Prepared by:
Colorado Department of Public Health and Environment

October 2003


# Colorado Department of Public Health and Environment 

For More Information, Contact:
Michael Wilson, Ph.D.
Disease Control and Environmental Epidemiology Division Colorado Department of Public Health and Environment

4300 Cherry Creek Drive South
Denver, Colorado 80246
303-692-2646
michael.wilson@state.co.us

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## ExECuTIVE Summary

This document is the second of two reports in a continuing series on the findings of cancer surveillance for communities in the northeast Denver metropolitan area, surrounding the Rocky Mountain Arsenal. The initial report, Analysis of Diagnosed vs. Expected Cancer Cases for the Northeast Denver Metropolitan Area in the Vicinity of the Rocky Mountain Arsenal, 1979-1996, was released in January, 2003. Cancer surveillance is one of the community health activities conducted by the Rocky Mountain Arsenal Medical Monitoring Program at the Colorado Department of Public Health and Environment. Cancer surveillance in the communities surrounding the arsenal was undertaken in response to recommendations made to the department by the Rocky Mountain Arsenal Medical Monitoring Advisory Group.

Cancers are common diseases, and therefore remain at the forefront of public health concern. Over 17,000 new cases of cancer are registered annually in Colorado, and Coloradans have, on average, an individual lifetime risk of developing cancer of approximately one chance in three. Whether an individual develops a cancer during his or her lifetime may be greatly influenced by a variety of complex factors that make determining causes a difficult task. We may, however, monitor incidence rates so as to be alert to significant deviation from the expected background rates. In Colorado, such monitoring is possible by using data available from the Colorado Central Cancer Registry, which is based at the Department of Public Health and Environment. All cancers diagnosed in Colorado are reported to the Cancer Registry with the exception of nonmelanoma skin cancers.

The objectives of cancer surveillance are to use cancer incidence data collected by the Colorado Central Cancer Registry to: (1) establish existing rates of cancer incidence prior to the soil remediation at the arsenal, (2) analyze cancer incidence rates for significant temporal or spatial changes during and after the arsenal soil remediation, and (3) investigate any increased, or otherwise unexplained, rates of cancer. This report addresses objectives 2 and 3 above for a four-year period, 1997-2000, beginning about the time that soil remediation commenced at the Rocky Mountain Arsenal. The January 2003 report for the same geographic area addressed objectives 1 and 3 by analyzing the 1979-1996 cancer incidence data (CDPHE, 2003).

The study design used in this analysis focuses on a numerical summary of cancer incidence in each of the communities surrounding the arsenal. The results aid in determining whether the number of certain cancers is greater or less than expected and whether that difference is statistically significant. The study does not make detailed examinations of individual cases and does not allow conclusions to be made about causal association between exposure and any single cancer or group of cancers.

The study examined three areas: Area 1 (north and west of the Rocky Mountain Arsenal including north Commerce City), Area 2 (Commerce City), and Area 3 (Montbello and Green Valley Ranch). Area 1 has been further subdivided into Areas 1a and 1b to better track cancer incidence in this region of rapid population growth.

Cancer rates of the Denver metropolitan area, excluding the study area, over the 1997-2000 time period were used as standards for estimating the expected numbers of cancers.

The year 1997 was selected as the starting point for the present analysis to coincide with the initiation of the arsenal soil remediation. For two reasons, however, cancer cases diagnosed during the 1997-2000 period are unlikely to be related to soil remediation activities at the Rocky Mountain Arsenal. These are: (1) air monitoring at the arsenal has not shown an ongoing or significant off-site release of arsenal-related contaminants, and (2) the process of cancer development and the associated disease latency suggest that if cancers were initiated during the 1997-2000 time period, diagnoses would not be expected until a later time period.

Similar to the finding of the Colorado Department of Public Health and Environment's prior report on cancer incidence in the northeast Denver metropolitan area for the period 1979-1996 (CDPHE, 2003), no generalized elevation of cancer was observed during the years 1997-2000. Elevations of cancer that were observed in the current analysis were again of specific anatomical sites, however, with differences from the earlier evaluation.

In the prior department analysis of cancer incidence in the study areas for the period 1979-1996, significantly higher than expected numbers of diagnoses were reported for certain anatomical sites and gender (CDPHE, 2003). Most of these elevations were not observed during the 19972000 period. Additionally, the patterns of cancer incidence in the study area populations during 1997-2000, such as the dominant forms of cancer, cell type distribution and age distribution, appear generally consistent with the trends in the comparison population and those described in the epidemiological literature.

The specific cancer sites observed to be statistically significantly elevated during 1997-2000 were lung cancer in both males and females in Areas 1a, 1 b and 1 Combined; pancreatic cancer in males and females in Areas 1a and 1 Combined; lymphoma in Area 2 males; and malignant brain cancer in males and females in Area 3. The cases contributing to each of these elevations were dispersed among the several census tracts of the respective areas, showing no unexpected groupings. The specific cancer sites observed to be statistically significantly low during 19972000 were prostate in males in Area 1b and 2, and melanoma in males and females in Area 2.

Smoking histories were previously reported as possibly playing a significant role in many cancers diagnosed during 1979-1996. This again appears likely for some cases reported in 19972000. Lung and pancreatic cancer are both smoking-related and Cancer Registry abstracts indicate that a significant number of the reported cases had a history of smoking. Other factors, such as exposure to carcinogens in the occupational, indoor, and ambient air also may contribute to the overall individual and population risk. Genetic predisposition and infectious agents are also potential factors that have been identified in the epidemiological literature.

Bladder cancer continues a trend first reported in earlier department and Colorado State University analyses, being elevated in Area 1 Combined during the period 1997-2000, though not achieving statistical significance. As in the earlier report, there is a high frequency of smoking histories among the cases. And similar to lung and pancreatic cancer, smoking is an important risk factor for bladder cancer, accounting for as many as 60 percent of all cases.

The elevation of lymphomas in Area 2 may be primarily attributable to the short period of observation, 1997-2000. Extending this period of observation back several years, when there was unexpectedly low lymphoma incidence, would result in very close agreement between the number of diagnosed and expected cases. The selection of geographic boundaries may also account for the apparent lymphoma elevation in Area 2 since in all of Area 1 Combined, there were no cases diagnosed over the 1997-2000 time period when several were expected. The causes of lymphomas are largely unknown. However, lymphocyte hyperactivity appears to be a significant factor. Infectious agents, chemical exposures and/or certain occupations have all been proposed as potential stimulants or acting as carcinogens through some other process.

The current scientific understanding of brain cancer and associated risk factors limits further analysis of the significant elevation of malignant brain cancer in Area 3. The incidence of benign brain tumors does not show a similar excess of cases nor pattern with respect to age at diagnosis. An elevation of malignant brain cancer was not observed in earlier time periods (1979-1996) in Area 3 and is not seen in Areas 1 or 2 during 1997-2000. The significant Area 3 elevation of malignant brain cancers in the 45-54 age group appears inconsistent with the typical case distribution in the general population in that brain cancer is most common among older age groups. This difference may, however, be an artifact of the small population of Area 3, relative to the comparison population, in combination with random chance.

A wide variety of brain cancer risk factors have been proposed in the epidemiological literature, including chemical, physical and infectious agents. None of these factors have been confirmed with the exception of ionizing radiation, congenital and genetic disorders and immunosupression.

Some of the statistically significant findings observed during the 1997-2000 period may be a chance occurrence. Among the 200 independent statistical tests performed, the number of elevated findings was equal to the number expected due to chance alone. The number of statistically significantly low findings was fewer than would be expected due to chance. Ratios based on less than three cases, however, were not statistically tested and this may account for the smaller number of significantly low findings.

The Rocky Mountain Arsenal Medical Monitoring Program at the Colorado Department of Public Health and Environment will continue general monitoring of the cancer incidence patterns in the northeast Denver metropolitan area and follow the incidence of those types of cancer identified as elevated prior to 2001.

## INTRODUCTION

This document reports findings of cancer surveillance for 1997-2000 for Colorado communities in the northeast Denver metropolitan area, surrounding the Rocky Mountain Arsenal (RMA). A prior report, Analysis of Diagnosed vs. Expected Cancer Cases for the Northeast Denver Metropolitan Area in the Vicinity of the Rocky Mountain Arsenal, 1979-1996, evaluated the same geographic area, but for an earlier time period (CDPHE, 2003).

Cancer surveillance is one of the community health activities conducted by the Rocky Mountain Arsenal Medical Monitoring Program at the Colorado Department of Public Health and Environment (CDPHE) ${ }^{1}$. Cancer surveillance in the communities surrounding the arsenal was undertaken in response to recommendations made to the department by the Rocky Mountain Arsenal Medical Monitoring Advisory Group².

Cancer is a general term applied to a wide variety of different diseases characterized by uncontrolled growth and spread of abnormal cells. These diseases are common within the population, and therefore remain at the forefront of public health concern. Over 17,000 new cases of cancer are registered annually in Colorado, and Coloradans have, on average, an individual lifetime risk of developing cancer of approximately one chance in three ${ }^{3}$. Whether an individual develops a cancer during his or her lifetime may be greatly influenced by a variety of factors, many of which are not currently understood. We do know that the development of cancer is a complex, multistage ${ }^{4}$, process involving both external (chemical, radiation, and viruses) and internal factors (hormonal, immune conditions, and inherited mutations). Unfortunately, this complexity and its associated latencies, that is, the time period between the initiation of the cancer and subsequent diagnosis ${ }^{5}$, have limited scientific efforts to identify causative factors or combinations of factors. We may, however, monitor incidence rates so as to be alert to significant deviation from the expected background rates. This in turn allows investigation of deviations with respect to potential environmental associations.

[^0]In Colorado, surveillance of cancer incidence is possible using data collected by the Colorado Central Cancer Registry at Colorado Department of Public Health and Environment. All cancers diagnosed in Colorado are reported to the Cancer Registry with the exception of non-melanoma skin cancers. The registry is mandated by Colorado law and by Colorado Board of Health regulation. Information is collected from all Colorado hospitals, pathology labs, outpatient clinics, physicians solely responsible for diagnosis and treatment and from state vital statistics. Pertinent data is registered on all malignant tumors, except basal and squamous cell carcinomas of the skin. All individual patient, physician, and hospital information is confidential as required by Colorado law.

## ObJECTIVES

The objectives of cancer surveillance are to use cancer incidence data collected by the Colorado Central Cancer Registry to: (1) establish existing rates of cancer incidence prior to the arsenal soil remediation, (2) analyze cancer incidence rates for significant temporal or spatial changes during and after the arsenal soil remediation, and (3) investigate any increased, or otherwise unexplained, rates of cancer.

This report addresses objectives 2 and 3 above for a four-year period, 1997-2000, beginning about the time that soil remediation commenced at the Rocky Mountain Arsenal. The January, 2003 report for the same geographic area addressed objectives 1 and 3 by analyzing the 19791996 cancer incidence data (CDPHE, 2003).

## Methods

The epidemiological study design used in this analysis of diagnosed and expected numbers of cancer cases is descriptive and ecological. The descriptive element provides a numerical summary of disease frequency, whereas the ecological component examines entire communities or populations, rather than individuals. Ecological studies have been conducted frequently in communities adjacent to potential environmental exposures, since they are efficient and can be completed within a reasonable period of time. Ecological studies are usually viewed as exploratory and hypothesis generating because the analyses made are for large or small groups of people, rather than for individuals. A weakness inherent in studies in which the analysis is at the group level, rather than the individual, is that information on potential confounders, for example, lifestyle, occupation, or residential history, is lacking or limited and the data cannot be fully examined for their effects. Another weakness of ecological studies is that, because potential exposure is not actually measured, geographical area of residence is used as a crude substitute. The use of a geographical area raises the likelihood of exposure misclassification, which reduces the ability of the study to observe a statistically significant difference between groups. Lastly, the design of this cancer incidence analysis does not allow conclusions to be made about causal association between exposure and any single cancer or group of cancers. The study design and results only aid in determining whether the number of certain cancers is greater or less than expected and whether that difference is statistically significant.

As part of this present investigation, cancer diagnosis counts were compared to expected counts for an area in the vicinity of the Rocky Mountain Arsenal for the time period of 1997-2000 when cancer reporting was complete and the 1990 and 2000 census years of population could be used. The boundaries of this area were selected for this analysis based on 1990 U.S. Census tract designations. The study area was composed of three smaller areas (Areas 1 through 3) based on the geography first described in the 1993 report Cancer Incidence in the Northeastern Denver Metro Area: Report of the Ad Hoc Panel (CDPHE, 1993). In the present investigation, as in the recent 2003 report, Area 1 has been further subdivided into Areas 1a and 1b to better track cancer incidence in this region of rapid population growth. All five of these subdivisions of the overall study area are described below and shown in Figure 1.

Area 1a, north of the Rocky Mountain Arsenal, was defined as census tract 85.12 with a population of 1,334 in 1980, 1,405 in 1990, and 2,194 in 2000. The boundaries were Henderson Rd., E. $124^{\text {th }}$ Ave., State Hwy. 51, E. $120^{\text {th }}$ Ave., Tower Rd., Irondale Rd. (E. $88^{\text {th }}$ Ave.), Buckley Rd., E. $96^{\text {th }}$ Ave., McKay Rd., and the South Platte River.

Area 1b, northwest of the Rocky Mountain Arsenal, was defined as census tracts 88.01 and 88.02 with a combined population of 7,766 in 1980, 6,971 in 1990, and 8,513 in 2000. The boundaries were McKay Rd., E. $96^{\text {th }}$ Ave., State Hwy. 2, E. $72^{\text {nd }}$ Ave., U.S. Hwy. 85 , E. $74^{\text {th }}$ Ave. (State Hwy. 224), and the South Platte River.

Area 1 was defined as Area 1a and Area 1b together with a combined population of 9,100 in 1980, 8,376 in 1990, and 10,707 in 2000.

