ROCKY MOUTAIN ARSENAL MEDICAL MONITORING PROGRAM

UPDATE OF CANCER INCIDENCE IN NORTHEAST DENVER RESIDENTS LIVING IN THE VICINITY OF THE ROCKY MOUNTAIN ARSENAL, 1997-2005

Prepared by:
Colorado Department of Public Health and Environment

May 2010

Colorado Department of Public Health and Environment

For More Information:

Jane Mitchell
Disease Control and Environmental Epidemiology Division
Environmental Epidemiology Section
Colorado Department of Public Health and Environment
4300 Cherry Creek Drive South
Denver, Colorado 80246
303-692-2644
jane.mitchell@state.co.us

E-Report available at:
http//www.cdphe.state.co.us/rma/index.htm

TABLE OF CONTENTS

UPDATE OF CANCER INCIDENCE IN NORTHEAST DENVER RESIDENTS LIVING IN THE VICINITY OF THE ROCKY MOUNTAIN ARSENAL, 1997-2005

INTRODUCTION

This document reports findings of cancer surveillance for 1997-2005 for communities in the northeast Denver metropolitan area, surrounding the Rocky Mountain Arsenal (RMA) in southern Adams County, Colorado. 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 Group[2].

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 18,000 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 process[4], involving both external (chemicals, radiation, and viruses) and internal factors (hormone levels, 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

1 The RMA Medical Monitoring Program was created by the RMA On-Post Record of Decision (ROD), and was signed by the U.S. Army, the U.S. Environmental Protection Agency (EPA), and the CDPHE on June 11, 1996, with concurrence of the U.S. Fish and Wildlife Service and Shell Oil Company.

2 The ROD stipulated that a Medical Monitoring Advisory Group (MMAG) 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 RMA-related health concerns of the community. The ROD directed that the MMAG include representatives from the affected communities, regulatory agencies, local governments, Army, Shell Oil Company, U.S. Fish and Wildlife Service, and independent technical advisors. The ROD 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 2 in 5 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.

diagnosis[5], have limited scientific efforts to identify causative factors or combinations of factors. We may, however, monitor incidence rates, to be alert to significant deviation from the expected background rates. This in turn allows investigation of deviations with respect to potential environmental associations.

In Colorado, surveillance of cancer incidence is possible using data collected by the Colorado Central Cancer Registry (CCCR) at CDPHE. 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, state Vital Statistics, and directly from physicians, where the physician is solely responsible for diagnosis and treatment of a particular cancer. Pertinent data are 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.

Current and past studies conducted to assess cancer outcome in communities around the RMA look at disease frequency at the group level. Cancer surveillance studies such as this allow public health officials to investigate whether cancer is occurring in numbers that are significantly higher than background rates. There are recognized limitations to these types of studies, however, including lack of data on important individual risk factors, such as exposure to carcinogens in the workplace or indoor or outdoor ambient air, and length of residence at the address recorded in the cancer registry records. An additional limitation is that it is not possible to control for the influence of common carcinogenic exposure such as traffic-related exposure to benzene or other industrial influences within a given study boundary. In addition, assignment to a broad geographic area, such as a census tract, must be used to indicate individual exposure status. While a variety of exposures may contribute to the overall individual and population risk of some of the cancers reported, these factors cannot be accounted for or fully assessed with this type of study.

For the current study, very large population growth was identified as another potentially significant factor that adds to the uncertainty of the study findings, because up-to-date census data, with important demographic detail such as age structure of the study population, will not be available until after the 2010 Census for areas that have grown very rapidly since the last study was released.

CANCER INVESTIGATION HISTORY AND STUDY OBJECTIVES

The objectives of the ongoing cancer surveillance, previously established by the Rocky Mountain Arsenal Medical Monitoring Advisory Group, are to use cancer incidence data collected by the Colorado Central Cancer Registry to: 1) establish existing rates of cancer incidence prior to the RMA soil remediation, 2) analyze cancer incidence rates for significant temporal or spatial changes during and after the RMA soil remediation, and 3) investigate any increased, or otherwise unexplained, rates of cancer.

