured meats. Because NOCs have been shown to cause brain cancer in animals, it has been hypothesized that NOCs may cause cancer in humans as well (Preston-Martin and Mack 1996). However, epidemiological studies have lent limited support to this hypothesis. Because most people have continuous, low-level exposure to NOCs throughout their lives, further study is needed to determine if an association exists between this exposure and the development of brain tumors (Preston-Martin 1996).

In recent years, there has been increasing public concern and scientific interest regarding the dangers of electromagnetic fields and brain cancer. To date, epidemiological studies have not confirmed an association between brain tumors and exposure to 50-60 Hz EMFs (extremely low frequency) from power lines, appliances, and industrial equipment. Recently, the National Institute of Environmental Health sciences concluded after a review of scientific studies conducted to date that "no association was seen in any of these studies" between cancers of the central nervous system and EMF exposure (NIEHS 1998).

Various studies on worker exposure to vinyl chloride and chemicals in the petrochemical industry have had conflicting results as to the association between these chemicals and the development of brain tumors. Studies investigating the possible association between occupational exposure of parents (in particular, paper or pulp-mill, aircraft, rubber, and electric workers) and the onset of brain tumors in their children have also provided inconsistent results (Preston-Martin and Mack 1996).

Other possible risk factors investigated for brain cancer have included alcohol consumption, use of barbiturates, smoking, exposure to second-hand smoke, pesticides, and infectious disease (i.e., tuberculosis and chicken pox). To date, studies on these risk factors have yielded inconclusive results.

D. Breast Cancer

Breast cancer is the most common type of cancer among women in both the United States and in Massachusetts. Female breast cancer incidence in Massachusetts is comparable to the incidence in females nation-wide. Although during the 1980s breast cancer in the U.S. increased about 4% per year, the incidence of this disease in recent years has leveled off to about 110 cases per 100,000 (ACS 1998a). Likewise, in Massachusetts the incidence of breast cancer rose approximately 28% from 1982 to 1986, however, from 1987 to 1994 breast cancer rates remained fairly constant (MCR 1997).

In 1999, it has been estimated that in the U.S. 175,000 women will be diagnosed with breast cancer (ACS 1999b). Worldwide, female breast cancer incidence has increased, mainly among the older age groups whose proportion in the population continues to increase (van Dijck et al. 1997). Well-recognized risk factors for breast cancer include increasing age, early age at menarche, late age at menopause, and late age at first pregnancy or birth, length of menstrual cycle, family history, and hormone levels. Other risk factors that have been investigated are diet,

body weight, lack of physical activity, alcohol, various environmental exposures, cigarette smoking, and benign breast disease.

Women of low socioeconomic status may be at an increased risk for breast cancer because they tend to have less access to medical care and lower screening participation rates than do women in higher social classes (Segnan 1997). However, women in higher social classes may also have an increased risk because of different reproductive patterns (i.e., parity, age at first birth, and age at menarche).

A family history of breast cancer is an indicator of increased breast cancer risk. A woman whose mother, sister, or daughter had breast cancer has two to three times the risk of developing breast cancer than a woman without a family history (Kelsey 1993). Epidemiological studies have found that females with a first-degree relative (mother, sister, or daughter) with premenopausal breast cancer have three-fold the risk of developing breast cancer. Meanwhile no increase risk has been found for females with a first degree relative with a postmenopausal breast cancer diagnosis (Broeders and Verbeek 1997). If women have a first degree relative with bilateral breast cancer (cancer in both breasts), then the risk increases five-fold (Broeders and Verbeek 1997). Moreover, if women have a mother, sister or daughter with bilateral and premenopausal breast cancer, the risk of developing breast cancer 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).

It is estimated that between 5 to 10% of breast cancers are inherited. In 1994 and 1995, two genes BRCA1 and BRCA2 were discovered, and this genetic predisposition accounts for approximately 40 to 50% of all the hereditary breast cancers in the general population (ACS 1998a). It has also been estimated that 50 to 60% of the persons who inherit BRCA1 or BRCA2 mutations will develop breast cancer by the age of 70 (ACS 1998a).

A woman's breast cancer risk is in great part determined by the cumulative exposure of the breast (tissue) to estrogen and progesterone (hormones present in a woman's body) (Henderson et al. 1996). Since during pregnancy the levels of estrogens are higher than during

any other period of a woman's life, it is suspected that early exposures to estrogen during fetal development might play a role in the risk of developing breast cancer late in life. Researchers have found that women whose mothers had toxemia during pregnancy (a condition associated with low levels of estrogen) had a statistically significant reduced risk of developing breast cancer. Some studies have shown that neonatal jaundice, severe prematurity, and being a fraternal twin are factors associated with increased levels of estrogen, and these factors may also increase the risk of developing breast cancer later in life (Ekbom et al. 1997). Evidence from studies that have included large numbers of long-term users of estrogen replacement therapy indicates a modest (less than two-fold) elevation in risk associated with 10-15 years or more of use (Kelsey 1993). However, investigations of the risks associated with the use of oral contraceptives have yielded inconsistent results.

High levels of estrogen at certain ages during specific reproductive events in a woman's life may also increase the risk of breast cancer. An early age at menarche (first menstrual period) results in a longer duration of exposure to estrogen during puberty and can increase the risk of breast cancer when compared to women who began menstruation at 14 years of age and older (MacMahon et al. 1992; Harris et al. 1992). In addition, women who experience later menopause (after the age of 50) also have a slightly increased risk (ACS 1998a). Furthermore, the combined effect of an early menarche and a late menopause causes a longer exposure to estrogen and progesterone, which have been associated with an increase in risk (Lipworth 1995). In fact, women that have been actively menstruating for 40 or more years are thought to have twice the risk of developing breast cancer than those with less than 30 years of menstrual activity (Henderson et al. 1996). Also, the risk of breast cancer has been shown to increase linearly with age at first live birth. Women who gave birth for the first time after age 30 have shown a four-to-five fold excess in risk compared to those gave birth for the first time prior to 18 years of age (Brinton et al. 1983).