Area 2, west of the Rocky Mountain Arsenal, was defined as census tracts 87.03, 87.05, 87.06, and 89.01 with a combined population of 17,292 in 1980, 15,740 in 1990, and 18,939 in 2000. The boundaries were E. $74^{\text {th }}$ Ave. (State Hwy. 224), U.S. Hwy. 85, E. $72^{\text {nd }}$ Ave., State Hwy. 2, Quebec, Denver-Adams County Line, and the South Platte River.

Area 3, south of the Rocky Mountain Arsenal, was defined as census tracts 41.05, 83.03, 83.04, $83.05,83.06,83.10,83.11$, and 83.12 with a combined population of 16,828 in 1980, 21,626 in 1990, and 39,311 in 2000. The boundaries were the Denver-Adams County Line, E. $56^{\text {th }}$ Ave., Picadilly Rd., Denver-Adams County Line, Tower Rd., Denver-Adams County Line, E. $46^{\text {th }}$ Ave., Denver-Adams County Line, Montview Blvd., Syracuse, E. $23{ }^{\text {rd }}$ Ave., Quebec, E. $48{ }^{\text {th }}$ Ave., Denver-Adams County Line, and Quebec.

This analysis examined all diagnosed malignancies combined, as well as cancers of the 30 anatomical sites listed in Table 1. All cases of cancer diagnosed between 1997 and 2000 that were residents in the study areas at the time of diagnosis were identified. Data for an analysis of this type is obtained from the Colorado Central Cancer Registry. The address at the time of diagnosis for each case was used to assign residence within the census boundaries.

Identification and registration of cancer cases by the Cancer Registry involves standard processes including searching hospital medical charts, pathology laboratory records, and examining death certificate information.

| Table 1 - Anatomical sites of cancers included in the Analysis of |  |
| :---: | :---: |
| Diagnosed vs. Expected Cancer Cases for the Northeast Denver Area in |  |
| the Vicinity of the Rocky Mountain Arsenal, 1997-2000. |  |
| Salivary Gland | Kidney |
| Oral | Thyroid |
| Nasopharynx | Other Endocrine |
| Other Pharynx | Brain |
| Esophagus | Bone |
| Stomach | Leukemia |
| Small Intestine | Multiple Myeloma |
| Colorectal | Lymphoma |
| Liver | Soft Tissue |
| Other Biliary | Prostate |
| Pancreas | Testis |
| Larynx | Female Breast |
| Lung | Cervix |
| Melanoma | Uterus |
| Bladder | Ovary |
|  |  |

U.S. Census counts of population by age, race/ethnicity, and gender for 1990 and 2000 were obtained from the Colorado Division of Local Government (State Demographers Office) or from the U.S. Census website.

Cancer rates of the Denver metropolitan area, excluding the study area, over this time period were used as standards for calculating expected numbers of cancers for the areas because: (1) complete age-specific rates by race/ethnicity and gender were available from the Cancer Registry, and (2) the Denver metropolitan area serves as a local standard of comparison, which is preferable to using a statewide or national standard since these areas may be less likely to reflect local background cancer rates. The Denver metropolitan area is defined as the six counties of Adams, Arapahoe, Boulder, Douglas, and Jefferson, and the City and County of Denver. This local standard area included what now is the new City and County of Broomfield, except for a small section in Weld County, which has a population of nine people.

Cancer rates from the Cancer Registry for men and women of comparable race/ethnic groups and age groups were used to calculate the expected number of cancers for the areas. A cancer rate is the number of new cancer cases diagnosed per 100,000 population in a one-year period of time. The population in each area, stratified by age, gender, and race/ethnicity, was multiplied by the cancer rate for each age group, gender, and race/ethnic group in the comparison population to produce the expected number of cancers.

Figure 1. Analysis of diagnosed vs. expected cancer cases for the northeast Denver area in the vicinity of the Rocky Mountain Arsenal, Colorado, 1997-2000 - Surveillance Areas 1a, 1b, 2, and 3.


A diagnosed-to-expected ratio is then calculated by dividing the number of cancers diagnosed in the area by the number of expected cases. If the ratio is greater than 1 , then more cancer cases than expected were reported in the area. When this occurs, the next step is to look more closely at that relationship. It is important to know if that ratio could have been higher by chance alone, so a confidence interval is calculated for the ratio. The confidence interval has a lower number or minimum value and a higher number, maximum value. It is common to use a 95 percent confidence interval which means that we are 95 percent sure that the true ratio is within the range between the lower and higher values. If the ratio is greater than 1 but the confidence interval includes the number 1, then the ratio is within expected statistical limits. If the confidence interval does not include the number 1, then the ratio is statistically significant. A statistically significant elevated ratio means that there were more diagnosed cases than expected and that there is less than a 5 percent chance that this greater number is due to chance alone.

Because the estimate of expected cancers is based on the larger Denver metropolitan region population, this estimate will be a central tendency, or average number, of expected cases for the time period, 1997-2000. Cancer rates for specific populations, such as in smaller cities, towns, or neighborhoods, will likely be either higher or lower than the "expected average." Smaller populations tend to show greater variability. The variability of small populations is statistically reflected in the 95 percent confidence interval for the ratio of diagnosed to expected cases. Confidence intervals for small populations are wider than for large populations. When the expected number of cancer cases is small, slight increases can result in seemingly large diagnosed to expected ratios. For example, if only one case of cancer is expected in a small population in a given year, and two were actually diagnosed, the ratio would of course show a doubling of cases. But, in this situation, twice the number of expected cases would be within expected statistical limits. Statistical testing was not done on ratios with less than three diagnosed cases because of the inherent variability in such small numbers.

When statistically significant elevations of diagnosed-to-expected ratios were observed, other data recorded in the Cancer Registry abstract were also reviewed. These data help to characterize potential exposure commonalities among the cases, including the presence of important known risk factors for certain cancers, and separating selected anatomical categories of cancer into cell types. The case abstract data reviewed included occupation, smoking history, alcohol consumption and cell type.

## Findings

Tables A1- A15, located in the Appendix, display the number of diagnosed cancers in each of the study areas (Area 1a, 1b, 1 Combined, 2, and 3) by cancer type and gender for 1997-2000, compared to the number that would be expected based on the population of male and female residents in the areas by race/ethnicity and age groups. Tables A16-A22, also located in the Appendix, display additional detail for selected areas, gender groups and/or cancer types that had statistically high findings or particularly relevant findings when compared to the previously reported 1979-1996 time frame (CDPHE, 2003). Cancer rates from the Cancer Registry for men and women of comparable race/ethnic and age groups were used to calculate the expected number of cancers for the areas. The ratios of diagnosed to expected cases along with the 95
percent confidence intervals for these ratios provide information about the relative rate of cancer in these areas. Note that observed/expected ratios and confidence intervals are displayed with rounding to two decimal points.

Area 1a, 1b and Combined Area 1 - Tables A1-A9 display statistics for Areas 1a, 1b, and the combined Area 1 for 1997-2000 for males and females separately and combined.

Area 1a - Tables A1-A3 show that the number of all cancers combined diagnosed in Area 1a was generally close to the number expected in this area during this time period. There were a few exceptions to this general finding. Table A1 shows that for 1997-2000 there was more pancreatic cancer diagnosed in Area 1a than expected (three cases compared to about one expected). Pancreatic cancer was also elevated in Area 1b and Area 1 Combined. Further information about this elevation is reported under "Combined Area 1." Tables A1-A3 show that for 1997-2000 there were more lung cancer cases diagnosed than expected in Area 1a. For males and females combined, Table A1 shows that 10 cases were reported, compared to about three cases expected, for a statistically high ratio of 3.36. Lung cancer elevations also were found in Area 1b and the Combined Area 1 for both genders, as reported in the Combined Area 1 section of this report.

Area 1b - Tables A4-A6 show that the number of all cancers combined diagnosed in Area 1b was generally close to the number expected in this area during this time period. Tables A4-A6, however, display lung cancer elevations for both genders. Again, lung cancer elevations are described in more detail in the Combined Area 1 section of this report. Prostate cancer in males was found to be statistically significantly low, with 9 cases diagnosed and approximately 17 expected, a ratio of 0.52 , as shown in Table A5.

Combined Area 1 - Tables A7-A9 show that the number of all cancers combined diagnosed in Area 1 was generally close to the number expected in this area during the period 1997-2000. Two cancers, however, pancreas and lung, had statistically high ratios, and the number of bladder cancer cases was high enough to be within one case of statistical significance.

Table A7 shows that there were eight pancreatic cancer diagnoses among males and females in Area 1 compared to about three or four cases expected during 1997-2000, for a statistically high ratio of 2.40. Tables A8 and A9 show a ratio for males of 2.20 and for females of 2.63, though these individual ratios were within expected statistical variation. Table A16 shows that almost all of these pancreatic cancers, seven out of eight cases, were among White, non-Hispanic persons and the ratio for only White, non-Hispanic cases was statistically high at 3.03 (seven cases compared to about two or three cases expected). No specific age category showed a statistically high ratio. Colorado Central Cancer Registry abstracts showed a variety of occupations among these eight cases. Three of the eight cases ( 38 percent) had a history of smoking documented in Cancer Registry abstracts. Including only abstracts where smoking information was recorded, 50 percent of cases were smokers. Four of eight cases ( 50 percent) had a history of alcohol usage, and including only abstracts where alcohol information was recorded, 67 percent of cases had used alcohol. The distribution of histological cell types among these eight cases was similar to the distribution found in the Denver metropolitan area. Adenocarcinomas accounted for 50 percent of cases in Area 1 Combined compared to 69 percent
of Denver cases, other carcinomas accounted for 25 percent of cases in Area 1 compared to 11 percent of Denver cases, and the remainder were coded to a general malignant neoplasm category, 25 percent of Area 1 cases compared to 20 percent of Denver cases.

Table A7 shows that there were 34 lung cancer diagnoses in Area 1 Combined compared to about 15 or 16 cases expected during 1997-2000 for a statistically high ratio of 2.22. Tables A8 and A9 show similar findings for the male (2.25) and female (2.18) ratio individually, with each being statistically high. Table A17 shows that most cases, or 30 out of 34 , were White, nonHispanic and the ratio for only White, non-Hispanic cases was statistically high at 2.52, 30 cases compared to about 12 expected. The distribution of cases by age showed elevations in every age group from 55 and above with the ratios of 3.16 with 11 cases compared to about three or four expected and 2.57, 16 cases compared to about six or seven expected, for the 55-64 and 65-74 age groups, respectively, being statistically high. Cancer Registry abstracts showed a variety of occupations among these 34 cases. About 76 percent of these cases, or 26 out of 34, had a history of smoking. Limiting this calculation to only cases with smoking information recorded on the abstracts, 93 percent of lung cancer cases, 26 out of 28 , were smokers. There were no uncommon histological cell types recorded and the distribution of types of lung cancer in Area 1 Combined during 1997-2000 were similar to the Denver metropolitan area. The cases included the major forms of lung cancer, squamous cell carcinomas, 24 percent vs. 19 percent in metropolitan Denver; large cell carcinomas, 9 percent vs. 10 percent; small cell carcinomas, 24 percent vs. 16 percent; adenocarcinomas, 29 percent vs. 34 percent; and all other types, 14 percent vs. 21 percent.

Table A7 also shows that there were 10 male and female bladder cancer cases in Area 1 Combined compared to about five or six cases expected during 1997-2000, resulting in a ratio of 1.84, which is within one case of being statistically high. Table A8 shows that males account for most of the elevated finding with nine cases compared to about four cases expected for a ratio of 2.15. Table A18 shows that all of the bladder cancer cases were White, non-Hispanic, and the ratio for White, non-Hispanic cases only was statistically high at 2.14 with 10 cases compared to about five expected. The distribution of cases by age showed an elevation in the 65-74 age group with the ratio of 4.33 being statistically high with eight cases compared to about two cases. Cancer Registry abstracts showed a variety of occupations for these 10 cases. Five of the 10 cases or 50 percent had a history of smoking documented in Cancer Registry abstracts. Including only abstracts where smoking information was recorded, five of seven cases or 71 percent were smokers. Almost all of the 10 bladder cancers were transitional cell carcinomas or 80 percent, consistent with the predominance of this cell type for this cancer.

Area 2 - Tables A10-A12 show that the number of all cancers combined diagnosed in Area 2 was generally close to the number expected in this area during the 19972000 time period. Melanoma in males and females is shown in Table A10 to be statistically significantly low, with 5 cases diagnosed and approximately 13 expected; a ratio of 0.39 . Prostate cancer in males was shown in Table A11 to have a statistically significantly low ratio of 0.59 , with 23 cases diagnosed and approximately 39 expected. Table A11 also shows that there
were 14 male lymphomas diagnosed in Area 2 during 1997-2000 compared to about seven or eight cases expected for a statistically high ratio of 1.87. Table A20 shows, however, that none of the ratios for the race/ethnic categories or specific age groups was statistically elevated.

Of the 14 cases diagnosed in Area 2, two were Hodgkin's disease and 12 were non-Hodgkin's lymphoma. Of the 12 non-Hodgkin's lymphoma cases, 50 percent were B-cell tumors vs. 46 percent in the Denver metropolitan area generally, 8 percent were T-cell tumors vs. 6 percent in the metropolitan area, and 42 percent were other or unknown vs. 48 percent in the metropolitan area.

As previously reported (CDPHE, 2003), the number of childhood cases of acute lymphoblastic leukemia (ALL) in Area 2 during the 1989-1996 time period was significantly elevated. The current analysis shown in Tables A10 and A19 for 1997-2000 does not show a persistence of that leukemia elevation. Table A10 shows that there were 10 leukemias diagnosed compared to about seven cases expected among all age groups, for a ratio of 1.42 , which is within expected statistical variation. As seen in Table A19, none of the ratios for the race/ethnic categories or specific age groups were statistically elevated. There was no childhood leukemia reported in Area 2 over the 1997-2000 time period.