5 Latency is the period between the causative event and the diagnosis of the disease. Cancer latency may last a few years to as many as 30 years or more.

This report addresses objectives 2 and 3 above for a nine-year period, 1997-2005, beginning about the time that soil remediation commenced at the Rocky Mountain Arsenal. An earlier 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 published January, 2003 and addressed objectives 1 and 3 by analyzing 1979-1996 cancer data for the geographic area first described in the 1993 report *Cancer Incidence in the Northeastern Denver Metro Area: Report of the Ad Hoc Panel* (CDPHE 1993) (see Figure 1). A second analysis of cancer incidence, the initial post-baseline analysis, was published in October 2003. This study reported on cancer incidence for the time period 1997-2000. The current study evaluates an additional five years of cancer data for the same cancer types and geographic area assessed in the October 2003 report. A summary of findings from all three of these cancer surveillance reports is provided in Table 3 in the discussion section of this report.

METHODS

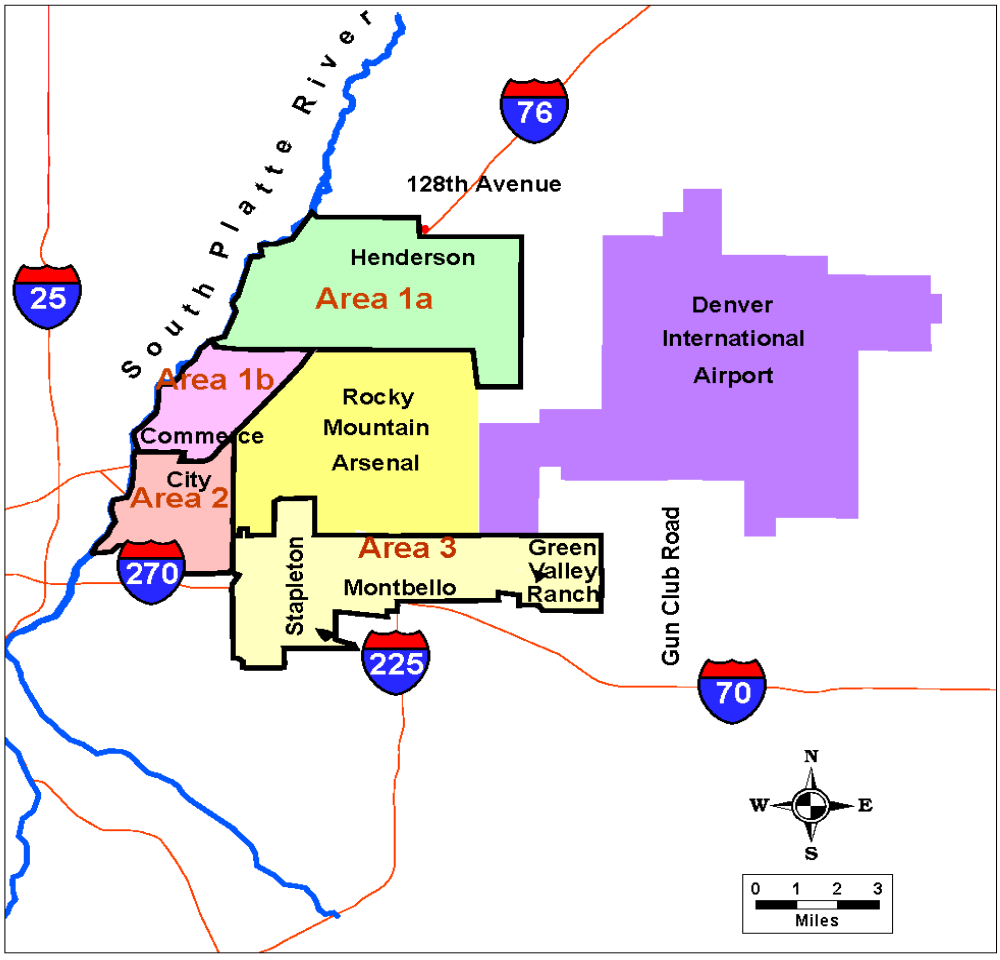
Virtually all cancer cases diagnosed since 1979 in the area of interest are identified and registered with the state Cancer Registry. Identification and registration of cancer cases involves standard processes including searching hospital medical charts, pathology laboratory records, and examining death certificate information.