Several benign breast diseases have also been associated with the onset of breast cancer. The most common type of benign breast disease is chronic cystic or fibrocystic disease. Women with cystic breast disease have been found to have a two-to-three fold increased risk of

developing breast cancer (Henderson et al. 1996). In addition, certain types of benign breast disease have been associated with an increase in a woman's risk of breast cancer.

Diet, particularly fat intake, is another factor suggested to increase a woman's risk for breast cancer. Currently, it is thought that the type of fat in a woman's diet might be more important than the total fat intake (ACS 1998a; Wynder et al. 1997). Monounsaturated fats (e.g., olive oil and canola oil) are related to a lower risk and polyunsaturated fats (e.g., corn oil, tub margarine) and saturated fats in meat are linked to an elevated risk (ACS 1998a). With respect to a woman's individual weight, the risk of breast cancer becomes less clear (ACS 1998a). Many studies have found that a heavy body weight elevates the risk for breast cancer in postmenopausal women (Kelsey 1993). In postmenopausal women who are obese, the principal source of estrogen in the body is fat tissue (McTiernan 1997). Regular exercise is thought to reduce the exposure to cyclic estrogens and progesterones, hormones believed to play an important role in the carcinogenesis of the breast (Thune et al. 1997).

Alcohol consumption has been shown to be directly related to an increased risk of breast cancer (Swanson et al. 1997; ACS 1998a). However, this relationship still needs further evaluation. Women who consumed one alcoholic beverage daily in comparison to non-drinkers had a very small increase in risk, but those who had 2 to 5 drinks daily had about 1.5 times the risk of women who drink no alcohol (ACS 1998a). Furthermore, it is known that alcohol consumption affects the endocrine system, but the effects of alcohol on estrogen metabolism have not been fully investigated (Swanson et al. 1997).

To date, no specific environmental factor has been positively associated with the development of breast cancer. The role of cigarette smoking, among other environmental factors, in the development of breast cancer is not clear. Some studies suggest a relationship between passive smoking and an increase risk of breast cancer; however, the lack of evidence from studies that include direct smoke exposure make it difficult to confirm this association (Laden and Hunter 1998).

Individual exposure to high dose radiation can pose a risk of developing breast cancer. High-dose exposures to ionizing radiation have been shown to cause cancer from studies of survivors of the atomic bomb blasts in Hiroshima and Nagasaki as well as patients that have been subjected to radiotherapy in treatments (for tuberculosis, post-partum mastitis, and cervical cancer) (Laden and Hunter 1998). However, it has not been shown that radiation exposures experienced by the general public (people living in areas of known elevated radiation levels due to industrial accidents or nuclear activities) are related to an increase in breast cancer risk (Laden and Hunter 1998). Furthermore, investigations about electromagnetic field exposures in relation to breast cancer have been inconclusive.

Evaluations of certain occupations have not clearly identified any risk factors pertaining to breast cancer (Goldberg and Labreche 1996). Currently, no evidence has been found showing an excess of risk of developing breast cancer among nuclear workers who are exposed to ionizing radiation (Goldberg and Labreche 1996). Due to insufficient evidence, the risk of a chemist or other women exposed to extremely low frequency electromagnetic fields can not be estimated (Goldberg and Labreche 1996). Further, studies on the relationship between solvent exposure and occupations, such as dry cleaning workers and shoe manufacturers, have not provided enough supportive evidence to determine an association with breast cancer (Goldberg and Labreche 1996). A more recent study of occupational exposures found that women exposed to organic solvents had an elevated rate of breast cancer that could not be explained by known reproductive or social factors (Hansen 1999). However, further work is needed to determine which organic solvents are responsible for this association.

The geographic variations observed in the incidence and mortality of breast cancer in the U.S. and around the world have prompted researchers to investigate the role of the environment in the observed increase of breast cancer. Variations have been found in breast cancer incidence rates observed in the Northeastern, Midwest and Southern regions of the U.S., and a slightly higher breast cancer rate has been observed for postmenopausal women in California (Laden et al. 1997). Also, a linear relationship between breast cancer incidence and increasing population density had been documented (Nasca et al. 1992). In the Northeastern region of the U.S., females older than 50 have shown higher mortality rates (20 to 50%) compared to females that

live in the Southern U.S. (Laden et al. 1997). However, there is not enough evidence linking environmental factors to the regional variation of breast cancer in the U.S. (Laden et al. 1997).

Exposure to chemical substances has not been associated with certainty to an increased risk of breast cancer. However, exposure to some chemical pollutants such as chlorinated hydrocarbon pesticides, DDT, DDE, and PCBs are suspected of increasing breast cancer risk. It has been hypothesized that because these compound are weakly estrogenic (affect estrogen production and metabolism) they can increase the risk of breast cancer (Davis et al. 1993; Laden and Hunter 1998). Studies on breast cancer and exposure to certain pesticides have provided inconclusive evidence but still may represent a potential risk factor for breast cancer.

E. Hodgkin's Disease

Hodgkin's disease is a form of cancer that involves the lymphatic system and can be classified in four histologic (or cellular) types. These four types of Hodgkin's disease occur with different frequencies according to age at diagnosis and the aggressiveness of the histologic type (Mueller 1996). However, due to substantial improvement in effective therapy for this disease, mortality rates have decreased approximately 55% over the last ten decades. This decrease in mortality is also due to a better early stage prognosis (Mueller 1996).

Epidemiologic studies have shown that Hodgkin's disease is more common among men than women and more common among whites than blacks. Although the disease is relatively rare among children, two peaks in the age distribution have been observed for this cancer type. The first peak occurs in young adults usually between the ages of 15 to 39 and the second peak occurs in older adults 55 years and above.