Area 3 - Tables A13-A15 show that the number of all cancers combined diagnosed in Area 3 was generally close to the number expected in this area during 1997-2000. Cancers of one organ system, malignant brain and central nervous system cancers, did have higher diagnosed to expected ratios. Table A13 shows that the ratio of 2.29 for malignant brain and central nervous system cancers diagnosed in Area 3 during 1997-2000, with 12 cases compared to about five or six expected, was statistically high. Tables A14 and A15 show that the elevation was also seen in males and females individually, with the male ratio being 1.82, six cases compared to about three expected, and the female ratio being 3.07 with six cases compared to about two expected. The female ratio was statistically high. Table A21 shows that the 12 cases were distributed mostly between the White, non-Hispanic group for a ratio of 2.09, four cases compared to about two expected, and the Black group for a ratio of 2.92 , five cases compared to about two expected, though both of these ratios were within expected statistical variation. The same table shows that only the 45-54 age group had a statistically high ratio (4.45) with six cases compared to about one or two expected. The Cancer Registry abstracts showed a variety of occupations among the 12 cases. Astrocytomas and glioblastomas were the most common brain tumors, accounting for 67 percent of malignant cases in Area 3 compared to about 57 percent of malignant cases in the Denver metropolitan area. No other malignant cell type had more than one case represented in this time period.

The Colorado Central Cancer Registry also collects data on benign brain tumor incidence. Table A22 was prepared to evaluate the effect of combining malignant and benign brain tumors in Area 3. Combining malignant and benign tumors resulted in 16 cases diagnosed compared to about 14 cases expected for a ratio of 1.17 , which is within expected statistical variation. Ratios for the race/ethnic groups were not elevated, but an elevation seen in the 45-54 age group persisted largely due to the number of malignant brain cancers (see Table A21), with the ratio of 2.66 (8 cases compared to about three cases expected) being statistically high.

## Multiple Comparisons

In this study of cancer in the northeast Denver area for the period 1997-2000, with 200 independent statistical tests conducted on separate cancer sites by gender for several different areas, there were five ratios statistically higher than expected with 2.5 percent of the tests compared to about 2.5 percent predicted by chance alone and three ratios statistically lower than expected with 1.5 percent of the tests compared to about 2.5 percent predicted by chance alone. Including all cancers combined, both genders combined for all cancers and cancers of individual anatomical sites, and additional tests done by race/ethnicity and age for several cancers, a total of 507 comparisons were made. Of these comparisons, 21 ratios, or 4.1 percent of the tests, were statistically higher than expected and five ratios, or 1 percent of the tests, were statistically lower than expected.

## DISCUSSION

Cancer incidence, when compared to a standard population with statistical testing procedures, allows the identification of subpopulations with "higher than average" rates of specified categories of cancer. To interpret this information, however, other information may be readily available from the Cancer Registry, including potential risk factors, such as occupation, smoking history, and alcohol consumption; the frequency of cancer of specific anatomical sites; and the distribution of histological cell type within those anatomical sites. An equally important source of information for interpretation of cancer incidence data is the epidemiological literature. The significance of these types of information is discussed below.

Review of occupational data may reveal patterns suggesting areas for further study. Certain occupations may have exposures to specific carcinogenic agents, and broad categories of occupation, such as farming and industrial work, may involve exposures to a variety of carcinogens. Data contained in the Cancer Registry case abstract do not, however, provide a complete picture of the life-long occupational experience.

In many cases, a history of tobacco use is recorded in the Cancer Registry abstracts, and this information provides at least some information about a significant exposure to a known carcinogen. Exposure to tobacco and tobacco smoke, including smoking, passive inhalation, and use of smokeless tobacco, accounts for nearly one-third of all cancer cases in developed countries (American Cancer Society, 2001). Continuous, active smoking involves by far the greatest risk. The most pronounced risk is for cancer of the lung and larynx, and this risk may be 10-30 times greater than for non-smokers (Wynder, 1998; Doll et al., 1994). Increased cancer risk is also evident for other organ tissues including the oropharynx, esophagus, pancreas, bladder, kidney, colorectal, and acute myelocytic leukemia. Suggestive evidence has associated smoking with hepatocellular cancer, squamous cell carcinoma of the uterine cervix, and possibly, breast cancer (Morabia et al., 1996; Lash and Aschengrau, 1999).

Case history of alcohol consumption also may be recorded in the Cancer Registry abstracts. Similar to tobacco use, this record provides some information about an important exposure related to cancer incidence. Excess consumption of alcohol is associated with cancer of the oral
cavity, pharynx, esophagus, liver and possibly the pancreas. Although only pancreatic cancer was elevated in the study area during the 1997-2000 time period, epidemiological evidence suggests that approximately 5 percent of cancer deaths in the U.S. are related to alcohol consumption, but other factors may also be involved (American Cancer Society, 2001).

The distribution of reported cancers among the three most common anatomical sites can be reviewed for consistency with the expected distributions based on the comparison population. Nationally and in Colorado, the three most prominent cancers among males are prostate, lung/bronchus, and colorectal. Among females, the most common cancers are breast, colorectal, and lung/bronchus. The percentage at which these cancers are represented among all cancer in Colorado is shown in Table 2.

| Table 2 - Major sites and percentages of cancer among male and female <br> Colorado residents |  |  |  |
| :---: | :---: | :---: | :---: |
| Males | Females |  |  |
| Prostate | $29.5 \%$ | Breast | $37.0 \%$ |
| Lung \& Bronchus | $12.1 \%$ | Colorectal | $9.9 \%$ |
| Colorectal | $10.9 \%$ | Lung \& Bronchus | $9.4 \%$ |

Information on the type of cancer cell or cancer cell morphology for specific anatomical sites may be obtained from Cancer Registry records. For example, reference may be made to both squamous and small cell carcinoma of the lung. The distinction of cell type is important for the pathologist as it provides important information related to treatment and prognosis. To the epidemiologist, however, the distinction aids in separating cancer of a specific site into different diseases and etiologies. Differentiation also allows the epidemiologist to compare the distribution, or relative frequency, of cancer cell types among cases in the study population to that of the comparison population. Comparing distributions is yet another way to search for similar or differing patterns of disease within the study population that might suggest a unique causative or associated factor.

A substantial body of scientific and medical information is recorded in the epidemiological literature describing the relationship between cancer, population incidence, and the known or potentially associated risk factors. To add greater perspective to the present analysis of cancer incidence in the northeast Denver area, the epidemiological literature is summarized here for those cancers found to be statistically significantly elevated.

Comparison to Prior Analysis, 1979-1996 - In the prior Colorado Department of Public Health and Environment analysis of cancer incidence in Areas 1-3, for the period 1979-1996, significantly higher than expected numbers of diagnoses were reported for certain anatomical sites and gender (CDPHE, 2003). These findings are summarized in Table 3. Most of these elevations are not observed during the 1997-2000 period, as described in this report. For some types of cancer, kidney (Areas 1a and 1b), hypo- and oropharynx (Area 1b), larynx (Area 1b), stomach (Area 1 Combined), and salivary gland (Area 3), zero or only one additional case was reported during 1997-2000. For lung (Area 2), larynx (Area 2), cervix (Area 2), and multiple
myeloma (Area 3), three or more additional cases were diagnosed during 1997-2000, but did not exceed the expected statistical variation.

Table 3. Summary of statistically significantly elevated cancer incidence in the northeast Denver metropolitan area, by study area, time period, gender, and anatomical site for the period 1979-1996 (CDPHE, 2003).

| Area | Time Period | Gender | Cancer Site |
| :--- | :--- | :--- | :--- |
| 1a | $1979-1988$ | Males | Kidney |
| 1b | $1979-1996$ | Males | Other Pharynx |
| 1b | $1989-1996$ | Males | Larynx |
| 1b | $1989-1996$ | Female | Kidney |
| 1 Combined | $1979-1996$ | Males | Bladder |
| 1 Combined | $1979-1996$ | Males | Stomach |
| 1 \& 2 | $1979-1996$ | Males \& Females | Lung |
| 2 | $1979-1988$ | Males \& Females | Larynx |
| 2 | $1979-1996$ | Females | Cervix |
| 2 | $1989-1996$ | Males \& Females | Leukemia |
| 3 | $1989-1996$ | Females | Salivary Gland |
| 3 | $1979-1988$ | Females | Multiple Myeloma |

In the earlier department analysis for the 1989-1996 period, leukemia in Area 2 was reported to be elevated in the 0-4 and 5-9 age groups, with the 5-9 age group ratios being statistically high. Pooling the two age groups into a 0-9 age category also showed a statistically high ratio. In the current analysis for 1997-2000, however, leukemias are within expected statistical variation and there were no new childhood leukemias reported in Area 2 over the 1997-2000 time period.

Bladder cancer was significantly elevated in Area 1 Combined males during the 1979-1996 period. The number of new bladder cancer diagnoses reported during 1997-2000 was within expected statistical variation. However, this number was 1 case less than that necessary for statistical significance. This finding is discussed below (see Bladder Cancer).

Lung cancer in Area 1, males and females, continues to be statistically elevated during 19972000. This finding is also discussed below (see Lung Cancer).

Summary of Current Analysis, 1997-2000 - A limited variety of cancers were found to be statistically elevated within the study areas of the northeast Denver metropolitan area during 1997-2000. This information is summarized in Table 4 and discussed in detail in the following paragraphs.

All Cancers Combined - The distribution of reported cancers among the three most common anatomical sites was reviewed and found to be generally consistent with the expected distribution (Table 2, above), based on a Colorado statewide comparison.

In Area 1a, 1b and Area 1 Combined, departures from expected case counts of all cancers combined were all within expected statistical variation and largely influenced by the elevated number of lung and pancreatic cancer diagnoses. Bladder cancer also was elevated in Area 1 Combined, though not significantly. The number of all diagnosed cancers combined in Area 2 was similar to the number expected; however, there was a significantly elevated finding for lymphoma in males. In Area 3, the number of all cancers combined was also similar to the number expected in this area during the study time period. The one notable finding in Area 3 was a statistically significant elevation of malignant brain cancer. Each of these observations is discussed below and a summary of statistically significantly elevated cancers is presented in Table 4. The specific cancer sites observed to be statistically significantly low during 1997-2000 were prostate in males in both Area 1b and 2, and melanoma in males and females in Area 2.

| Table 4. Summary of statistically significantly elevated cancer incidence in the Northeast Denver Metropolitan Area, by study area, gender, and anatomical site for the period 1997-2000. |  |  |  |
| :---: | :---: | :---: | :---: |
| Area | Gender | Cancer Site | Appendix Table Number |
| 1a \& 1 Combined | Males \& Females | Pancreas | A1, A7, A16 |
| 1a, 1b \& 1 Combined | Males \& Females | Lung | A1, A2, A4-A9, A17 |
| 2 | Males | Lymphoma | A11, A20 |
| 3 | Males \& Females | Malignant Brain | A13, A15, A21, A22 |

Gender and Race/Ethnicity - An elevation of a particular cancer in one gender, but not the other, tends to argue for a causative or co-factor not present among the entire population. Conversely, an elevation in both genders may suggest shared risk factors.

As described in the methods section of this report, this evaluation entailed comparing cancer diagnoses to expected cancer counts based on age group, gender, and race/ethnicity. The distribution of the number of diagnosed cancer cases was expressly reviewed where there were at least eight cancers and significant elevations were observed by gender. Anatomical sites, or types, of cancer for which the race/ethnicity distribution was reviewed for one or both genders were pancreas, lung, bladder, leukemia, lymphoma, and brain. Statistically significant elevations were not observed among all racial/ethnic segments of the population. In each of these cases, statistically significant elevations were limited to White, non-Hispanic cases. This difference may be attributable to the size of each population segment within the study areas. Additionally, and as with differences in rates of diagnosis between genders, an elevation of a particular cancer in one race/ethnicity, but not the other, tends to argue for a causative or co-factor not present among the entire population. And again similar to gender differences, an elevation in more than one race/ethnic group may suggest shared risk factors.

Bladder Cancer - Bladder cancer was very close to being statistically elevated in Area 1 Combined during the period 1997-2000. The 2.15 ratio for males and the 1.84 ratio for both genders combined were each within one case of being statistically significant. Most cases were observed in persons aged 55 years and older. Higher ratios of bladder cancer were also reported for this area for the 1979-1996 time period (CDPHE, 2003).

Smoking is a primary risk factor for bladder cancer accounting for as many as 60 percent of all cases. Cancer Registry case abstracts showed that 71 percent of the individual cases diagnosed in Area 1 had a smoking history.

One-fourth of bladder cancer cases in the United States are estimated to be associated with occupational exposures. Associated occupations that have been reported in the literature include those in the rubber, textile, leather, paint, chemical and petroleum industries. Other risk factors for bladder cancer include arsenic exposure, chronic bladder infections and other diseases of the urinary tract. Men are two to three times more likely than women to get bladder cancer and people with family members who have bladder cancer are more likely to develop the disease.

A bladder cancer elevation in Area 1 males was also described in the 1993 report Cancer Incidence in the Northeastern Denver Metro Area: Report of the Ad Hoc Panel (CDPHE, 1993). The significant elevation was observed for the period 1981-1985; the ratio for a longer time period, 1979-1988 was elevated, but not significantly. Subsequent to the 1993 report, a casecontrol study of bladder cancer in Adams County, Colorado, was conducted by Colorado State University in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR 1996). The study examined all known male and female cases for the period 1982 through part of 1991. The case-control study found that for these cases, a history of bladder infection and smoking were significant risk factors, as has been demonstrated in the literature. The ability of the study to detect other risk factors was, however, limited by the small number of cases and controls who could be located for interview.

Lung Cancer - Area 1 showed statistically elevated incidence of lung cancer among males and females during 1997-2000. Most cases were among persons aged 55 years and older. Among cases for which information about smoking was recorded, 93 percent had a history of smoking. There were no uncommon histological cell types recorded and the distribution of types of lung cancer in Area 1 during 1997-2000 was similar to the Denver metropolitan area. The cases included the major forms of lung cancer; squamous cell carcinomas, large cell carcinomas, small cell carcinomas and adenocarcinomas.