As part of the present investigation, a common method of analysis was used to compare cancer diagnosis counts for an area in the vicinity of the Rocky Mountain Arsenal, for the time period of 1997-2005, to expected counts, using the remainder of the Denver metropolitan area as a standard or comparison population. The expected number of cancers was calculated by multiplying the cancer site-specific incidence rates in the standard population, adjusted by age, sex, and race/ethnicity, and then applying these rates to the study population.

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 rather than the individual level, 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.

Figure 1. Study area for the analysis of diagnosed vs. expected cancer cases for the northeast Denver area in the vicinity of the Rocky Mountain Arsenal, Colorado, 1997-2005, Surveillance Areas 1a, 1b, 2, and 3.



The boundaries for the current study 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 earlier reports, Area 1 has been further subdivided into Areas 1a, 1b, and Area 1 Combined, 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, 2,194 in 2000, and 14,843 in 2005. Its boundaries were Henderson Rd., E. 124th Ave., State Hwy. 51, E. 120th Ave., Tower Rd., Irondale Rd. (E. 88th Ave.), Buckley Rd., E. 96th 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, 8,513 in 2000, and 8,013 in 2005. Its boundaries were McKay Rd., E. 96th Ave., State Hwy. 2, E. 72nd Ave., U.S. Hwy. 85, E. 74th Ave. (State Hwy. 224), and the South Platte River.

Area 1 Combined, was defined as Area 1a and Area 1b together with a combined population of 9,100 in 1980, 8,376 in 1990, 10,707 in 2000, and 22,856 in 2005.

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, 18,939 in 2000, and 19,863 in 2005. Its boundaries were E. 74th Ave. (State Hwy. 224), U.S. Hwy. 85, E. 72nd 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 (see the description of a slight revision to the current study area in the footnote, below)[6], 83.03,

6 For this 1997-2005 report, the area defined as census tract 41.05 in Area 3 (located at the western end of Area 3, mostly west of Peoria, which includes the old Stapleton Airport) has not been included in the analysis for the following reasons: (1) the presence of a large prison population in this census tract; (2) the relatively new Stapleton development area, located in this tract, had no population until the last few years of this report's time period (i.e., a population increase from almost none to 700 persons in the year of 2003; an increase of about 600 persons in 2004, and an additional increase of about 800 persons in 2005); and (3) the Stapleton area has had no detailed population estimates available by age, race, and sex from the U.S. Census or the Denver Regional Council of Governments. The Denver County Jail, located in this census tract, houses about 2500 inmates and accounted for almost all of the population listed for census tract 41.05 up until the year 2003 when the Stapleton area started adding population. Also, the Denver Reception and Diagnostic Center, located just east of the jail at 10900 Smith Road, includes a 36 bed infirmary and a 480 bed maximum security facility that processes, tests, and classifies offenders entering the system, prior to their placement at one of Colorado's permanent facilities. Offenders are given a complete diagnostic evaluation, including medical and dental assessments, which, for some, may lead to a cancer diagnosis with the patient's address assigned to this facility in this census tract, even though the offender had been living elsewhere just prior to diagnosis. For these reasons, census tract 41.05 has been excluded from this study.

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, 39,311 in 2000, and 51,928 in 2005. Its boundaries were the Denver-Adams County Line, E. 56th Ave., Picadilly Rd., Denver-Adams County Line, Tower Rd., Denver-Adams County Line, E. 46th Ave., Denver-Adams County Line, Montview Blvd., Syracuse, E. 23rd Ave., Quebec, E. 48th 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. Labels for cancer sites have been edited for clarity and to add more detail since the 1997-2000 report, but refer to the same cancer sites as used in past publications. All cases of cancer diagnosed between 1997 and 2005 that were residents in the study area were identified. The address at the time of diagnosis for each case was used to assign residence within the census boundaries.