The bimodal age distribution of this disease suggests that two distinct etiologies (or causes) for Hodgkin's disease may be involved for each group. Specifically, researchers have proposed that among young adults, Hodgkin's disease is caused by a biological agent of low infectivity. Among individuals of older ages, the cause is believed to be similar to those of other lymphomas (Mueller 1996). Furthermore, the clinical and cellular features of Hodgkin's disease

suggest a chronic infectious process is related to the disease. The two viruses that have been linked most specifically to this disease are the Epstein-Barr virus (EBV) and the more recently discovered human herpesvirus-6 (HHV-6) (NCI 1996, Mueller 1996). EBV is a common herpesvirus. Several reports of Hodgkin's disease have been documented in individuals with primary EBV infection. In addition, several studies have also shown that young adults who have developed infectious mononucleosis have a significantly higher risk of developing Hodgkin's disease (NCI 1996; Mueller 1996).

Hodgkin's disease trends reveal that the disease has become increasingly associated with populations both of middle to higher socioeconomic status and small family size. Occupational exposures to workers in the chemical industry and woodworkers have also been suggested in several epidemiologic studies to be associated with the development of Hodgkin's disease. However, specific chemical exposures related to the development of this disease have not been identified (NCI 1996).

F. Leukemia

Leukemia literally means "white blood cell;" a reference to the excessive numbers of white blood cells or leukocytes in the peripheral blood of individuals diagnosed with leukemia. The serious symptoms of the disease, however, are caused by lack of normally functioning cells or platelets. This deficiency is brought about through the proliferation of cells that resemble a stage in normal blood cell development but are incapable of performing the functions of mature blood cells (Miller et al. 1990; Clarkson 1980). These functions include fighting off foreign invaders to the body by attacking them or releasing harmful substances.

Leukemia is a group of malignancies of the white blood cells, classified as separate histologic or cell types. Epidemiologic studies have shown that each histologic type of leukemia is an individual disease with specific characteristics, patterns of survival, and etiologic factors. There are four major subtypes into which most histologic types of leukemia can be categorized: ALL, CLL, CML, and AML. Epidemiologic research over the past twenty-five years has

revealed patterns of incidence and risk factors that vary for each subtype. While there are known and suspected risk factors for each subtype, leukemia cases are relatively rare and despite a large body of research, risk factors that have been identified for different leukemia subtypes only account for a small percentage of cases (Linet 1985).

ALL or acute lymphocytic leukemia occurs predominantly in children. This is the subtype of leukemia diagnosed in the one Rockland case reported less than 19 years of age. Incidence rates drop off among middle-aged people, and increase again among older individuals (ACS 1995). The known risk factors for ALL are ionizing radiation and benzene exposure. Suspected risk factors are genetic, viral, environmental, and occupational. Studies indicate that acute leukemias (ALL and AML) occur at an increased rate in a variety of congenital disorders including Down syndrome and Klinefelter's syndrome (Schneiberg and Golde 1994). Other research has supported viral factors. However, a specific viral agent has not been identified. An increased risk of childhood ALL has been associated with exposure to several chemicals and possible paternal occupational exposure to hydrocarbons (Linet 1985).

CLL is chiefly an adult disease; 90% of cases occur in people over 50 years of old and this was the pattern observed for Rockland. CLL is the most common type of leukemia in the United States and occurs more often in males than females (Miller et al. 1990). Adult T cell leukemia is a type of CLL caused by a virus, Human T-Cell Leukemia/Lymphoma Virus-1 (HTLV-1). The only known risk factor for other types of CLL is exposure to benzene. No association has been found between CLL and exposure to ionizing radiation (Linet 1985; Scheinberg and Golde 1994). Studies showing strong patterns of incidence in families suggest a genetic etiology. An association between CLL and autoimmune diseases suggests that immunologic factors may play an important role. Several viruses linked with leukemia in animals have been shown to cross species barriers; research suggests that human proximity to sick farm animals and pets may increase the risk of CLL. Rubber workers and particularly tire builders, have a higher incidence of CLL. Exposure to some chemical wastes in the environment may also be a risk factor (Linet 1985; Scheinberg and Gold 1994; Linet and Cartwright 1996).

AML may occur in children up to the age of 19, with incidence increasing rapidly beyond age 20. The majority of Rockland cases were between 20 and 60 years of age. The known risk factors for AML are similar to those for ALL; exposure to ionizing radiation and benzene (Linet 1985). Recent studies suggest that viral and genetic factors play a less important role in the development of AML than in ALL and CLL. Suspected risk factors for AML include occupational and environmental exposures and certain drug therapies, such as chloramphenicol and phenylbutazone (Linet 1985). Suspected chemical exposures include petroleum products and organic solvents. AML as a secondary malignancy is increasing among people who have previously had non-Hodgkin's lymphoma, multiple myeloma, breast cancer, ovarian cancer, and lung cancer. A high risk of secondary AML in people who have had Hodgkin's lymphoma has also been documented. However, it is uncertain whether secondary AML is a result of chemotherapy or radiation treatment for a previous cancer, or whether secondary AML occurs as part of the natural history of the previous cancer, as a result of improved cancer therapy may have lengthened survival time (Linet 1985). Cigarette smoking has also been identified as a possible risk factor in the development of AML (Siegel 1993).

Of all the leukemias, CML is among the least understood. CML is an acquired genetic disorder, characterized by the presence of the Philadelphia chromosome. CML can occur at any age but is most often observed in people between 30-50 years of age. The only known risk factor is exposure to ionizing radiation, based on studies of atomic bomb survivors (Golde and Guliti 1994). Occupational exposure to benzene has also been associated with the development of CML, however a causal relationship has not been established. Exposure to other chemical agents in the work place or environment is suspected in this disease (Linet 1985). Cigarette smoking is also suspected in the development of CML, however, the association is weak at best (Siegel 1993). Current research has suggested that heredity and immunologic factors do not appear to be important in the development of CML (Linet 1985).