The lung cancer risk associated with tobacco is discussed in greater detail above. Cigarette smoking specifically, however, is by far the most important risk factor for the development of lung cancer (American Cancer Society, 1995). For example, a woman smoking 1 to 20 cigarettes per day has a greater than 10 -fold increase in risk of developing lung cancer than a woman that has never smoked.

Other known environmental risk factors for lung cancer include exposure to asbestos, arsenic, radon, and other forms of air pollution. Other air pollutants that may be carcinogenic to the lung are diesel exhaust, pitch and tar, dioxin, chromium, cadmium and nickel compounds.

Pancreatic Cancer - The number of pancreatic cancer cases diagnosed among males and females in the Combined Area 1 during the period 1997-2000 was statistically significantly elevated (Table A7). Each gender individually was within expected statistical variation (Table A8 and A9). Table A16 shows that almost all of these pancreatic cancers, seven out of eight cases, were among White, non-Hispanic persons and that among White, nonHispanic cases only, the statistical elevation remains significant. Seven of the eight cases were also among the 55 years and older age groups. The eight cases were distributed throughout Area 1 and not grouped in any one census track. Cancer Registry abstracts show a variety of occupations among these eight cases. Thirty-eight percent of the cases had a documented history of smoking, and including only abstracts where smoking information was recorded, 50 percent of cases were smokers. Four of eight cases or 50 percent had a history of alcohol usage, and including only abstracts where alcohol information was recorded, 67 percent of cases had used alcohol. The distribution of histological cell types among these eight cases was similar to the distribution found in the Denver metropolitan area. Adenocarcinomas accounted for 50 percent of cases in Area 1 Combined compared to 69 percent of Denver cases, other carcinomas accounted for 25 percent of cases in Area 1 compared to 11 percent of Denver cases, and the remainder were coded to a general malignant neoplasm category with 25 percent of Area 1 cases compared to 20 percent of Denver cases.

During 1997-2000, pancreatic cancer was slightly higher and lower than expected in Areas 2 and 3 respectively, but in both areas the differences were within expected statistical variation. In the prior evaluation for the time period 1979-1996, the numbers of diagnoses of pancreatic cancer was within expected statistical variation (CDPHE, 2003).

The epidemiological literature indicates that the incidence of pancreatic cancer increases with age; most people being diagnosed between 60 to 80 years. The findings in the Combined Area 1 are consistent with this general observation. The literature also reports that smoking is estimated to account for about 30 percent of pancreatic cancer cases, with another $8-10$ percent attributed to hereditary genetic predisposition (Konner and O’Reilly, 2002; Ghadirian et al, 2002.) Additionally, smoking is the strongest risk factor in familial pancreatic cancer, particularly among males and those under age 50 (Rulyak et al., 2003). As discussed above, a history of smoking may have contributed to 30-50 percent of the cases diagnosed in the Combined Area 1. Coffee consumption has not been associated with pancreatic cancer and an association with alcohol consumption has been inconsistent (Michaud et al., 2001).

Generally, men are at higher risk for pancreatic cancer, though in recent years the incidence gap between genders appears to be shrinking, possibly due to increased cigarette smoking among women. Some studies, however, have suggested that reproductive factors, particularly childbearing, may reduce pancreatic cancer in women (Skinner et al., 2003).

Other risk factors may include a history of diabetes, chronic pancreatitis, and colonization by the bacterium Helicobacter pylori. A variety of dietary factors have been associated with pancreatic cancer, particularly meat intake and the manner in which it is cooked. Well-done barbecued and pan-fried meats typically contain high levels of carcinogenic byproducts (Risch, 2003; Anderson et al., 2002). Certain occupations have also been suggested as increasing the risk of pancreatic cancer, including coal gas, metal, leather-tanning and textile workers. Others include chemist and those with chronic exposed to DDT (Konner and O’Reilly, 2002).

Lymphoma - Lymphomas are a variety of cancers originating in the lymphatic system, a component of the immune system. Lymphomas are subdivided into two types, Hodgkin's and non-Hodgkin's. While Hodgkin's lymphoma, also known as Hodgkin's Disease, is a single form of cancer with a distinctive cell type, accounting for about 14 percent of lymphoma in the general population, non-Hodgkin's lymphoma (NHL) is a broader group of diseases arising from B- or T-lymphocytes (ACS, 2001). For the present analysis, the category lymphoma includes all lymphomas.

Table A11 shows that there was a statistically significant elevation in the number of male lymphomas diagnosed in Area 2 during 1997-2000. Table A20 shows, however, that none of the ratios for the race/ethnic categories or specific age groups was statistically elevated. A majority of the lymphoma cases diagnosed in Area 2 were among persons 45 years and older, and all were distributed throughout the area census tracks. Of the 14 cases diagnosed in Area 2, two were Hodgkin's disease and 12 were non-Hodgkin's lymphoma. Of the 12 non-Hodgkin's lymphoma cases, 50 percent were B-cell tumors vs. 46 percent in the Denver metropolitan area; eight percent were T-cell tumors vs. six percent in the metropolitan area; and 42 percent were other or unknown vs. 48 percent in the metropolitan area.

During the previous time period evaluated for Area 2, 1989-1996 (CDPHE, 2003), there were fewer cases of lymphoma diagnosed than expected with seven cases diagnosed compared to about 13 cases expected. This reduced count of diagnosed cases may be related to the higher than expected number diagnosed during the 1997-2000 period. The number of male lymphomas over the longer time period of 1989-2000 was equal to the expected number with 21 cases compared to about 21 cases expected, for a ratio of 1.01 , which is within expected statistical variation.

Also noteworthy is the absence of any lymphoma diagnoses in the Combined Area 1 during the period 1997-2000, when about seven cases were expected. This observation again demonstrates the impact of redefining temporal and spatial boundaries. In Area 3 during the same time period, the number of lymphoma diagnoses was only slightly lower than expected.

The causes of lymphomas are in most cases unknown, but appear to be linked to lymphocyte hyperactivity induced by chronic antigenic stimulation (Romagnanai et al., 1985). Infection by agents, particularly Epstein-Barr virus (EBV), has been suggested as a risk factor for Hodgkin's disease. Patients with the human immunodeficiency virus (HIV) or who have undergone bone marrow transplantation are at greater risk for developing the disease (Rowlings et al., 1999).

Numerous investigations of environmental factors, such as occupation, have been reported. Of
the industries evaluated, woodworking showed the most consistent link with an increased risk of Hodgkin's disease, but not all studies showed a positive association. Certain chemicals, chlorophenols and pesticides, have been reported as possible risk factors for Hodgkin's disease, but these finding have been inconsistent and ambiguous (McCunney, 1999). Agricultural workers have a slightly elevated risk of developing Hodgkin's disease. Exposures common among those in this occupational group include infectious microorganisms, pesticides, fuels and lubricants. Despite extensive epidemiological investigation, however, no specific etiologic exposure has been identified. Furthermore, female farmers, when evaluated separately, do not show an association between the general farming occupation and Hodgkin's disease, suggesting that activities related to farming specific to males, or other unidentified factors may play the important etiologic role (Khuder et al., 1999).

Non-Hodgkin's lymphoma is the fifth and sixth most common forms of cancer in men and women, respectively, and its measured incidence increased significantly in recent decades, particularly among the elderly. Risk factors for non-Hodgkin's lymphoma may include treatment with immunosuppressive agents or the presence of an immune disorder. Agricultural workers appear to be at greater risk for non-Hodgkin's lymphoma (Pickle et al., 1987; Pearce et al., 1985). This risk may be associated with a variety of occupational exposures including infectious agents such as zoonotic viruses (Pearce and Bethwaite, 1992). Chemical agents may also play a role, though observations are equivocal. Chemicals that have been suggested as potential risk factors include pesticides, fertilizers fuels and solvents (Wigle et al., 1990). A significant association with occupations dealing in metals and metal products has also been reported (Blair et al., 1993).

As with gastrointestinal cancers, the bacterium Helicobacter pylori has been identified as a potential risk factor for gastric non-Hodgkin’s lymphoma (Parsonnet et al., 1994). Early evidence that exposure to hair dye among women or their children may increase the risk of nonHodgkin's lymphoma has not been supported in the current literature (Holly et al, 1998; Holly et al., 2002). Non-Hodgkin's lymphoma has not been associated with tobacco or alcohol use.

Brain and Central Nervous System Cancer - In Area 3, malignant brain and central nervous system cancers had a statistically high diagnosed to expected ratio during 19972000. Tables A14 and A15 show that the elevation was also seen in males and females separately, with the female ratio being significantly high. Table A21 shows that of the 12 cases diagnosed, most were among the White, non-Hispanic group and the Black subgroup, though both of these ratios were within expected statistical variation. The same table shows that only the 45-54 age group had a statistically high ratio with six cases compared to about one or two expected.

The Colorado Central Cancer Registry abstracts showed a variety of occupations among the 12 cases. The frequency of cell types was consistent with the distribution within the Denver metropolitan area.

Brain cancer was neither statistically significantly high nor low in the other two study areas, 1 Combined and 2, during the period 1997-2000. Additionally, no statistically significant findings were observed related to the number of malignant brain cancers in Areas 1-3 during prior years of evaluation, 1979-1996 (CDPHE, 2003). The malignant brain and other central nervous system cancers reported in Area 3 among persons 25 years and older were dispersed among the census tracts within the area, showing no unexpected grouping.

The terms benign and malignant do not strictly apply to most central nervous system tumors. Benign central nervous system tumors frequently recur due to infiltration of normal tissue. Benign tumors may also differentiate into biologically more aggressive tumors. Additionally, brain tumors seldom metastasize to other organs of the body and are therefore not truly biologically malignant (ACS, 2001). Regardless of these characteristics, all brain tumors are potentially life threatening due to the restricted volume of the brain cavity and crowding of adjacent brain tissue. For this reason the Colorado Central Cancer Registry records both benign and malignant central nervous system tumors. The incidence of both is evaluated for this report.

Table A22 was prepared to evaluate the effect of combining malignant and benign brain tumors in Area 3 for the 1997-2000 time period. Combining malignant and benign tumors resulted in a ratio of 1.17, which is within expected statistical variation. Ratios for the race/ethnic groups were not elevated, but an elevation seen in the 45-54 age group persisted largely due to the number of malignant brain cancers (see Table A21).

Brain tumors are more common in males than females, except meningiomas, a central nervous system cancer arising from the tissue surrounding the brain, which are more common in females. Brain tumors also are more common among whites than people of other races, and among the elderly. Gliomas, tumors in the supporting tissue of the brain, are more common among persons with family members previously diagnosed with this disease.

A wide variety of risk factors have been investigated, including injuries and physical, chemical, and infectious agents. Only three factors, however, have been firmly identified, ionizing radiation; certain congenital and genetic disorders; and immunosuppression. The cause of a majority of brain cancer is unknown.

Considerable research into electromagnetic radiation exposure as a risk factor for brain cancer has not produced definitive results (Inskep et al., 1997; Gurney and van Wijngaarden, 1999).

Chronic exposure to a variety of chemical agents may increase the risk of brain cancer, but none have been confirmed. These exposures are most likely to occur to a significant degree in occupational settings and include synthetic rubber and polyvinyl chloride manufacturing, and the petroleum, petrochemical and agricultural industries (Legler et al., 1999). Agricultural factors may include both chemical, such as pesticides and fuels, and zoonotic agents (Holly et al., 1998). The associations of childhood brain cancer with parental occupation in the chemical industry and with residential pesticide exposure have also been studied, but with conflicting results (McKeanCowdin et al., 1998; Davis et al., 1993).

An association has not been reported between brain cancer and personal use of tobacco and
alcohol (Legler et al, 1999). Furthermore, no associations have been reported between prenatal or postnatal exposure to tobacco smoke and pediatric brain cancer, nor to maternal exposure prior to pregnancy (Filippini et al., 2002). Nitrosamides and nitrosamines, recognized as neurocarcinogens in experimental animal studies, are potentially significant risk factors for human brain cancer. Associations have been inconsistent, but exposure may occur through a variety of commercial products and dietary sources (Inskip et al. 1997). Exposure to infectious agents and head injuries has also been investigated as potential risk factors but without definitive results (Wrensch et al., 2001).

Multiple Comparisons - Studies examining multiple health outcomes in several subpopulations may observe statistically elevated rates of those outcomes simply due to chance. This statistical phenomenon is commonly referred to as the "multiple comparisons" problem. If these tests are conducted at a 95 percent confidence level, about 5 percent of the tests are predicted to be statistically significant by chance alone; about 2.5 percent may be statistically higher than expected; and 2.5 percent lower. In this study of cancer in the northeast Denver area for the period 1997-2000, with 200 independent statistical tests conducted on separate cancer sites by gender for several different areas, there were five ratios statistically higher than expected, 2.5 percent of the tests compared to about 2.5 percent predicted by chance alone. Three ratios were statistically lower than expected, with 1.5 percent of the tests compared to about 2.5 percent predicted by chance alone. Including all cancers combined, both genders combined for all cancers and cancers of individual anatomical sites, and additional tests done by race/ethnicity and age for several cancers, a total of 507 comparisons made. Of these comparisons, 21 ratios or 4.1 percent of the tests were statistically higher than expected and five ratios or 1 percent of the tests were statistically lower than expected. This outcome does not suggest an overall marked departure from that predicted. Ratios based on less than three cases were not tested statistically due to the inherent instability of small numbers. Not testing these low case count ratios likely accounts for some of the lower percentage of statistically lower than expected tests found.

## Conclusions

Similar to the finding of the Colorado Department of Public Health and Environment's prior report on cancer incidence in the northeast Denver metropolitan area for the period 1979-1996 (CDPHE, 2003), no generalized elevation of cancer was observed during the years 1997-2000. Elevations of cancer that were observed in the current analysis were again of specific anatomical sites, however, with differences from the earlier evaluation.