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, 1997-2005.*

Salivary Gland	Kidney
Oral cavity	Thyroid
Nasopharynx	Other Endocrine
Other Oral and Pharynx	Brain and Other Nervous System
Esophagus	Bone
Stomach	Leukemia
Small Intestine	Multiple Myeloma
Colorectal	Lymphoma
Liver and Intrahepatic Bile Duct	Soft Tissue
Other Biliary	Prostate
Pancreas	Testis
Larynx	Female Breast
Lung and Bronchus	Cervix
Melanoma	Uterus
Bladder	Ovary

Notes:
Oral cavity includes tongue, floor of mouth, and gum.
Other Oral and Pharynx includes tonsil, oropharynx, and hypopharynx.
Other Biliary includes gallbladder, extrahepatic bile duct, and ampulla of vater.
Other Endocrine includes thymus, adrenal gland, and other endocrine glands.
Bone includes bone and joints.

In order to estimate the number of cancers expected in the areas under study, it is critical to use the best available population data by gender, race and age for each year of the evaluation period. In this way, as the population grows or declines year by year in each area, the combination of the number of persons and years of follow-up (person-years) contributes to the number of cancers that would be expected. For example, if an area has high growth in the latter years of the evaluation period, the larger number of residents contributes fewer years toward the total expected number of cancers than if they had lived there the entire period. For this study, U.S. Census counts of population for census tracts 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. For the years after 2000, estimated population totals by census tract and year from the Denver Regional Council of Governments (DRCOG) for 2001-2005 were also used. For Area 3, zip code population estimates by age and gender were available for 2005, which were used to provide age and sex proportions (not counts) that were applied to DRCOG total population estimates for 2005 for the census tracts there. Yearly populations by age and sex could then be interpolated from 2000 to 2005. Zip code 80239 was used for the Montbello census tracts of 83.04, 83.05, 83.06, 83.11, and 83.12. Zip code 80249 was used for the developments within census tract 83.03 (Green Valley Ranch, Parkfield, and Gateway). Zip code boundaries in the vicinity of the other areas of this report did not match up well enough with the boundaries of Area 1a, Area 1b, or Area 2 to use this estimation technique, so DRCOG estimates of total population growth by year from 2001 to 2005, available for the census tracts in these areas, were used instead. More precise detailed population counts by age, race, and sex at the census tract level will be available from the U.S. Census for the year 2010 sometime in mid-2011 or 2012. The 2010 population counts will allow for full interpolation between 2000 and 2010 by age, sex, and race for each census tract.

Cancer rates from 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 CCCR, 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 6 counties of Adams, Arapahoe, Boulder, Broomfield, Douglas, and Jefferson, and the City and County of Denver.

Cancer rates from the Cancer Registry for men and women of comparable race/ethnic groups and ages 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 study area, stratified by age, gender, and race/ethnicity, was multiplied by the cancer rate for each age, gender, and race/ethnic group in the comparison population to produce the expected number of cancers.

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 (minimum value) and a higher number (maximum value). It is common to use a 95 percent confidence interval, which means, with 95 percent certainty, the true ratio occurs 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, 1997-2005. 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 tend to be 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 allow separation of selected anatomical categories of cancer into cell types. The case abstract data reviewed for this study included occupation, smoking history, and tumor-specific information, such as histology (or cell type of the tumor), anatomical sub-site, and multiple tumor sequencing. Available data were reviewed for discernable patterns within and across geographic areas.

RESULTS

The results of the study show that overall cancer incidence was not statistically elevated in both genders for any of the three geographic areas. Statistically significant elevations in cancer incidence were identified for nine of the thirty anatomic sites studied, including, lung, colorectal, stomach, small intestine, nasopharynx, bladder, larynx, leukemia, and extrahepatic bile dust/gallbladder. However, these elevations were not consistent across location, gender, race, or time.

Tables A1- A15, located in Appendix 1, 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 1997-2005, 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-A26, 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 compared to previous time periods. Cancer rates from the Cancer Registry for males and females of comparable race/ethnic groups and ages 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.