G. Liver Cancer

Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver (El-Serag et al. 1999). HCC occurs most frequently in Asia, Africa and the Mediterranean basin (Akriviadis et al. 1998). Liver cancer rates can vary substantially by country and although more than 80% of liver cancer cases occur in the developing world, incidence is rapidly increasing in the United States (Stuver 1998; Ince et al. 1999. In the United States, men are more likely than women to develop HCC and blacks are affected twice as often as whites (El-Serag et al. 1999). Although the risk of developing HCC increases with increasing age, the disease can occur in persons of any age.

Chronic infection with the hepatitis B virus (HBV) and hepatitis C virus (HCV) are major risk factors for developing HCC (Fattovich 1998). In fact, scientists estimate that 10 to 20% of people infected with HBV will develop cancer of the liver. HBV is very common in Africa, China, and the Middle East. It has been estimated that approximately 4 million persons in the United States have chronic HCV infection (Ince et al. 1999). Risk factors of the transmission of HBV and HCV include intravenous drug use, sharing of needles, unsafe sexual practices and transfusion of and contact with unscreened blood and blood products (El-Serag et al. 1999). In addition, mothers who are infected with these viruses can pass them on to their children.

Also, numerous epidemiological studies have identified chronic alcohol consumption as a significant risk factor associated with liver cancer (Seitz et al. 1998). Although alcohol is not a carcinogen itself, laboratory studies have shown that it may act as a cocarcinogen in the development of HCC (El-Serag et al. 1999). Some studies have suggested that alcohol and chronic HCV infection may act in concert with one another to accelerate liver disease and damage. One study conducted in Italy showed that people with alcoholism and HCV infection often developed liver disease, which was followed by cirrhosis and the development of HCC (Bellantani et al. 1994).

Cirrhosis is perhaps the most important risk factor related to the development of HCC. Cirrhosis is a progressive disease, which causes inflammation and scar tissue to form on the liver. Scientists have estimated that 70 to 90% of individuals diagnosed with HCC also have cirrhosis (Johnson 1996). Other risk factors associated with the development of HCC such as alcoholism and HBV and HCV infection also cause cirrhosis.

Additional risk factors related to the development of liver cancer include long-term exposure to aflatoxins, vinyl chloride, and thorium dioxide. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be exposed to aflatoxins if they consume peanuts and meal that have been stored under hot, humid conditions (Bellantani et al. 1994). Vinyl chloride has been primarily used in the United States to make polyvinyl chloride (PVC), a substance used in a variety of plastic consumer products and home furnishings (ATSDR 1997). Vinyl chloride is a known human carcinogen and occupational exposure to this chemical has been associated with the development of liver cancer.

In addition, radioactive thorium dioxide, widely used in clinical medicine as a diagnostic agent in Europe and Japan until 1950, is a known factor related to liver cancer. Research has shown that fifteen years following exposure to this substance, the development of cirrhosis and HCC occurred in individuals who were exposed (Bellantani et al. 1994). The impact of both thorium dioxide and vinyl chloride was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now regulated (ACS 1999c).

H. Lung Cancer

Several histologic or cell types of lung cancer have been observed. Among the most common types are squamous cell carcinoma, adenocarcinoma, small (oat) cell carcinoma, and large cell carcinoma. These histologic types 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 (Thun et al. 1997).

The incidence of lung cancer increases sharply with age peaking at about age 60 or 70. Only 2% of lung cancers occur before the age of 40. Lung cancer incidence is greater among men than women and has greater incidence among blacks than whites. The highest rates of lung cancer have been reported among urban black men (Blot and Fraumeni 1996). Trends in lung cancer incidence have revealed that the disease has become increasingly associated with populations of lower socioeconomic status, since these individuals may smoke more (Blot and Fraumeni 1996).

Smoking is the biggest risk factor associated with the development of lung cancer. In 1985, an estimated 90% of lung cancer cases among men and 79% among women were attributed to cigarette smoking (Blot and Fraumeni 1996). An increase in cigarette smoking among women has produced lung cancer incidence rates that more closely resemble those which are experienced by males. Female lung and bronchus cancer incidence in Massachusetts increased by nearly 3% from 1990 to 1996 (MDPH 1999). The risk of developing lung cancer depends on the intensity of one's smoking habits (i.e., duration of habit, amount smoked, tar yield of cigarette, and filter type). The risk declines after smoking cessation however, investigators estimate it can take ten to twenty years of not smoking for long-term, heavy smokers to reduce their risk of developing lung cancer. Studies suggest that the risk for developing lung cancer will always be about twice as high for ex-smokers compared to non-smokers (Blot and Fraumeni 1996).

Occupational exposure has been identified as playing a role in the development of lung cancer. Occupations such as underground miners, chemical workers, paper and pulp workers, carpenters, butchers and meat packers, and shipyard workers are some of the occupations associated with increased risk of developing lung cancer. This is a result of exposure to chemical compounds such as arsenic, asbestos, chloromethyl ethers, chromium, vinyl chloride, and ionizing radiation that may be found in such workplaces. The occupational exposure to such compounds in conjunction with cigarette smoking dramatically increases the risk of developing lung cancer (Blot and Fraumeni 1996).

Exposure to radon (a naturally occurring gas produced by the breakdown of radium and uranium) has been associated with increased risk of developing lung cancer in underground miners (Blot and Fraumeni 1996). While exposure to radon gas by underground miners is recognized to cause lung cancer, the risk associated with low-level indoor radon exposure has only recently been assessed. A recent report by the National Research Council concluded that radon exposures in homes, especially in combination with smoking, contributes to increased lung cancer incidence (NRC 1998).