The year 1997 was selected as the starting point for the present analysis to coincide with the initiation of the Rocky Mountain Arsenal soil remediation. For two reasons, however, cancer cases diagnosed during the 1997-2000 period are unlikely to be related to soil remediation activities at the arsenal. These are: (1) air monitoring at the arsenal has not shown an ongoing or significant off-site release of arsenal-related contaminants, and (2) the process of cancer development and the associated disease latency suggest that if cancers were initiated during the 1997-2000 time period, diagnoses are not expected until a later time period.

In the prior Colorado Department of Public Health and Environment analysis of cancer incidence in the study areas for the period 1979-1996, significantly higher than expected numbers of diagnoses were reported for certain anatomical sites and gender (CDPHE, 2003). Most of these elevations are not observed during the 1997-2000 period.

The patterns of cancer incidence in the study area populations during 1997-2000, such as the dominant forms of cancer, cell type distribution and age distribution, appears generally consistent with the trends in the comparison population and those described in the epidemiological literature.

The specific cancer sites observed to be statistically significantly elevated during 1997-2000 were lung cancer in both males and females in Areas 1a, 1b and 1 Combined; pancreatic cancer in males and females in Areas 1a and 1 Combined; lymphoma in Area 2 males; and malignant brain cancer in males and females in Area 3. The cases contributing to each of these elevations were dispersed among the several census tracts of the respective areas, showing no unexpected groupings. The specific cancer sites observed to be statistically significantly low during 19972000 were prostate in males in Area 1b and 2, and melanoma in males and females in Area 2.

Smoking histories were previously reported as possibly playing a significant role in many cancers diagnosed during 1979-1996. This again appears likely for some cases reported in 19972000. Lung and pancreatic cancer are both smoking-related and Cancer Registry abstracts indicate that a significant number of the reported cases had a history of smoking. Other factors, such as exposure to carcinogens in the occupational, indoor and ambient air may also contribute to the overall individual and population risk. Genetic predisposition and infectious agents too are potential factors that have been identified in the epidemiological literature.

Bladder cancer continues a trend first reported in earlier Colorado Department of Public Health and Environment and Colorado State University analyses, being elevated in Area 1 Combined during the period 1997-2000, though not achieving statistical significance. As in the earlier report, there is a high frequency of smoking histories among the cases. And similar to lung and pancreatic cancer, smoking is an important risk factor for bladder cancer, accounting for as many as 60 percent of all cases.

The elevation of lymphomas in Area 2 may be primarily attributable to the short period of observation, 1997-2000. Extending this period of observation back several years, when there was unexpectedly low lymphoma incidence, would result in very close agreement between the number of diagnosed and expected cases. The selection of geographic boundaries also may account for the apparent lymphoma elevation in Area 2 since, in all of Area 1 Combined, there were no cases diagnosed over the 1997-2000 time period when several were expected. The causes of lymphomas are largely unknown. However, lymphocyte hyperactivity appears to be a significant factor. Infectious agents, chemical exposures and or certain occupations have all been proposed as potential stimulants or acting as carcinogens through some other process.

The current scientific understanding of brain and CNS cancer and associated risk factors limits further analysis of the significant elevation of malignant brain cancer in Area 3. The incidence of benign brain tumors does not show a similar excess of cases nor pattern with respect to age at
diagnosis. An elevation of malignant brain cancer was not observed in the earlier time periods of 1979-1996 in Area 3 and is not seen in Areas 1 or 2 during 1997-2000. The significant Area 3 elevation of malignant brain cancers in the 45-54 age group appears inconsistent with the typical case distribution in the general population in that brain and central nervous system cancer is most common among older age groups. This difference may, however, be an artifact of the small population of Area 3, relative to the comparison population, in combination with random chance. A wide variety of brain cancer risk factors have been proposed in the epidemiological literature, including chemical, physical, and infectious agents. None of these factors have been confirmed with the exception of ionizing radiation, congenital and genetic disorders and immunosupression.

For the four-year time period evaluated in this report, 1997-2000, case counts are too small to assess differences or similarities among racial and ethnic subgroups of the Area 1-3 populations. In the evaluation of 1979-1996 data, discussed in an earlier Colorado Department of Public Health and Environment report, statistically significant elevations of certain cancers were limited to the White, non-Hispanic subgroup. Similar analysis of data collected from 1997 onward will be possible once several more years of case reporting are complete.

Lastly, some or all of the statistically significant findings observed during the 1997-2000 period may be chance occurrences. Among the 200 independent statistical tests performed, the number of elevated findings was equal to the number expected due to chance alone. The number of statistically significantly low findings was fewer than would be expected due to chance. Ratios based on less than three cases, however, were not statistically tested and this may account for the smaller number of significantly low findings.

## RECOMMENDATIONS

Cancer surveillance is one of the community health activities conducted by the Rocky Mountain Arsenal Medical Monitoring Program, which is based at the Colorado Department of Public Health and Environment and was undertaken in response to recommendations made by the Rocky Mountain Arsenal Medical Monitoring Advisory Group. This report focuses on the period 1997-2000, following the initiation of the arsenal soil remediation activities. Comparison is also made to the prior period of analysis, 1979-1996. The department will continue general monitoring of the cancer incidence patterns in the northeast Denver metropolitan area and follow the incidence of those types of cancer identified as elevated prior to 2001.

The findings of this report should be communicated to Denver Health, the Denver Environmental Health Department and the Tri-County Health Department to assist these agencies in characterizing cancer incidence and the presence of known and potential risk factors in their respective jurisdictions.

The findings of this report should also be communicated to the Comprehensive Cancer Control Section at the Department of Public Health and Environment to improve cancer control strategies in the northeast Denver metropolitan area.

## References

Colorado Department of Public Health and Environment. Rocky Mountain Arsenal Medical Monitoring Program. Analysis of Diagnosed vs. Expected Cancer Cases for the Northeast Denver Metropolitan Area in the Vicinity of the Rocky Mountain Arsenal, 1979-1996. 2003.

Bailar JC, Ederer F. Significance Factors for the Ratio of a Poisson Variable to its Expectation. Biometrics 20(3):639-43, 1964.

American Cancer Society's Textbook of Clinical Oncology. Lenhard Jr RE, Jr, Osteen RT, Gansler T (eds.), Amer Cancer Soc, 2001.

Wynder EL. The Past, Present, and Future of the Prevention of Lung Cancer. Cancer Epidemiol Biomarkers Prev. 7:735-48, 1998.

Doll R, Peto R, Wheatley K, et al. Mortality in Relation to Smoking: 40 years' Observations of Male British Doctors. Br Med J 309:901-11, 1994.

Morabia A, Bernstein M, Heritier S, Khatchatrian N. Relation of Breast Cancer with Passive and Active Exposure to Tobacco Smoke. Am J Epidemiol 143:918-28, 1996.

Lash TL, Aschengrau A. Active and Passive Cigarette Smoking and the Occurrence of Breast Cancer. Am J Epidemiol 149:5-12, 1999.

Konner J, O’Reilly, E. Pancreatic Cancer; Epidemiology, Genetics, and Approaches to Screening. Oncology 16(12):1615-22, 2002

Ghadirian P, Liu G, Gallinger S, Schmocker B, et al. Risk of Pancreatic Cancer Among Individual with a Family History of Cancer of the Pancreas. Intl J Cancer 97(6):807-10, 2002.

Rulyak SJ, Lowenfels, AB, Maisonneuve P, and Brentnall, TA. Risk Factors for the Development of Pancreatic Cancer in Familial Pancreatic Cancer Kindreds. Gastroenterology 124(5):1292-9, 2003.

Michaud DS, Giovannucci E, Willett WC, et al. Coffee and Alcohol Consumption and the Risk of Pancreatic Cancer in Two Prospective United States Cohorts. Cancer Epidemiol, Biomark Prev 10(5):429-37, 2001.

Skinner HG, Michaud DS, Colditz GA, Giovannucci EL, et al. Parity, Reproductive Factors, and the Risk of Pancreatic Cancer in Women. Cancer Epidemiol, Biomark Prev 12(5):433-8, 2003.

Risch HA. Etiology of Pancreatic Cancer, with a Hypothesis Concerning the Role of N-Nitroso Compounds and Excess Gastric Acidity. J Natl Cancer Inst. 95(13):948-60, 2003.

Anderson KE, Sinha R, Kulldorff M, Gross M, et al. Meat Intake and Cooking Techniques: Associations with Pancreatic Cancer. Mut Res 506-507:225-31, 2002.

American Cancer Society Textbook of Clinical Oncology. Murphy GP, Lawrence Jr W, Lenhard RE (eds). 2nd ed., 1995.

Colorado Department of Public Health and Environment. Cancer Incidence in the Northeastern Denver Metro Area: Report of the Ad Hoc Panel. 1993.

Agency for Toxic Substances and Disease Registry. Reproductive, Neurobehavioral, and Other Disorders in Communities Surrounding the Rocky Mountain Arsenal. Colorado State University/Agency for Toxic Substance and Disease Registry. 1996.

Romagnani S, Ferrini PLS, Ricci M. The Immune System Derangement in Hodgkin’s Disease. Semin Hematol 22:41-55, 1985.

Rowlings PA, Curtis RE, Passweg JR, Deeg HJ, et al. Increased Incidence of Hodgkin’s Disease After Allogeneic Bone Marrow Transplantation. J Clin Oncol 17(10):3122-7, 1999.

McCunney RJ. Hodgkin’s Disease, Work, and the Environment. J Occup Environ Med 41(1):36-46, 1999.

Khuder SA, Mutgi AB, Schaub EA, Tano BDK. Meta-Analysis of Hodgkin’s Disease Among Farmers. Scand J Environ Health 25(5):436-41, 1999.

Pickle, LW, Mason TJ, Howard N, et al. Atlas of U.S. Cancer Mortality Among Whites: 19501980. DHHAS Pub No (NIH) 87-2900. U.S. DHHS. 1987.

Pearce NE, Smith AH, and Fisher DO. Malignant Lymphoma and Multiple Myeloma Linked with Agricultural Occupations in a New Zealand Cancer Registry-based Study. Am J Epidemiol. 121:225-37, 1985.

Pearce N and Bethwaite P. Increasing Incidence of Non-Hodgkin’s Lymphoma: Occupational and Environmental Factors. Cancer Res (Suppl) 52:5496s-5500s, 1992.

Wigle DT, Semenciw RM, Wilkins K, et al. Mortality Study of Canadian Farm Operators: NonHodgkin’s Lymphoma Mortality and Agricultural Practices in Saskatchewan. J Natl Cancer Inst 82:575-82, 1990.

Blair A, Linos A, Stewart PA, Burmeister LF, et al. Evaluation of Risks for Non-Hodgkin's Lymphoma by Occupation and Industry Exposures from a Case-control Study. Am J Indust Med 23:301-12, 1993.

Parsonnet J, Hanson S, Rodriguez L, Gelb A, et al. Helicobacter pylori Infection and Gastric Lymphoma. N Engl J Med 330(18):1267-71, 1994.

Holly EA, Lele C, Bracci PM. Hair-color Products and Risk for Non-Hodgkin’s Lymphoma: A Population-based Study in the San Francisco Bay Area. Am J Public Health 88(12):1767-73, 1998.

Holly EA, Bracci PM, Hong MK, Mueller BA, Preston-Martin S. West Coast Study of Childhood Brain Tumours and Maternal use of Hair-colouring Products. Paedoatr Perinat Epidemiol 16(3):226-35, 2002.

Inskip PD, Linet MS, Heineman EF. Etiology of Brain Tumors in Adults. Epidemiol Rev 17(2):382-414, 1995.

Gurney JG, van Wijngaarden E. Extremely Low Frequency Electromagnetic Fields (EMF) and Brain Cancer in Adults and Children: Review and Comment. Neuro-oncol 1(3):212-20, 1999.

Legler J, Reis L, Smith M, Warren J, et al. Brain and Other Central Nervous System Cancers: Recent Trends in Incidence and Mortality. J Natl Can Inst, 91(16):1382-90, 1999.

Holly EA, Bracci PM, Nueller, BA, and Preston-Martin S. Farm Animal Exposures and Pediatric Brain Tumors: Results from the United States West Coast Childhood Brain Tumor Study. Can Epidemiol Biomark Prev 7:797-802, 1998.

McKean-Cowdin, R, Preston-Martin S, Pogoda, JM, Holly EA, Mueller, BA, Davis RL. Parental Occupation and Childhood Brain Tumors: Astroglial and Primitive Neuroectodermal Tumors. J Occup Environ Med 40(4):332-40, 1998.

Davis JR, Brownson RC, Garcia R, Bentz BJ, Turner A. Family Pesticide Use and Childhood Brain Cancer. Arch Environ Contam Toxicol 24:87-92, 1993.

Filippini G, Maisonneuve P, McCreie M, Peris-Bonet R, et al. Relation of Childhood Brain Tumors to Exposure of Parents and Children to Tobacco Smoke: The SEARCH International Case-control Study. Surveillance of Environmental Aspect Related to Cancer in Humans. Intl J Can 100(2):206-13, 2002.

Wrensch M, Weinberg A, Wiencke J, Miike R, et al. Prevalence of Antibodies to Four Herpesvirsuses among Adults with Glioma and Controls. Am J Epidemiol 154(2):161-5, 2001.

Gurney JG, Preston-Martin S, McDaniel AM, Mueller, BA, and Holly, EA. Head Injury as a Risk Factor for Brain Tumors in Children: Results from a Multicenter Case-control Study. Epidemiol 7(5):485-9, 1996.

Wrensch M, Miike R, Lee M, Neuhaus J. Are Prior Head Injuries or Diagnostic X-rays Associated with Glioma in Adults? The Effects of Control Selection Bias. Neuroepidemiology 19(5):234-44, 2000.