Specific anatomic cancer sites have been underlined in this section of the report to assist the reader with locating the pertinent information in the table being referenced. A summary of the general study findings by cancer type is presented on pages 17-28.

Area 1a, 1b and Combined Area 1 – Tables A1-A9 display statistics for Areas 1a, 1b, and Combined Area 1 for 1997-2005 for males and females separately and together.

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 was one exception to this general finding. Table A1 shows that for 1997-2005 there were more cases of other biliary cancers (including gallbladder and extrahepatic bile duct cancers) diagnosed in Area 1a than expected (five cases compared to about one case expected). Three of these five cases were cancers of the gallbladder. Since this elevation was also reflected in Combined Area 1 (six cases compared to about two cases expected), further information about this elevation is reported under "Combined Area 1."

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. Table 5 shows a bladder cancer elevation among males in Area 1b during 1997-2005 (14 cases compared to about seven or eight cases expected) for a statistically high ratio of 1.88. Table A16 shows the distribution of cases by race and age. Only White, non-Hispanic persons had a ratio that was statistically high at 2.14 (13 cases compared to about six cases expected). The distribution of cases by age showed a statistically high ratio of 2.68 only in the 65-74 age group with seven cases compared to about two or three

cases expected. CCCR abstracts showed a variety of occupations for the 14 cases. Nine of the 14 cases (64%) had a history of smoking documented in Cancer Registry abstracts. Including only abstracts where smoking information was recorded, nine of 11 cases (82%) were smokers. Almost all of the 14 bladder cancers were transitional cell carcinomas (79%), consistent with the predominance of this cell type for this cancer.

Tables A4 - A6 also display colorectal and lung cancer elevations for both genders in Area 1b. Since these colorectal and lung cancer elevations were also reflected in Combined Area 1, they are described in more detail in the Combined Area 1 Section of this report.

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 1997-2005. However, three types of cancer, colorectal, other biliary, and lung, had statistically high ratios.

Table A7 shows that there were 58 colorectal cancer diagnoses among both males and females together in Combined Area 1, compared to about 42 cases expected during 1997-2005, for a statistically high ratio of 1.37. Table A8 shows a similar finding for the male ratio (1.52), which is statistically high, with 35 cases compared to about 23 cases expected. Table A17 shows the distribution of cases by race and age. The ratio for Hispanic cases only was statistically high at 1.76 (16 cases compared to about nine cases expected). The only age group to show a statistically high ratio (2.30) was the 55-64 age group with 19 cases compared to about eight cases expected. CCCR abstracts showed a variety of occupations among these 58 cases. Twenty-four of the 58 cases (41%) had a history of smoking documented in Cancer Registry abstracts. Including only abstracts where smoking information was recorded, 57% of cases were smokers. The anatomical distribution among these 58 colorectal cancer cases was similar to the distribution found in the Denver metropolitan area. The ascending colon accounted for 24% of Area 1 colorectal cancer cases compared to 30% of Denver cases, the hepatic and splenic flexure and transverse colon accounted for 24% of Area 1 cases compared to 13% of Denver cases, and the descending colon accounted for 52% of Area 1 cases compared to 57% of Denver cases. Eight of the 58 colorectal cancers (14%) were diagnosed among just four individuals, each with double primary tumors during this time period, which is about twice the percentage of multiple tumors among colorectal cancer cases compared to the Denver area. Colorectal cancers in Combined Area 1 were detected at an earlier stage of disease than was typical in the rest of the Denver metropolitan area, especially in three of the last four years of this study period.

Table A7 shows that there were six other biliary cancers (including gallbladder cancer) diagnosed among both males and females together in Combined Area 1, compared to about two cases expected during 1997-2005 for a statistically high ratio of 2.79. Four of these six cases were cancers of the gallbladder. Five of these six cases were diagnosed in Area 1a where males and both genders combined had statistically high ratios, 8.93 and 6.74, respectively (see Tables A1 and A2). Table A18 shows that the distribution of these cancers by race and age revealed no statistically high ratios in Combined Area 1.