I. Melanoma

The incidence of malignant melanoma increases with age and rises steeply until the age of 50. Melanoma occurs slightly more often in females than in males (Armstrong and English 1996). The incidence of malignant melanoma is higher among whites and lighter skinned individuals than dark-skinned individuals such as Africans, Asians, and Polynesians. The primary risk factor for melanoma is excessive exposure to ultraviolet radiation (i.e., the sun) for individual skin type. Several recent studies have found significantly increased risks of melanoma following repeated severe (i.e., blistering) sunburns, particularly during childhood and teenage years (Holly et al. 1995; NCI 1996). Freckles, an indicator of sun sensitivity and sun damage, are also associated with increased risk (Holly et al. 1995; NCI 1996). Factors associated with increased risk of melanoma include: a history of sunburns, fair skin, number of moles, presence of dysplastic or other atypical moles, previous melanoma, family history of melanoma, and immunosuppression (Holly et al. 1995; NCI 1996).

J. Non-Hodgkin's Lymphoma (NHL)

Lymphomas are cancers involving the cells of the lymphatic system. The majority of lymphomas involve the lymph nodes and spleen but the disease may also affect other areas within the body (Skarin et al. 1996). Non-Hodgkin's lymphoma (NHL) is a classification of all lymphomas with the exclusion of Hodgkin's disease. Thus, NHL is a mixed group of diseases that is characterized by the malignant increase in specific cells of the immune system (B or T

lymphocytes). The various types of NHL are thought to represent different diseases with different causes (Scherr and Mueller 1996). Epidemiologic studies have established that the primary factors related to developing NHL are infectious in nature. Although some studies have suggested that environmental (i.e., chemical) exposures may be related to the development of NHL, no definitive association has been established.

NHL can occur at all ages. However, the incidence of this disease generally increases with age. Among all NHL types there is a greater male to female ratio and white to black ratio (NCI 1996). In Massachusetts, the incidence of NHL among males increased by 52% from 1982 to 1994. Among Massachusetts women, the incidence of NHL increased by 21% between 1982 and 1994 (MCR 1997).

Nationally, the increase in NHL incidence has been attributed to changes in case classification, better diagnostic techniques, greater exposure to causative agents, and the increasing incidence of AIDS-related lymphomas (Devesa et al. 1992; Scherr and Mueller 1996). Although the primary factors related to the development of NHL include conditions that suppress the immune system, viral infections, and certain occupational exposures, these factors are thought to account for only a portion of the increase observed in this cancer type (Scherr and Mueller 1996).

NHL is more common among people who have abnormal or compromised immune systems; such as those with inherited diseases that suppress the immune system, organ transplant recipients, and individuals with autoimmune disorders (Scherr and Mueller 1996). NHL has also been reported to occur more frequently among individuals with conditions that require medical treatment resulting in suppression of the immune system, such as cancer chemotherapy. Current evidence suggests that the development of NHL is related to suppression of the individual's immune system as a result of treatment rather than the treatment itself (Scherr and Mueller 1996).

Several viruses have been shown to play a role in the development of NHL. Among organ transplant recipients, suppression of the immune system required for acceptance of the transplant leads to a loss of control or the reactivation of viruses that have been dormant in the body (e.g., Epstein-Barr Virus [EBV] and herpesvirus infections). In addition, cancer-causing viruses are known to cause lymphoma in various animals. Therefore, it has been proposed that these types of viruses may also be associated with the development of NHL among humans without compromised immune systems (Scherr and Mueller 1996). The infection with the human HTLV-1 virus is known to cause T-cell lymphoma among adults. However, although this type of virus is known to cause lymphoma, it is a relatively rare infection and most likely contributes only a small amount to the total incidence of NHL (Scherr and Mueller 1996). Since EBV infection is common among the general population and has been shown to play a role in the development of most cases of transplant and AIDS-related NHL, several studies have been conducted to determine the role of EBV infection in the development of NHL in general. These studies have shown that although viruses are causal factors for some subtypes of NHL, their role in the development of NHL as a whole may not be large (Scherr and Mueller 1996).

Some occupations have been associated with an increased risk of developing NHL, specifically occupations related to chemicals or agriculture. Farmers, herbicide and pesticide applicators, and grain workers appear to have the most increased risk (Zahm 1990, 1993; Tatham et al. 1997). Studies conducted among agricultural workers have demonstrated increases in NHL among those using herbicides for more than 20 days per year and individuals who mix or apply herbicides. A greater incidence of NHL appears to be related specifically to exposure to the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) and a class of insecticides called organophosphates (Wigle et al. 1990; Zahm et al. 1990, 1993). Further studies of exposure to these chemicals and NHL incidence have shown that the increased risk is attributed to a specific impurity, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, present in these herbicides. However, reports of accidental industrial exposures to TCDD have not demonstrated an increased risk of NHL (Scherr and Mueller 1996). An elevated risk for NHL development has also been noted among fence workers, orchard workers, and meat workers.

In addition, epidemiological studies of long-term users of permanent hair coloring products have found an increased incidence of NHL (Zahm et al. 1992; Scherr and Mueller 1996). However, a recent population-based study found no association between the use of hair-color products and an increased risk of developing this type of cancer. The researchers further stated that results from this study and previous studies, including experimental animal studies, provide little convincing evidence linking NHL with normal use of hair-color products (Holly et al. 1998).

Some studies have also found a positive association with the incidence of NHL and smoking. A study that evaluated the history of tobacco use and deaths from NHL determined that people who had ever smoked had a two-fold increase of dying from NHL as compared to those who never smoked. Further a four-fold increase was found among the heaviest smokers (Linet et al. 1992). In addition, a more recent study that primarily examined occupation and NHL risk found a significant association with high levels of cigarette smoking and all NHL types (Tathem et al. 1997).

K. Pancreatic Cancer

Pancreatic cancer is the ninth most common cancer in the United States, but is the fifth leading cause of cancer death. 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. From 1920 to 1965 mortality from this disease increased nearly 200% from 2.9 to 8.2 per 100,000 people, but since about 1970, both mortality and incidence rates have been relatively stable. The increases earlier in the century are believed to be due, in part, to improved diagnosis (Anderson et al. 1996).