## Appendix

# Analysis of Diagnosed vs. Expected Cancer Cases for the Northeast Denver Area in the Vicinity of the Rocky Mountain Arsenal, 1997-2000 

Data Tables

Tables A1- A15 display the number of diagnosed cancers in each of the study areas (Area 1a, 1b, 1,2 , and 3 ) by cancer type and gender for 1997-2000 compared to the number that would be expected based on the population of male and female residents in the areas by race/ethnicity and age. Tables A16-A22 display additional detail for selected areas, gender groups and/or cancer types that had statistically high findings or particularly relevant findings to compare to previous time periods.

| Table A1 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1a, 1997-2000 - Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 41 | 29.763 | 1.38 | (0.99-1.87) |
| Salivary Gland | 1 | 0.077 | 12.99 | NC |
| Oral | 2 | 0.283 | 7.07 | NC |
| Nasopharynx | 0 | 0.026 | 0.00 | NC |
| Other Pharynx | 0 | 0.178 | 0.00 | NC |
| Esophagus | 0 | 0.277 | 0.00 | NC |
| Stomach | 1 | 0.391 | 2.56 | NC |
| Small Intestine | 0 | 0.097 | 0.00 | NC |
| Colorectal | 2 | 2.851 | 0.70 | NC |
| Liver | 0 | 0.278 | 0.00 | NC |
| Other Biliary | 1 | 0.113 | 8.85 | NC |
| Pancreas | 3 | 0.604 | $4.97{ }^{*}$ | (1.02-14.52) |
| Larynx | 0 | 0.241 | 0.00 | NC |
| Lung | 10 | 2.973 | $3.36{ }^{* *}$ | (1.62-6.18) |
| Melanoma | 2 | 1.878 | 1.07 | NC |
| Bladder | 3 | 1.124 | 2.67 | (0.55-7.80) |
| Kidney | 0 | 0.691 | 0.00 | NC |
| Thyroid | 0 | 0.521 | 0.00 | NC |
| Other Endocrine | 0 | 0.044 | 0.00 | NC |
| Brain | 1 | 0.519 | 1.93 | NC |
| Bone | 0 | 0.073 | 0.00 | NC |
| Leukemia | 1 | 0.789 | 1.27 | NC |
| Mult. Myeloma | 0 | 0.290 | 0.00 | NC |
| Lymphoma | 0 | 1.374 | 0.00 | NC |
| Soft Tissue | 0 | 0.225 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.

* Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A2 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1a, 1997-2000 - Males |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 23 | 15.238 | 1.51 | (0.96-2.27) |
| Salivary Gland | 1 | 0.042 | 23.81 | NC |
| Oral | 1 | 0.190 | 5.26 | NC |
| Nasopharynx | 0 | 0.017 | 0.00 | NC |
| Other Pharynx | 0 | 0.147 | 0.00 | NC |
| Esophagus | 0 | 0.216 | 0.00 | NC |
| Stomach | 1 | 0.265 | 3.77 | NC |
| Small Intestine | 0 | 0.068 | 0.00 | NC |
| Colorectal | 0 | 1.596 | 0.00 | NC |
| Liver | 0 | 0.195 | 0.00 | NC |
| Other Biliary | 1 | 0.053 | 18.87 | NC |
| Pancreas | 2 | 0.334 | 5.99 | NC |
| Larynx | 0 | 0.192 | 0.00 | NC |
| Lung | 6 | 1.751 | $3.43{ }^{*}$ | (1.26-7.47) |
| Melanoma | 1 | 1.091 | 0.92 | NC |
| Prostate | 4 | 4.505 | 0.89 | (0.24-2.27) |
| Testis | 0 | 0.289 | 0.00 | NC |
| Bladder | 2 | 0.862 | 2.32 | NC |
| Kidney | 0 | 0.467 | 0.00 | NC |
| Thyroid | 0 | 0.155 | 0.00 | NC |
| Other Endocrine | 0 | 0.027 | 0.00 | NC |
| Brain | 1 | 0.318 | 3.14 | NC |
| Bone | 0 | 0.044 | 0.00 | NC |
| Leukemia | 1 | 0.469 | 2.13 | NC |
| Mult. Myeloma | 0 | 0.181 | 0.00 | NC |
| Lymphoma | 0 | 0.806 | 0.00 | NC |
| Soft Tissue | 0 | 0.129 | 0.00 | NC |

[^1]| Table A3 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1a, 1997-2000 - Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 18 | 14.525 | 1.24 | (0.73-1.96) |
| Salivary Gland | 0 | 0.035 | 0.00 | NC |
| Oral | 1 | 0.093 | 10.75 | NC |
| Nasopharynx | 0 | 0.009 | 0.00 | NC |
| Other Pharynx | 0 | 0.031 | 0.00 | NC |
| Esophagus | 0 | 0.061 | 0.00 | NC |
| Stomach | 0 | 0.126 | 0.00 | NC |
| Small Intestine | 0 | 0.029 | 0.00 | NC |
| Colorectal | 2 | 1.256 | 1.59 | NC |
| Liver | 0 | 0.083 | 0.00 | NC |
| Other Biliary | 0 | 0.060 | 0.00 | NC |
| Pancreas | 1 | 0.270 | 3.70 | NC |
| Larynx | 0 | 0.049 | 0.00 | NC |
| Lung | 4 | 1.222 | 3.27 | (0.89-8.37) |
| Melanoma | 1 | 0.787 | 1.27 | NC |
| Female Breast | 4 | 5.917 | 0.68 | (0.18-1.73) |
| Cervix | 1 | 0.291 | 3.44 | NC |
| Uterus | 1 | 0.620 | 1.61 | NC |
| Ovary | 1 | 0.588 | 0.00 | NC |
| Bladder | 1 | 0.262 | 3.82 | NC |
| Kidney | 0 | 0.224 | 0.00 | NC |
| Thyroid | 0 | 0.366 | 0.00 | NC |
| Other Endocrine | 0 | 0.017 | 0.00 | NC |
| Brain | 0 | 0.201 | 0.00 | NC |
| Bone | 0 | 0.029 | 0.00 | NC |
| Leukemia | 0 | 0.320 | 0.00 | NC |
| Mult. Myeloma | 0 | 0.109 | 0.00 | NC |
| Lymphoma | 0 | 0.568 | 0.00 | NC |
| Soft Tissue | 0 | 0.096 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.

* Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level) NC=not calculated (see text)

| Table A4 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1b, 1997-2000 - Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers Diagnosed | Cancers Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 120 | 116.883 | 1.03 | (0.85-1.23) |
| Salivary Gland | 0 | 0.323 | 0.00 | NC |
| Oral | 1 | 1.057 | 0.95 | NC |
| Nasopharynx | 0 | 0.102 | 0.00 | NC |
| Other Pharynx | 1 | 0.696 | 1.44 | NC |
| Esophagus | 1 | 1.134 | 0.88 | NC |
| Stomach | 2 | 1.798 | 1.11 | NC |
| Small Intestine | 0 | 0.440 | 0.00 | NC |
| Colorectal | 19 | 12.408 | 1.53 | (0.92-2.39) |
| Liver | 3 | 1.439 | 2.08 | (0.43-6.10) |
| Other Biliary | 1 | 0.573 | 1.75 | NC |
| Pancreas | 5 | 2.733 | 1.83 | (0.59-4.28) |
| Larynx | 0 | 1.015 | 0.00 | NC |
| Lung | 24 | 12.337 | $1.95{ }^{* *}$ | (1.25-2.89) |
| Melanoma | 4 | 5.920 | 0.68 | (0.18-1.73) |
| Bladder | 7 | 4.304 | 1.63 | (0.65-3.35) |
| Kidney | 1 | 2.970 | 0.34 | NC |
| Thyroid | 1 | 1.753 | 0.57 | NC |
| Other Endocrine | 0 | 0.175 | 0.00 | NC |
| Brain | 4 | 1.912 | 2.09 | (0.57-5.35) |
| Bone | 1 | 0.345 | 2.90 | NC |
| Leukemia | 7 | 3.048 | 2.30 | (0.92-4.74) |
| Mult. Myeloma | 2 | 1.311 | 1.53 | NC |
| Lymphoma | 0 | 5.644 | 0.00 | NC |
| Soft Tissue | 0 | 0.905 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.

* Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A5 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1b, 1997-2000 - Males |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers Diagnosed | Cancers Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 61 | 60.457 | 1.01 | (0.83-1.38) |
| Salivary Gland | 0 | 0.194 | 0.00 | NC |
| Oral | 0 | 0.712 | 0.00 | NC |
| Nasopharynx | 0 | 0.071 | 0.00 | NC |
| Other Pharynx | 1 | 0.564 | 1.77 | NC |
| Esophagus | 1 | 0.862 | 1.16 | NC |
| Stomach | 0 | 1.190 | 0.00 | NC |
| Small Intestine | 0 | 0.285 | 0.00 | NC |
| Colorectal | 10 | 6.840 | 1.46 | (0.70-2.69) |
| Liver | 2 | 1.016 | 1.97 | NC |
| Other Biliary | 0 | 0.256 | 0.00 | NC |
| Pancreas | 2 | 1.481 | 1.35 | NC |
| Larynx | 0 | 0.799 | 0.00 | NC |
| Lung | 14 | 7.147 | 1.96* | (1.07-3.29) |
| Melanoma | 2 | 3.503 | 0.57 | NC |
| Prostate | 9 | 17.364 | 0.52* | (0.24-0.98) |
| Testis | 0 | 1.010 | 0.00 | NC |
| Bladder | 7 | 3.325 | 2.11 | (0.85-4.34) |
| Kidney | 1 | 1.907 | 0.52* | NC |
| Thyroid | 1 | 0.516 | 1.94 | NC |
| Other Endocrine | 0 | 0.106 | 0.00 | NC |
| Brain | 2 | 1.139 | 1.76 | NC |
| Bone | 0 | 0.208 | 0.00 | NC |
| Leukemia | 3 | 1.869 | 1.61 | (0.33-4.69) |
| Mult. Myeloma | 1 | 0.799 | 1.25 | NC |
| Lymphoma | 0 | 3.310 | 0.00 | NC |
| Soft Tissue | 0 | 0.513 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A6 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1b, 1997-2000 - Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 59 | 56.426 | 1.05 | (0.83-1.43) |
| Salivary Gland | 0 | 0.129 | 0.00 | NC |
| Oral | 1 | 0.345 | 2.90 | NC |
| Nasopharynx | 0 | 0.031 | 0.00 | NC |
| Other Pharynx | 0 | 0.132 | 0.00 | NC |
| Esophagus | 0 | 0.272 | 0.00 | NC |
| Stomach | 2 | 0.608 | 3.29 | NC |
| Small Intestine | 0 | 0.155 | 0.00 | NC |
| Colorectal | 9 | 5.568 | 1.62 | (0.74-3.07) |
| Liver | 1 | 0.423 | 2.36 | NC |
| Other Biliary | 1 | 0.317 | 3.15 | NC |
| Pancreas | 3 | 1.252 | 2.40 | (0.49-7.01) |
| Larynx | 0 | 0.216 | 0.00 | NC |
| Lung | 10 | 5.190 | 1.93* | (0.93-3.54) |
| Melanoma | 2 | 2.417 | 0.83 | NC |
| Female Breast | 13 | 21.702 | 0.60 | (0.32-1.02) |
| Cervix | 1 | 1.262 | 0.79 | NC |
| Uterus | 2 | 2.338 | 0.86 | NC |
| Ovary | 1 | 2.187 | 0.46 | NC |
| Bladder | 0 | 0.979 | 0.00 | NC |
| Kidney | 0 | 1.063 | 0.00 | NC |
| Thyroid | 0 | 1.237 | 0.00 | NC |
| Other Endocrine | 0 | 0.069 | 0.00 | NC |
| Brain | 2 | 0.773 | 2.59 | NC |
| Bone | 1 | 0.137 | 7.30 | NC |
| Leukemia | 4 | 1.179 | 3.39 | (0.92-8.68) |
| Mult. Myeloma | 1 | 0.512 | 1.95 | NC |
| Lymphoma | 0 | 2.334 | 0.00 | NC |
| Soft Tissue | 0 | 0.392 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level) NC=not calculated (see text)