CCCR abstracts showed little detail about occupations for the six cases. Three of the six cases (50%) had a history of smoking documented in Cancer Registry abstracts. Two-thirds of these other biliary cancer cases (four out of six) were gallbladder cancers, which have a history of gallstones 75-90% of the time, according to the scientific literature.

Table A7 shows that there were 67 lung cancer diagnoses in Combined Area 1, compared to about 45 cases expected during 1997-2005, for a statistically high ratio of 1.50. Table A8 also shows a statistically high finding for the male ratio (1.74) with 43 cases compared to about 25 cases expected. Tables A4-A6 also show similar elevations in Area 1b, with males and females combined and males alone having statistically high ratios. Table A19 shows that most cases were White, non-Hispanic (56 cases out of 67 cases) and the ratio for White, non-Hispanic cases only was statistically high at 1.58 (56 cases compared to about 35 cases expected. The distribution of cases by age showed elevations in most age groups from 45 and above, with the ratio of 2.48 (23 cases compared to about nine cases expected) for the 55-64 age group being statistically high. Cancer Registry abstracts showed a variety of occupations among these 67 cases. About 85 percent of these cases (57 out of 67 cases) had a history of smoking. Limiting this calculation to only cases with smoking information recorded on the abstracts, 97 percent of lung cancer cases (57 out of 59) were smokers. There were no uncommon histological cell types recorded and the distribution of types of lung cancer in Combined Area 1, during 1997-2005, was similar to the Denver metropolitan area. The cases included the major forms of lung cancer, squamous cell carcinomas (19% vs. 18% in metropolitan Denver), large cell carcinomas (4% vs. 6%), small cell carcinomas (22% vs. 13%), adenocarcinomas (31% vs. 35%), and all other types (24% vs. 28%).

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 1997-2005. There were four types of cancer found to have statistically higher numbers of cases than expected: stomach, female larynx, lung, and male leukemias.

Table A10 shows that there were 17 stomach cancers diagnosed in Area 2 compared to about 10 cases expected for a statistically high ratio of 1.79. Table A11 shows a statistically high ratio for males (2.12), as well, (12 cases compared to about six cases expected). As shown in Table A12, there were 5 cases of stomach cancer diagnosed in Area 2 females, compared to about 4 expected, which was not statistically high. Table A20 shows the distribution of stomach cancers in Area 2 by race and age. Most cases were white, non-Hispanic (11 cases compared to about four cases expected) and the ratio of diagnosed to expected cases of 2.48 was statistically high. The 55-64 age group was the only age group to have a statistically high ratio (3.71), with five cases compared to about one or two cases expected. Cancer Registry abstracts showed a variety of occupations among these 17 cases. About 47 percent of these cases (eight out of 17 cases) had a history of smoking. Limiting this calculation to only cases with smoking information recorded on the abstracts, 62 percent of stomach cancer cases (eight out of 13) were smokers. There were no uncommon histological cell types recorded and the distribution of types of stomach cancer in Area 2 during 1997-2005 was similar to the Denver metropolitan area. Adenomas and adenocarcinomas accounted for 66% of Area 2

cases and 65% of Denver cases, carcinomas accounted for 12% of Area 2 cases and 4% of Denver cases, and cystic, mucinous, and serous neoplasms represented 24% of Area 2 cases and 21% of Denver cases.

Table A12 shows that there were four female larynx cancers diagnosed in Area 2 during 1997-2005 compared to about one case expected for a statistically high ratio of 3.74. Table A21 shows that there were too few cases among the race/ethnic categories or specific age groups to perform statistical testing. Little occupation information was found on Cancer Registry abstracts, but 75% of the cases had a positive history of smoking (3 out of 4 cases). These four cases were all squamous cell carcinomas, consistent with the predominance of this cell type for larynx cancers; 92% of Denver area cases were this cell type.