The majority of pancreatic cancer cases occur between age 65 and 79, with the median age at diagnosis being 71 years (Miller et al. 1992). Men are approximately 30% more likely to

develop pancreatic cancer than are women. Also, African Americans are more likely to develop this cancer than are Asian American or white Americans (ACS 1998b).

The most consistent and only established risk factor for pancreatic cancer is cigarette smoking (Anderson et al. 1996). According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS 1998b). Studies have estimated that the risk of pancreatic cancer is two to six times greater in heavy smokers than in nonsmokers (Anderson et al. 1996).

There is some evidence to suggest that certain dietary factors may be related to the development of pancreatic cancer. Increased risks of pancreatic cancer are usually associated with animal protein and fat consumption. Decreased risks for the disease are usually associated with fruit and vegetable consumption (Anderson et al. 1996). Although older studies suggested that coffee and alcohol consumption may be risk factors for pancreatic cancer, more recent studies have not confirmed this association (ACS 1998b). Diabetes mellitus and chronic pancreatitis have been associated with pancreatic cancer, but the reasons for these associations are largely unknown (ACS 1998b).

Numerous occupations have been investigated for their potential role in the development of pancreatic cancer, but studies have not produced consistent results. Since the 1970s, almost 100 studies have been completed looking at occupations ranging from asbestos workers to office clerks. Working with ionizing radiation, asbestos, fossil fuel products, and various other chemicals (including DDT and its derivatives) has been associated with pancreatic cancer in some studies, still other contrasting studies have found no link between these possible agents and pancreatic cancer (Anderson et al. 1996). Many of the studies mentioned previously have a sample population that is too small to be able to provide consistent meaningful results. This is due to the fact that the incidence of pancreatic cancer is relatively low, especially when classified by job title.

Finally pancreatic cancer seems to run in some families. According to the ACS, an inherited tendency to develop pancreatic cancer may account for approximately 3% of cases (ACS 1998b). Pancreatic cancer has been observed in both familial clustering among siblings as well as in individuals of consecutive generations (Anderson et al. 1996).

References:

ACS. 1995. Cancer Facts and Figures. American Cancer Society, Atlanta Georgia, 1995.

ACS. 1996. Cancer Manual (9th ed.) American Cancer Society, Massachusetts Division. Boston, Massachusetts, 1996.

ACS. 1998a. Cancer facts and figures: breast cancer. American Cancer Society. http//www.cancer.org/bcn/info/brrisk.html.

ACS. 1999a. Brain/central nervous system (CNS) tumors in children. American Cancer Society. http//www3.cancer.org/cancerinfo/main_cont.asp?st=wi&ct=4.

ACS. 1999b. Cancer facts and figures: breast cancer. From American Cancer Society. http//www.cancer.org/statistics/eff98/selected cancers.html.

ACS. 1999c. Liver cancer: prevention and risk factors, liver cancer resource center. American Cancer Society. http://www3.cancer.org/cancerinfo.

Akriviadis, E.A., J.M. Llovett, S.C. Efrimidis, D. Shouval, R. Canelo, B. Ringe, and W.C. Meyers. 1998. Hepzotecellular Carcinoma. Brit. J. Surg. 84:1219-31.

ATSDR. 1997. Toxicological profile for vinyl chloride (update). Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, GA.

Bellantani, S., Tiribelli C., Saccoccio G., Sodde M., Fratti N., DeMartin C., Cristianini G. 1994. Prevalence of chronic liver disease in the general population of northern Italy: the Dionysos study. Hepatology. 20:1442-9.

Black, P.M. 1991. Brain tumors (first of two parts). N. Eng. J. Med. 324(21): 1471-76.

Blot and Fraumeni 1996. Cancers of the lung and pleura. pp. 637-665. In: Cancer epidemiology and prevention. Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

Bondy, M.L., and M.R. Wrensch. 1996. Epidemiology of primary malignant brain tumors. Baillieres Clin. Neurol. 5 (2):251-70.

Brinton, L.A., R. Hoover, and J.F. Fraumeni. 1983. Reproductive factors in the etiology of breast cancer. Br. J. Cancer. 47:757-762.

Broeders, M.J.M., and A.L.M. Verbeek. 1997. Breast cancer epidemiology and risk factors. Quart. J. Nucl. Med. 41:179-188.

Clarkson, B. 1980. The acute leukemias. pp. 798-808. In: Harrison's principles of internal medicine. Ninth Edition. Isselbacher, K.J., et al. (eds.). McGraw-Hill, New York, NY.

Cotran, R.S., V. Kumar, and S.L. Robbins. 1994. Pathologic basis of disease. W.B. Saunders Company, Philadelphia, PA.

Davis, D.L., H.L. Bradlow, M. Wolff, T. Woodruff, D.G. Hoel, and H. Anton-Colver. 1993. Medical hypothesis: Xenoestrogens as preventable causes of breast cancer. Environ. Health Perspect. 101(5):372-377.

Devesa, S.S., W. Blot. 1995. Recent cancer trends in the United States. J. Ntl. Can. Inst. 87(3):5-12.

Devesa, S.S., T. Fears. 1992. Non-Hodgkin's lymphoma time trends: United States and international data. Cancer Res. (Suppl.) 52:5492s-549s.

Ekbom, A., C.C. Hsieh, L. Lipworth. 1997. Intrauterine environment and breast cancer risk in women: a population-based study. J. Ntl. Can. Inst. 89(1):71-76.

El-Serag, H.B., and A.C. Mason. 1999. Rising incidence of hepatocellular carcinoma in the United States. N. Eng. J. Med. 340(10):745-50.