| Table A7 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1 Combined, 1997-2000 - Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers Expected | Diagnosed / Expected | 95\% C.I. for <br> Ratio |
| All Cancers | 161 | 146.647 | 1.10 | (0.93-1.26) |
| Salivary Gland | 1 | 0.400 | 2.50 | NC |
| Oral | 3 | 1.340 | 2.24 | (0.46-6.55) |
| Nasopharynx | 0 | 0.127 | 0.00 | NC |
| Other Pharynx | 1 | 0.876 | 1.14 | NC |
| Esophagus | 1 | 1.411 | 0.71 | NC |
| Stomach | 3 | 2.188 | 1.37 | (0.28-4.01) |
| Small Intestine | 0 | 0.537 | 0.00 | NC |
| Colorectal | 21 | 15.259 | 1.38 | (0.85-2.10) |
| Liver | 3 | 1.716 | 1.75 | (0.36-5.11) |
| Other Biliary | 2 | 0.686 | 2.92 | NC |
| Pancreas | 8 | 3.339 | 2.40* | (1.03-4.72) |
| Larynx | 0 | 1.256 | 0.00 | NC |
| Lung | 34 | 15.310 | $2.22{ }^{* *}$ | (1.53-3.11) |
| Melanoma | 6 | 7.797 | 0.77 | (0.28-1.68) |
| Bladder | 10 | 5.427 | 1.84 | (0.89-3.39) |
| Kidney | 1 | 3.661 | 0.27 | NC |
| Thyroid | 1 | 2.274 | 0.44 | NC |
| Other Endocrine | 0 | 0.218 | 0.00 | NC |
| Brain | 5 | 2.430 | 2.06 | (0.67-4.81) |
| Bone | 1 | 0.418 | 2.39 | NC |
| Leukemia | 8 | 3.837 | 2.09 | (0.90-4.10) |
| Mult. Myeloma | 2 | 1.601 | 1.25 | NC |
| Lymphoma | 0 | 7.018 | 0.00 | NC |
| Soft Tissue | 0 | 1.131 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A8 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1 Combined, 1997-2000 - Males |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 84 | 75.696 | 1.11 | (0.87-1.35) |
| Salivary Gland | 1 | 0.235 | 4.26 | NC |
| Oral | 1 | 0.902 | 1.11 | NC |
| Nasopharynx | 0 | 0.087 | 0.00 | NC |
| Other Pharynx | 1 | 0.712 | 1.40 | NC |
| Esophagus | 1 | 1.078 | 0.93 | NC |
| Stomach | 1 | 1.454 | 0.69 | NC |
| Small Intestine | 0 | 0.353 | 0.00 | NC |
| Colorectal | 10 | 8.436 | 1.19 | (0.57-2.18) |
| Liver | 2 | 1.211 | 1.65 | NC |
| Other Biliary | 1 | 0.309 | 3.24 | NC |
| Pancreas | 4 | 1.816 | 2.20 | (0.60-5.63) |
| Larynx | 0 | 0.991 | 0.00 | NC |
| Lung | 20 | 8.898 | $2.25 * *$ | (1.37-3.47) |
| Melanoma | 3 | 4.594 | 0.65 | (0.13-1.91) |
| Prostate | 13 | 21.869 | 0.59 | (0.32-1.02) |
| Testis | 0 | 1.298 | 0.00 | NC |
| Bladder | 9 | 4.187 | 2.15 | (0.99-4.08) |
| Kidney | 1 | 2.374 | 0.42 | NC |
| Thyroid | 1 | 0.670 | 1.49 | NC |
| Other Endocrine | 0 | 0.133 | 0.00 | NC |
| Brain | 3 | 1.456 | 2.06 | (0.42-6.02) |
| Bone | 0 | 0.252 | 0.00 | NC |
| Leukemia | 4 | 2.338 | 1.71 | (0.47-4.38) |
| Mult. Myeloma | 1 | 0.980 | 1.02 | NC |
| Lymphoma | 0 | 4.116 | 0.00 | NC |
| Soft Tissue | 0 | 0.643 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A9 - Number of Cancer Diagnoses Compared to the Expected Number in Area 1 Combined, 1997-2000 - Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 77 | 70.951 | 1.09 | (0.86-1.37) |
| Salivary Gland | 0 | 0.165 | 0.00 | NC |
| Oral | 2 | 0.438 | 4.57 | NC |
| Nasopharynx | 0 | 0.040 | 0.00 | NC |
| Other Pharynx | 0 | 0.164 | 0.00 | NC |
| Esophagus | 0 | 0.333 | 0.00 | NC |
| Stomach | 2 | 0.734 | 2.72 | NC |
| Small Intestine | 0 | 0.184 | 0.00 | NC |
| Colorectal | 11 | 6.823 | 1.61 | (0.81-2.88) |
| Liver | 1 | 0.505 | 1.98 | NC |
| Other Biliary | 1 | 0.377 | 2.65 | NC |
| Pancreas | 4 | 1.523 | 2.63 | (0.72-6.72) |
| Larynx | 0 | 0.265 | 0.00 | NC |
| Lung | 14 | 6.412 | 2.18* | (1.19-3.66) |
| Melanoma | 3 | 3.203 | 0.94 | (0.19-2.74) |
| Female Breast | 17 | 27.618 | 0.62* | (0.36-0.99) |
| Cervix | 2 | 1.553 | 1.29 | NC |
| Uterus | 3 | 2.958 | 1.01 | (0.21-2.96) |
| Ovary | 1 | 2.775 | 0.36 | NC |
| Bladder | 1 | 1.240 | 0.81 | NC |
| Kidney | 0 | 1.287 | 0.00 | NC |
| Thyroid | 0 | 1.604 | 0.00 | NC |
| Other Endocrine | 0 | 0.085 | 0.00 | NC |
| Brain | 2 | 0.974 | 2.05 | NC |
| Bone | 1 | 0.166 | 6.02 | NC |
| Leukemia | 4 | 1.499 | 2.67 | (0.73-6.82) |
| Mult. Myeloma | 1 | 0.621 | 1.61 | NC |
| Lymphoma | 0 | 2.902 | 0.00 | NC |
| Soft Tissue | 0 | 0.488 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.

* Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level) NC=not calculated (see text)

| Table A10 - Number of Cancer Diagnoses Compared to the Expected Number in Area 2, 1997-2000 - Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers Diagnosed | Cancers Expected | Diagnosed / Expected | 95\% C.I. for <br> Ratio |
| All Cancers | 268 | 268.879 | 1.00 | (0.96-1.23) |
| Salivary Gland | 0 | 0.753 | 0.00 | NC |
| Oral | 3 | 2.400 | 1.25 | (0.26-3.65) |
| Nasopharynx | 0 | 0.226 | 0.00 | NC |
| Other Pharynx | 2 | 1.576 | 1.27 | NC |
| Esophagus | 2 | 2.618 | 0.76 | NC |
| Stomach | 8 | 4.281 | 1.87 | (0.81-3.68) |
| Small Intestine | 2 | 1.033 | 1.94 | NC |
| Colorectal | 34 | 29.581 | 1.15 | (0.79-1.61) |
| Liver | 5 | 3.390 | 1.47 | (0.48-3.45) |
| Other Biliary | 1 | 1.425 | 0.70 | NC |
| Pancreas | 10 | 6.583 | 1.52 | (0.73-2.79) |
| Larynx | 4 | 2.338 | 1.71 | (0.47-4.38) |
| Lung | 32 | 28.906 | 1.11 | (0.76-1.56) |
| Melanoma | 5 | 12.732 | 0.39* | (0.13-0.92) |
| Bladder | 9 | 9.877 | 0.91 | (0.42-1.73) |
| Kidney | 10 | 6.869 | 1.46 | (0.70-2.68) |
| Thyroid | 4 | 3.798 | 1.05 | (0.29-2.69) |
| Other Endocrine | 0 | 0.400 | 0.00 | NC |
| Brain | 4 | 4.269 | 0.94 | (0.26-2.40) |
| Bone | 0 | 0.802 | 0.00 | NC |
| Leukemia | 10 | 7.051 | 1.42 | (0.68-2.61) |
| Mult. Myeloma | 2 | 3.152 | 0.63 | NC |
| Lymphoma | 20 | 13.114 | 1.53 | (0.93-2.36) |
| Soft Tissue | 2 | 2.097 | 0.95 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.

* Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

|  | Cancers Diagnosed | Cancers Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| :---: | :---: | :---: | :---: | :---: |
| All Cancers | 142 | 136.210 | 1.04 | (0.92-1.27) |
| Salivary Gland | 0 | 0.449 | 0.00 | NC |
| Oral | 2 | 1.595 | 1.25 | NC |
| Nasopharynx | 0 | 0.154 | 0.00 | NC |
| Other Pharynx | 2 | 1.256 | 1.59 | NC |
| Esophagus | 2 | 1.954 | 1.02 | NC |
| Stomach | 4 | 2.735 | 1.46 | (0.40-3.74) |
| Small Intestine | 2 | 0.639 | 3.13 | NC |
| Colorectal | 20 | 15.736 | 1.27 | (0.78-1.96) |
| Liver | 4 | 2.332 | 1.72 | (0.47-4.39) |
| Other Biliary | 0 | 0.598 | 0.00 | NC |
| Pancreas | 7 | 3.433 | 2.04 | (0.82-4.20) |
| Larynx | 3 | 1.814 | 1.65 | (0.34-4.84) |
| Lung | 18 | 16.274 | 1.11 | (0.65-1.75) |
| Melanoma | 3 | 7.489 | 0.40 | (0.08-1.17) |
| Prostate | 23 | 38.709 | 0.59* | (0.38-0.89) |
| Testis | 1 | 2.171 | 0.46 | NC |
| Bladder | 8 | 7.528 | 1.06 | (0.46-2.09) |
| Kidney | 4 | 4.280 | 0.93 | (0.25-2.39) |
| Thyroid | 0 | 1.085 | 0.00 | NC |
| Other Endocrine | 0 | 0.237 | 0.00 | NC |
| Brain | 3 | 2.478 | 1.21 | (0.25-3.54) |
| Bone | 0 | 0.494 | 0.00 | NC |
| Leukemia | 7 | 4.265 | 1.64 | (0.66-3.38) |
| Mult. Myeloma | 0 | 1.855 | 0.00 | NC |
| Lymphoma | 14 | 7.505 | $1.87{ }^{*}$ | (1.02-3.13) |
| Soft Tissue | 1 | 1.156 | 0.87 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A12 - Number of Cancer Diagnoses Compared to the Expected Number in Area 2, 1997-2000 - Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 126 | 132.669 | 0.95 | (0.91-1.29) |
| Salivary Gland | 0 | 0.304 | 0.00 | NC |
| Oral | 1 | 0.805 | 1.24 | NC |
| Nasopharynx | 0 | 0.072 | 0.00 | NC |
| Other Pharynx | 0 | 0.320 | 0.00 | NC |
| Esophagus | 0 | 0.664 | 0.00 | NC |
| Stomach | 4 | 1.546 | 2.59 | (0.70-6.62) |
| Small Intestine | 0 | 0.394 | 0.00 | NC |
| Colorectal | 14 | 13.845 | 1.01 | (0.55-1.70) |
| Liver | 1 | 1.058 | 0.95 | NC |
| Other Biliary | 1 | 0.827 | 1.21 | NC |
| Pancreas | 3 | 3.150 | 0.95 | (0.20-2.78) |
| Larynx | 1 | 0.524 | 1.91 | NC |
| Lung | 14 | 12.632 | 1.11 | (0.61-1.86) |
| Melanoma | 2 | 5.243 | 0.38 | NC |
| Female Breast | 40 | 49.597 | 0.81 | (0.58-1.10) |
| Cervix | 4 | 2.954 | 1.35 | (0.37-3.46) |
| Uterus | 8 | 5.430 | 1.47 | (0.63-2.90) |
| Ovary | 2 | 5.031 | 0.40 | NC |
| Bladder | 1 | 2.349 | 0.43 | NC |
| Kidney | 6 | 2.589 | 2.32 | (0.85-5.05) |
| Thyroid | 4 | 2.713 | 1.47 | (0.40-3.77) |
| Other Endocrine | 0 | 0.163 | 0.00 | NC |
| Brain | 1 | 1.791 | 0.56 | NC |
| Bone | 0 | 0.308 | 0.00 | NC |
| Leukemia | 3 | 2.786 | 1.08 | (0.22-3.15) |
| Mult. Myeloma | 2 | 1.297 | 1.54 | NC |
| Lymphoma | 6 | 5.609 | 1.07 | (0.39-2.33) |
| Soft Tissue | 1 | 0.941 | 1.06 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level) NC=not calculated (see text)

| Table A13 - Number of Cancer Diagnoses Compared to the Expected Number in Area 3, 1997-2000 - Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed <br> / Expected | 95\% C.I. for Ratio |
| All Cancers | 318 | 313.537 | 1.01 | (0.97-1.21) |
| Salivary Gland | 0 | 1.056 | 0.00 | NC |
| Oral | 2 | 2.724 | 0.73 | NC |
| Nasopharynx | 2 | 0.353 | 5.67 | NC |
| Other Pharynx | 0 | 2.574 | 0.00 | NC |
| Esophagus | 3 | 3.305 | 0.91 | (0.19-2.65) |
| Stomach | 6 | 5.477 | 1.10 | (0.40-2.39) |
| Small Intestine | 1 | 1.392 | 0.72 | NC |
| Colorectal | 32 | 32.225 | 0.99 | (0.68-1.40) |
| Liver | 3 | 5.737 | 0.52 | (0.11-1.53) |
| Other Biliary | 1 | 2.167 | 0.46 | NC |
| Pancreas | 5 | 7.149 | 0.70 | (0.23-1.63) |
| Larynx | 1 | 3.274 | 0.31 | NC |
| Lung | 37 | 33.050 | 1.12 | (0.79-1.54) |
| Melanoma | 2 | 10.083 | 0.20 | NC |
| Bladder | 12 | 7.037 | 1.71 | (0.88-2.98) |
| Kidney | 8 | 8.317 | 0.96 | (0.41-1.89) |
| Thyroid | 6 | 6.192 | 0.97 | (0.35-2.11) |
| Other Endocrine | 0 | 1.076 | 0.00 | NC |
| Brain | 12 | 5.248 | $2.29 *$ | (1.18-3.99) |
| Bone | 0 | 1.477 | 0.00 | NC |
| Leukemia | 9 | 8.539 | 1.05 | (0.48-2.00) |
| Mult. Myeloma | 4 | 5.426 | 0.74 | (0.20-1.88) |
| Lymphoma | 13 | 15.118 | 0.86 | (0.46-1.47) |
| Soft Tissue | 2 | 3.493 | 0.57 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A14 - Number of Cancer Diagnoses Compared to the Expected Number in Area 3, 1997-2000 - Males |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 160 | 165.073 | 0.97 | (0.82-1.13) |
| Salivary Gland | 0 | 0.337 | 0.00 | NC |
| Oral | 0 | 2.203 | 0.00 | NC |
| Nasopharynx | 2 | 0.275 | 7.27 | NC |
| Other Pharynx | 0 | 2.122 | 0.00 | NC |
| Esophagus | 3 | 2.485 | 1.21 | (0.25-3.53) |
| Stomach | 5 | 4.181 | 1.20 | (0.39-2.79) |
| Small Intestine | 1 | 0.700 | 1.43 | NC |
| Colorectal | 17 | 18.138 | 0.94 | (0.54-1.50) |
| Liver | 3 | 3.946 | 0.76 | (0.16-2.22) |
| Other Biliary | 0 | 1.116 | 0.00 | NC |
| Pancreas | 3 | 4.435 | 0.68 | (0.14-1.98) |
| Larynx | 1 | 2.626 | 0.38 | NC |
| Lung | 21 | 19.545 | 1.07 | (0.66-1.64) |
| Melanoma | 1 | 5.424 | 0.18 | NC |
| Prostate | 54 | 46.826 | 1.15 | (0.82-1.42) |
| Testis | 4 | 3.463 | 1.16 | (0.31-2.95) |
| Bladder | 10 | 5.474 | 1.83 | (0.88-3.36) |
| Kidney | 5 | 5.485 | 0.91 | (0.30-2.13) |
| Thyroid | 3 | 1.832 | 1.64 | (0.34-4.79) |
| Other Endocrine | 0 | 0.641 | 0.00 | NC |
| Brain | 6 | 3.296 | 1.82 | (0.67-3.97) |
| Bone | 0 | 1.158 | 0.00 | NC |
| Leukemia | 5 | 5.325 | 0.94 | (0.30-2.19) |
| Mult. Myeloma | 1 | 3.378 | 0.30 | NC |
| Lymphoma | 8 | 9.552 | 0.84 | (0.36-1.65) |
| Soft Tissue | 1 | 1.599 | 0.63 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A15 - Number of Cancer Diagnoses Compared to the Expected Number in Area 3, 1997-2000 - Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Cancers <br> Diagnosed | Cancers <br> Expected | Diagnosed / Expected | 95\% C.I. for Ratio |
| All Cancers | 158 | 148.464 | 1.06 | (0.93-1.29) |
| Salivary Gland | 0 | 0.719 | 0.00 | NC |
| Oral | 2 | 0.521 | 3.84 | NC |
| Nasopharynx | 0 | 0.078 | 0.00 | NC |
| Other Pharynx | 0 | 0.452 | 0.00 | NC |
| Esophagus | 0 | 0.820 | 0.00 | NC |
| Stomach | 1 | 1.296 | 0.77 | NC |
| Small Intestine | 0 | 0.692 | 0.00 | NC |
| Colorectal | 15 | 14.087 | 1.06 | (0.59-1.76) |
| Liver | 0 | 1.791 | 0.00 | NC |
| Other Biliary | 1 | 1.051 | 0.95 | NC |
| Pancreas | 2 | 2.714 | 0.74 | NC |
| Larynx | 0 | 0.648 | 0.00 | NC |
| Lung | 16 | 13.505 | 1.18 | (0.68-1.92) |
| Melanoma | 1 | 4.659 | 0.21 | NC |
| Female Breast | 57 | 57.471 | 0.99 | (0.82-1.43) |
| Cervix | 8 | 5.324 | 1.50 | (0.65-2.96) |
| Uterus | 7 | 5.055 | 1.38 | (0.56-2.86) |
| Ovary | 9 | 4.571 | 1.97 | (0.90-3.74) |
| Bladder | 2 | 1.563 | 1.28 | NC |
| Kidney | 3 | 2.832 | 1.06 | (0.22-3.10) |
| Thyroid | 3 | 4.360 | 0.69 | (0.14-2.01) |
| Other Endocrine | 0 | 0.435 | 0.00 | NC |
| Brain | 6 | 1.952 | $3.07{ }^{*}$ | (1.13-6.70) |
| Bone | 0 | 0.319 | 0.00 | NC |
| Leukemia | 4 | 3.214 | 1.24 | (0.34-3.18) |
| Mult. Myeloma | 3 | 2.048 | 1.46 | (0.30-4.28) |
| Lymphoma | 5 | 5.566 | 0.90 | (0.29-2.10) |
| Soft Tissue | 1 | 1.894 | 0.53 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level) NC=not calculated (see text)