Table A10 shows that there were 87 lung cancer diagnoses in Area 2 compared to about 67 cases expected during 1997-2005 for a statistically high ratio of 1.31. Table A11 also shows a statistically high ratio for males (1.52) with 54 cases compared to about 36 cases expected. Table A22 shows that most cases were White, non-Hispanic (69 cases out of 87 cases) and the ratio for white, non-Hispanic cases only was statistically high at 1.44 (69 cases compared to about 48 cases expected). The distribution of cases by age showed a statistically high ratio (1.45) only in the 75+ age group (34 cases compared to about 23 cases expected). The one case in the 0-4 age group was diagnosed in a child whose very rare cancer was found to be linked to a likely familial syndrome. Cancer Registry abstracts showed a variety of occupations among these 87 cases. About 70 percent of these cases (61 out of 87 cases) had a history of smoking. Limiting this calculation to only cases with smoking information recorded on the abstracts, 94 percent of lung cancer cases in Area 2 (61 out of 65) were smokers. There were no uncommon histological cell types recorded and the distribution of types of lung cancer in Area 2 during 1997-2005 was similar to the Denver Metropolitan Area. The cases included the major forms of lung cancer, squamous cell carcinomas (20% vs. 20% in metropolitan Denver), large cell carcinomas (8% vs. 8%), small cell carcinomas (12% vs. 11%), adenocarcinomas (22% vs. 22%), and all other types (38% vs. 39%).

Table A11 shows that there were 19 male leukemias diagnosed in Area 2 during 1997-2005 compared to about 10 cases expected for a statistically high ratio of 1.93. As seen in Table A23, which displays race and age distributions for the 19 male leukemias, none of the age groups had statistically higher ratios and only Hispanics had a statistically higher number of cases than expected (nine cases compared to about three cases expected) for a ratio of 3.29. About 42 percent of the male leukemia cases (eight out of 19 cases) had a history of smoking. Limiting this calculation to only cases with smoking information recorded on the abstracts, 67 percent of the male leukemias (eight out of 12 cases) were smokers. There was a variety of leukemia types represented among the 19 male cases with distribution by type similar to the Denver area. Acute lymphocytic leukemia (ALL) accounted for 21% of Area 2 cases and 14% of Denver cases, acute myeloid leukemia (AML) accounted for 32% of Area 2 cases and 27% of Denver cases, chronic lymphoctyic leukemia (CLL) accounted for 26% of Area 2 cases and 31% of Denver cases and chronic myeloctyic leukemia accounted for 5% of Area 2 cases and 13% of

Denver cases. Only seven of 19 male leukemia cases in Area 2 had occupations listed on Cancer Registry abstracts, but five of these seven cases were truck drivers, all with AML, a type of leukemia associated with occupational exposure to benzene from diesel exhaust.

Area 3 - Table A13 shows that the number of all cancers combined diagnosed among both males and females together in Area 3 was statistically high (863 cases compared to about 794 cases expected for a ratio of 1.09). The ratio for males of 1.05 was not statistically high, but Table A15 shows that the ratio for female cancers of all types combined was statistically high at 1.12 with 440 cases compared to about 392 cases expected. It is interesting to note that this elevation among female cancers was comprised of a number of ratios of cancer types being modestly elevated, with only one type (bladder cancer) having a statistically high ratio. Of these other cancer types reported in higher than expected numbers in females, counts for all but one type (lung cancer) were well within expected statistical variation. The lung cancer ratio among females was 1.36 (44 cases compared to about 32 cases expected), which was one case short of being statistically higher than expected, and was sufficient alone to force the statistically high ratio for all cancers combined for females. In other words, excluding lung cancers, all other cancers combined for females in Area 3 were within expected statistical limits. The group of female lung cancers also had a strong connection with smoking; 60% of the cases had a CCCR abstract mention of a smoking history, and excluding cases with unknown smoking status, 84% of the cases had a positive smoking history. Tables A13-A15 display statistically high ratios for three individual types of cancer in Area 3, as well: nasopharynx in males, small intestine in males, and bladder in females.