Fattovich, G. 1998. Progression of hepatitis B and C to hepatocellular carcinoma in western countries. Hepatogastroenterology. 45(Suppl. 3): 1206-13.

Goldberg, M.S., and F. Labreche. 1996. Occupational risk factors for female breast cancer: a review. Occ. Environ. Med. 53:145-156.

Golde, D., and S. Guliti. 1994. The myeloproliferative diseases. pp. 1757-1764. In: Harrison's principles of internal medicine. Thirteenth Edition, Vol. 2. Isselbacher et al. (eds.). McGraw-Hill, Inc., New York, NY.

Hansen J. 1999. Breast cancer risk among relatively young women employed in solvent-using industries. Am J Ind Med 36:43-47.

Harris, J.R., M.E. Lippman, U. Veronesi, and W. Willet. 1992. Breast cancer (first of three parts). The New England Journal of Medicine. 327(5):319-328.

Hartge, P., D. Silverman, R. et. al. 1987. Changing cigarette habits and bladder cancer risk: A case control study. JNCI, Vol. 78, No. 6pp. 1119-25.

Henderson, B.E., M.C. Pike, L. Bernstein, and R.K. Ross. 1996. Breast cancer. pp. 1022-1035. In: Cancer epidemiology and prevention, Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

Higginson, J., C.S. Muir, and N. Munoz. 1992. Human cancer: epidemiology and environmental causes. Cambridge Monographs on Cancer Research, Cambridge University Press, Cambridge, Great Britain.

Holly, E.A., C. Lele, and P.M. Bracci. 1998. Hair color products and risk for non-Hodgkin's lymphoma: A population-based study in the San Francisco Bay area. Amer. J. Pub. Hlth. 88(12):1767-73.

Holly, E.A., D. Aston, R. Cress, D. Ahn, and J. Kristiansen. 1995. Cutaneous melanoma in women: I. Exposure to sunlight, ability to tan, and other risk factors related to ultraviolet light. Amer. J. Epi. 141(10):923-33.

Hoppin, J., P. Tolbert, D. Flanders, R. Zhang, D. Daniels, B. Ragesdale, E. Brann. 1999. Occupational risk factor for sarcoma subtypes. J. Epi. 10(3):300-06.

Ince, N., and J.R. Wands. 1999. The increasing incidence of hepatocellular carcinoma. N. Eng. J. Med. 340(10):798-99.

Isselbacher, et al. (eds.). 1994. Harrison's principles of internal medicine. Thirteenth Edition, McGraw-Hill, Inc, New York, NY.

Johnson, P. 1996. The epidemiology of hepatocellular carcinoma. Eur. J. Gastroent. Hepat. 8:845-49.

Kelsey, J.L. 1993. Breast cancer epidemiology. Epi. Rev. 15:7-17.

Kessler, L.G. 1992. The relationship between age and incidence of breast cancer. Cancer Supp. 69(7):1896-1903.

Laden, F., D. Spiegelman, L.M. et. al. 1997. Geographic variation in breast cancer incidence rates in a cohort of U.S. women. JNCI. 89(18):1373-1378.

Laden, F., and D.J. Hunter. 1998. Environmental risk factors and female breast cancer. Annu. Rev. Pub. Hlth. 19:101-123.

Linet, M.S. 1985. The leukemias: epidemiologic aspects. Oxford University Press, New York, NY.

Linet, M.S., J.K. Mclaughlin, A.W. Hsing, S. Wacholder, H.T. Co Chein, L.M. Schuman, E. Bjelke, and W.J. Blot. 1992. Is cigarette smoking a risk factor for non-Hodgkin's lymphoma or multiple myeloma? Results form the Lutheran Brotherhood Cohort Study. Leuk Res 16:621-624.

Linet, M.S., R.S. Cartwright. 1996. The Leukemias, pp. 841-892. In: Cancer epidemiology and prevention. Schottenfeld, D., J.F. Fraumeni, Jr. (eds.). 2nd edition. New York: Oxford University Press, 1996.

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

Malawer, M., M. Link, and S. Donaldson. 1997. Cancer principles and practice of oncology. Fifth Edition, Volume 2. DeVita, Jr., Hellman, S. Rosenberg (eds.). Lippencott-Raven, Philadelphia, PA.

MacMahon, B, P. Cole, M. Lin, C.R. Lowe, A.P. Mirra, B. Ravnihar, E.J. Salber, V.G. Valaoras, and S. Huasa. 1970. Age at first birth and breast cancer risk. Bull. Wld Hlth Org. 43(2):209-221.

MacMahon, B., D. Trichopoulos, B. Brown, et al. 1992. Age at menarche, probability of ovulation and breast cancer risk. Intntl. J. Cancer. 29:13-16.

MCR. 1997. Cancer incidence and mortality in Massachusetts 1987-1994: Statewide report. August 1997. Massachusetts Department of Public Health, Bureau of Health Statistics, Research and Evaluation, Massachusetts Cancer Registry. Boston, MA.

McTiernan, A. 1997. Exercise and breast cancer-time to get moving? N. Eng. J. Med. 336(18):1311-1312.

MDPH 1997. Cancer Incidence and Mortality in Massachusetts 1987-1994. Massachusetts Department of Public Health, Bureau of Health Statistics and Evaluation, Massachusetts Cancer Registry. August 1997.

MDPH 1999. Cancer Incidence And Mortality In Massachusetts 1992-1996. Massachusetts Department of Public Health, Bureau of Health Statistics and Evaluation, Massachusetts Cancer Registry. 1999.

Miller, K., D. rosenthal, K. Poliquin, H. Weinstein. 1990. Leukeia. In: Cancer Manual. 8th ed. Osteen, ed. American Cancer Society, Massachusetts Division, Boston, MA.

Miller, B., L. Ries, B. Hankey, et al. (eds.). 1992. Cancer statistics review: 1973-1989. NCI. NIH Publ No. 92-7289.