| Table A16 - Number of Pancreas Diagnoses by Race/Ethnicity and <br> by Age Compared to the Expected Number in Area 1, 1997-2000 - <br> Males and Females |  |  |  |  |
| :--- | :---: | :---: | :---: | :---: |
| Race/ <br> Ethnicity | Cancers <br> Diagnosed | Cancers <br> Expected | Ratio of <br> Diagnosed <br> to Expected | 95\% C.I. <br> for Ratio |
| White Non- <br> Hispanic | 7 | 2.311 | $3.03^{*}$ | $(1.22-6.25)$ |
| Hispanic | 1 | 0.932 | 1.07 | NC |
| Black | 0 | 0.035 | 0.00 | NC |
| Age | 1 | 0.123 | 8.13 | NC |
| $35-44$ | 0 | 0.361 | 0.00 | NC |
| $45-54$ | 2 | 0.677 | 2.95 | NC |
| $55-64$ | 3 | 1.133 | 2.65 | $(0.23-7.74)$ |
| $65-74$ | 2 | 1.021 | 1.96 | NC |
| $75+$ | 8 | 3.339 | $2.40^{*}$ | $(1.03-4.72)$ |
| Total |  |  |  |  |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.

* Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A17 - Number of Lung Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 1, 19972000 - Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
| Race/ Ethnicity | Cancers <br> Diagnosed | Cancers Expected | Ratio of Diagnosed to Expected | 95\% C.I. for Ratio |
| White NonHispanic | 30 | 11.930 | $2.52{ }^{* *}$ | (1.70-3.59) |
| Hispanic | 4 | 2.812 | 1.42 | (0.39-3.64) |
| Black | 0 | 0.179 | 0.00 | NC |
| Age |  |  |  |  |
| 55-64 | 11 | 3.479 | $3.16{ }^{* *}$ | (1.58-5.66) |
| 65-74 | 16 | 6.223 | $2.57{ }^{* *}$ | (1.47-4.17) |
| 75+ | 7 | 4.073 | 1.72 | (0.69-3.54) |
| Total | 34 | 15.310 | $2.22{ }^{* *}$ | (1.53-3.11) |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
${ }^{*}$ Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A18 - Number of Bladder Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 1, 1997-2000 - Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
| Race/ Ethnicity | Cancers Diagnosed | Cancers Expected | Ratio of Diagnosed to Expected | 95\% C.I. for Ratio |
| White NonHispanic | 10 | 4.675 | $2.14{ }^{*}$ | (1.03-3.93) |
| Hispanic | 0 | 0.698 | 0.00 | NC |
| Black | 0 | 0.023 | 0.00 | NC |
| Age |  |  |  |  |
| 55-64 | 1 | 1.205 | 0.83 | NC |
| 65-74 | 8 | 1.848 | $4.33{ }^{* *}$ | (1.87-8.52) |
| 75+ | 1 | 1.490 | 0.67 | NC |
| Total | 10 | 5.427 | 1.84 | (0.89-3.39) |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
${ }^{*}$ Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A19 - Number of Leukemia Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 2, 1997-2000 Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
| Race/ Ethnicity | Cancers Diagnosed | Cancers <br> Expected | Ratio of <br> Diagnosed <br> to <br> Expected | 95\% C.I. for Ratio |
| White NonHispanic | 9 | 5.078 | 1.77 | (0.81-3.36) |
| Hispanic | 1 | 1.744 | 0.57 | NC |
| Black | 0 | 0.129 | 0.00 | NC |
| Age |  |  |  |  |
| 0-4 | 0 | 0.374 | 0.00 | NC |
| 5-9 | 0 | 0.088 | 0.00 | NC |
| 10-14 | 0 | 0.181 | 0.00 | NC |
| 15-19 | 0 | 0.169 | 0.00 | NC |
| 20-24 | 0 | 0.057 | 0.00 | NC |
| 25-34 | 0 | 0.335 | 0.00 | NC |
| 35-44 | 1 | 0.560 | 1.79 | NC |
| 45-54 | 0 | 0.632 | 0.00 | NC |
| 55-64 | 3 | 0.710 | 4.23 | (0.87-12.35) |
| 65-74 | 2 | 1.693 | 1.18 | NC |
| 75+ | 4 | 2.249 | 1.78 | (0.48-4.55) |
| Total | 10 | 7.051 | 1.42 | (0.68-2.61) |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A20 - Number of Lymphomas by Race/Ethnicity and by Age <br> Compared to the Expected Number in Area 2, 1997-2000 - Males |  |  |  |  |
| :--- | :---: | :---: | :---: | :---: |
| Race/ <br> Ethnicity | Cancers <br> Diagnosed | Cancers <br> Expected | Ratio of <br> Diagnosed <br> to Expected | 95\% C.I. <br> for Ratio |
| White Non- <br> Hispanic | 8 | 4.369 | 1.83 | $(0.79-3.60)$ |
| Hispanic | 4 | 2.939 | 1.36 | $(0.37-3.48)$ |
| Black | 1 | 0.104 | 9.62 | NC |
| Age | 0 | 0.005 | 0.00 | NC |
| $0-4$ | 2 | 0.172 | 11.63 | NC |
| $5-9$ | 0 | 0.038 | 0.00 | NC |
| $10-14$ | 0 | 0.260 | 0.00 | NC |
| $15-19$ | 1 | 0.504 | 1.98 | NC |
| $20-24$ | 1 | 0.883 | 1.13 | NC |
| $25-34$ | 2 | 0.877 | 2.28 | NC |
| $35-44$ | 2 | 1.209 | 0.83 | NC |
| $45-54$ | 2 | 1.533 | 1.31 | NC |
| $55-64$ | 4 | 1.883 | 2.12 | $(0.58-5.43)$ |
| $65-74$ | 14 | 7.505 | $1.87^{*}$ | $(1.02-3.13)$ |
| $75+$ | Total | 0 | 0.00 | NC |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

Table A21 - Number of Malignant Brain and Central Nervous System Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 3, 1997-2000 - Males and Females

| Race/ Ethnicity | Cancers Diagnosed | Cancers Expected | Ratio of Diagnosed to Expected | $\begin{aligned} & \text { 95\% C.I. } \\ & \text { for Ratio } \end{aligned}$ |
| :---: | :---: | :---: | :---: | :---: |
| White NonHispanic | 4 | 1.916 | 2.09 | (0.57-5.34) |
| Hispanic | 2 | 1.363 | 1.47 | NC |
| Black | 5 | 1.712 | 2.92 | (0.95-6.82) |
| Age |  |  |  |  |
| 0-4 | 1 | 0.157 | 6.37 | NC |
| 5-9 | 1 | 0.118 | 8.48 | NC |
| 10-14 | 0 | 0.138 | 0.00 | NC |
| 15-19 | 0 | 0.198 | 0.00 | NC |
| 20-24 | 0 | 0.107 | 0.00 | NC |
| 25-34 | 1 | 0.816 | 1.23 | NC |
| 35-44 | 1 | 0.895 | 1.12 | NC |
| 45-54 | 6 | 1.348 | $4.45{ }^{* *}$ | (1.63-9.70) |
| 55-64 | 1 | 0.790 | 1.27 | NC |
| 65-74 | 1 | 0.327 | 3.06 | NC |
| 75+ | 0 | 0.352 | 0.00 | NC |
| Total | 12 | 5.248 | $2.29{ }^{*}$ | (1.18-3.99) |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.

* Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

| Table A22 - Number of Malignant and Benign Brain and Central Nervous System Cancer/Tumor Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 3, 1997-2000 Males and Females |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
| Race/ Ethnicity | Cancers Diagnosed | Cancers <br> Expected | Ratio of Diagnosed to Expected | 95\% C.I. for Ratio |
| White NonHispanic | 4 | 4.726 | 0.85 | (0.23-2.16) |
| Hispanic | 4 | 2.968 | 1.35 | (0.37-3.45) |
| Black | 7 | 5.259 | 1.33 | (0.53-2.74) |
| Age |  |  |  |  |
| 0-4 | 1 | 0.251 | 3.98 | NC |
| 5-9 | 1 | 0.176 | 5.68 | NC |
| 10-14 | 0 | 0.353 | 0.00 | NC |
| 15-19 | 0 | 0.301 | 0.00 | NC |
| 20-24 | 0 | 0.203 | 0.00 | NC |
| 25-34 | 1 | 1.829 | 0.55 | NC |
| 35-44 | 2 | 3.015 | 0.66 | NC |
| 45-54 | 8 | 3.011 | $2.66{ }^{*}$ | (1.15-5.23) |
| 55-64 | 1 | 2.128 | 0.47 | NC |
| 65-74 | 2 | 1.227 | 1.63 | NC |
| 75+ | 0 | 1.182 | 0.00 | NC |
| Total | 16 | 13.676 | 1.17 | (0.67-1.90) |

Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.
*Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
$\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).


[^0]:    1 The Rocky Mountain Arsenal On-Post Record of Decision called for the creation the Medical Monitoring Program. The Record of Decision was signed by the U.S. Army, the U.S. Environmental Protection Agency (EPA) and the Colorado Department of Public Health and Environment on June 11, 1996, with concurrence of the U.S. Fish and Wildlife Service and Shell Oil Company. The U.S. Army, serving as the lead agency, and Shell Oil Company implement the Record of Decision, which includes 31 restoration projects for contaminated soil, structures and ground water. Federal, state and local public health agencies conduct regulatory oversight.
    2 The Record of Decision stipulated that a Medical Monitoring Advisory Group be formed to evaluate information concerning exposure pathways and to identify and recommend appropriate public health actions and to communicate this information to the community. The advisory group recommendations defined goals, objectives and the methods of a program designed to respond effectively to arsenal-related health concerns of the community. The Record of Decision directed that the advisory group include representatives from the affected communities, regulatory agencies, local governments, U.S. Army, Shell Oil Company, U.S. Fish and Wildlife Service, and independent technical advisors. The Record of Decision stated that the primary goals of the Medical Monitoring Program are to monitor any off-post impact on human health due to the remediation and provide mechanisms for evaluation of human health on an individual and community basis, until such time as the soil remedy is completed.
    3 The cumulative lifetime risk of cancer in Colorado is 1 in 2 for males and 1 in 3 for females.
    4 The development of cancer, or carcinogenesis, is believed to be a multistage process involving replication of damaged DNA, reduced control of cell division and function, and transformation into a malignant tumor. 5 Latency is the period between the causative event and the diagnosis of the disease. Cancer latency may last a few years to 30 years or more.

[^1]:    Note: Diagnosed/Expected ratios that have a 95\% Confidence Interval that brackets the value 1.00 are not considered statistically high or low.

    * Ratio is statistically significant at $\mathrm{p}=0.05$ level. ( ${ }^{* *} \mathrm{p}=0.01$ level)
    $\mathrm{NC}=$ not calculated due to less than 3 diagnoses (see text for explanation).