Miller, R., J. Boice, and R. Curtis. 1996. Bone cancer. pp. 971-983. In: Cancer epidemiology and prevention, Second Edition. D. Schottenfeld, and J. F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

Mueller, N. E. 1996. Hodgkin's disease. pp. 893-919. In: Cancer epidemiology and prevention. Second Edition. D. Schottenfeld, and J. F. Fraumeni, Jr. (eds.). Oxford University Press. New York, NY.

Nasca, P.C., M.C. Mahoney, and P.E. Wolfgang. 1992. Population density and cancer incidence differentials in New York State, 1978-82. Cancer Causes and Control 3:7-15.

NCI. 1996. Cancer Rates and Risks. Fourth Edition. National Cancer Institute, National Institutes of Health, Publication No. 96-691.

NIEHS. 1998. Working Group Report: Assessment of health effects from exposure to power-line frequency electric and magnetic fields. National Institute of Environmetnal Health Sciences. NIH Publication No. 98-3981.

NRC. 1998. Health Effects of Exposure to Radon. National Research Council, Committee on Health Risks and Exposure to Radon (BEIR VI). National Academy Press, Washington, D.C.

Preston-Martin, S. 1996. Epidemiology of primary CNS neoplasms. Neurol Clin. 14 (2):273-90.

Preston-Martin, S., and W. Mack. 1996. Neoplasms of the nervous system. pp. 1231-1281. In: Cancer epidemiology and prevention. Second Edition. D. Schottenfeld, and J.F. Fraumeni (eds.). Oxford University Press, New York, NY.

Rothman, K., and J. Boice. 1982. Epidemiological analysis with a programmable calculator. Epidemiology Resources, Inc., Boston, MA.

Scheinberg, D., and D. Golde. 1994. The leukemias. pp. 1764-1774. In: Harrison's principles of internal medicine. Thirteenth Edition, Vol. 2. Isselbacher et al. (eds.). McGraw-Hill, Inc., New York, NY.

Scherr, P.A., and N.E. Mueller. 1996. Non-Hodgkin's lymphomas. pp. 920-945. In: Cancer epidemiology and prevention, Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

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

Seitz, H.K., G. Poschl, and U.A. Simanowski. 1998. Alcohol and cancer. Recent Dev. Alcohol. 14:67-95.

Siegel, M. 1993. Smoking and leukemia: evaluation of a causal hypothesis. Am. J. Epi. 138:1-9.

Silverman, D., A. Morrison, and S. Devesa. 1996. Bladder cancer. pp. 1156-1179. In: Cancer epidemiology and prevention, Second Edition. D. Schottenfeld, and J.F. Fraumeni, Jr. (eds.). Oxford University Press, New York, NY.

Stuver, S.O. 1998. Toward global control of liver cancer? Seminal of Cancer Biology. 8(4):299-306.

Suit, H., H. Mankin, J. Corson, A. Elias, and S. Singer. 1996. Cancer Manual, Ninth Edition. American Cancer Society, Atlanta, GA.

Swanson, C.A., R.J. Coates, K.E. Malone, M.D. Gammon, J.B. Schoenberg, D.J. Brogan, M. McAdams, N. Potischman, R.N. Hoover, and L.A. Brinton. 1997. Alcohol consumption and breast cancer risk among women under age 45 years. Epidemiology. 8:231-237.

Tatham, L., et al. 1997. Occupational risk factors for subgroups of non-Hodgkin's lymphoma. Epidemiology. 8(5):551-558.

Thun, J.J., C.A. Lally, J.T. Flannery, E.E. Calle, W.D. Flanders, and C.W. Heath, Jr. 1997. Cigarette smoking and changes in the histopathology of lung cancer. J. Ntl. Can. Inst. 89(21):1580-1586.

Thune, I, T Brenn, E. Lund, and M. Gaard. 1997. Physical activity and the risk of breast cancer. N. Eng. J. Med. 335(18):1269-1275.

van Dijck, M.A., M.J.M. Broeders, and A.L.M. Verbeek. 1997. Mammographic screening in older women, is it worthwhile? Drugs and Aging. 10(2):69-79.

Weiss, H., N.A. Potischman, L. Brinton, D. Brogan, R.J. Coates, M.D. Gammon, K.E. Malone, and J.B. Schoenberg. 1997. Prenatal and Perinatal Factors for Breast Cancer in Young Women. Epidemiology. 8(2):181-187.

Wigle, D.T., R.M. Semenciew, K. Wilkins, D. Riedel, L. Ritter, H.I. Morrison, and Y. Mao. 1990. Mortality study of Canadian male farm operators: non-Hodgkin's lymphoma mortality and agricultural practices in Saskatchewan. J. Ntl. Can. Inst. 82(7):575-582.

Wynder, E., L.A. Cohen, J.E. Muscat, B. Winters, H.T. Dwyer, and G. Blackburn. 1997. Breast cancer: Weighing the evidence for a promoting role of dietary fat. J. Ntl. Can. Inst. 98(11):766-775.

Zahm, S.H., D.D. Weisenburg, P.A. Babbitt, R.C. Saal, J.B. Vaught, K.P. Cantor, and A. Blair. 1990. A case-control study of non-Hodgkin's lymphoma and the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) in eastern Nebraska. Epidemiology. Vol. 1, No. 5, pp. 349-356.

Zahm, S.H., D.D. Weisenburg, P.A. Babbitt, R.C. Saal, J.B. Vaught, and A. Blair. 1992 Use of hair coloring products and the risk of lymphoma, multiple myeloma, and chronic lymphocytic leukemia. American Journal of Public Health. Vol. 82, pp. 990-997.

Zahm, S.H., D.D. Weisenburg, P.A. Babbitt, R.C. Saal, J.B. Vaught, and A. Blair. 1993. The role of agricultural pesticide use in the development of non-Hodgkin's lymphoma in women. Arch. Environ. Health. 48(5):353-58.