Table A13 shows that there were five nasopharynx cancers diagnosed in Area 3 during 1997-2005 compared to about one or two cases expected for a statistically high ratio of 4.38. Table A14 shows that the ratio for male nasopharynx cancers was also statistically high (4.41) with four cases compared to about one case expected. Table A24 shows that there were too few cases among the race/ethnic categories or specific age groups to perform statistical testing. Two of the five cases (one male and one female) were in patients born in Southeast Asia, an area where cancers of the nasopharynx are extremely common (ACS, 2001). Occupation information from the Cancer Registry abstracts was limited, but two of the remaining three cases had automobile service jobs. Four of the five cases diagnosed were carcinomas (the other case was listed only as malignant neoplasm). Two cases occurred in the 10-14 age group. One child was listed as having fetal alcohol syndrome. Both mothers of the two younger cases had a past history of cancer. It is also of interest to note that, in light of the fact that two cases had a potentially strong risk factor for this disease (i.e., Southeast Asia birthplace), even one less case (four cases rather than five cases) would have resulted in a ratio within expected statistical variation for this area.

Table A13 shows that there were eight small intestine cancers diagnosed in Area 3 during 1997-2005 compared to about three cases expected for a statistically high ratio of 2.46. Table A14 shows that the ratio for male small intestine cancers was also statistically high (3.93) with seven cases compared to about two cases expected. Table A25 shows that

one-half of the small intestine cancer cases were White, non-Hispanic, and the ratio for White, non-Hispanic cases only, was statistically high at 5.46 (4 cases compared to about one case expected). Only one of the age groups (55-64) had enough cases to test the ratio and it was not statistically high. Occupation information from the Cancer Registry abstracts was limited, showing no particular pattern of employment. About 38 percent of the small intestine cancer cases (three out of eight cases) had a history of smoking. Limiting this calculation to only cases with smoking information recorded on the abstracts, 43 percent of the cases (three out of seven cases) were smokers. There were no uncommon histological cell types recorded. The distribution of types of small intestine cancer reported in Area 3 during 1997-2005 was similar to the Denver metropolitan area. The cases included adenomas and adenocarcinomas (88% in Area 2 vs. 81% in Denver) and cystic, mucinous, and serous neoplasms (12% in Area 2 vs. 4% in Denver). It is again of interest to note that that only one less case of small intestine cancer (seven cases rather than eight cases) would have resulted in a ratio within expected statistical variation for this area.

Table A13 also shows that there were 29 male and female bladder cancer cases in Area 3 compared to about 18 cases expected during 1997-2005, resulting in a statistically high ratio of 1.64. Table A15 shows a similar finding for the female ratio (2.28), which is statistically high, with nine cases compared to about four cases expected. Table A26 shows that half of the bladder cancer cases were White, non-Hispanic, and the ratio for White, non-Hispanic cases only was statistically high at 1.86 (14 cases compared to about eight cases expected). The distribution of cases by age showed an elevation in the 35-44 age group, with the ratio of 7.54 being statistically high (six cases compared to about one case expected). CCCR abstracts showed a variety of occupations for these 29 cases. Eighteen of the 29 cases (62%) had a history of smoking documented in Cancer Registry abstracts. Including only abstracts where smoking information was recorded, 18 of 22 cases (82%) were smokers. Almost all of the 29 bladder cancers were transitional cell carcinomas (97%), consistent with the predominance of this cell type for this cancer.

Table 2 provides a summary of all statistically elevated cancer counts for the time period 1997-2005. Cancers that were statistically lower than expected included: female breast (Areas 1a, 1b, 1 Combined, and Area 2); prostate (Area 1b, 1 Combined, and Area 2); lymphoma (males in Area 1 Combined); melanoma (females in Area 2 and Area 3, and males & females combined in Area 2 and Area 3); and multiple myeloma (for males & females combined in Area 3).

Table 2. Summary of statistically elevated cancer findings, by area and gender, for the period 1997-2005.

Cancer Site	**Combined AREA1**			**AREA 1A**			**AREA 1B**			**AREA 2**			**AREA 3**		
	M	F	B	M	F	B	M	F	B	M	F	B	M	F	B
Bladder							X							X	X
Lung	X		X				X		X	X		X			
Larynx											X				
Colorectal	X		X				X		X						
Other Biliary/ Gallbladder			X	X		X									
Small Intestine													X		X
Stomach										X		X			
Nasopharynx													X		X
Leukemia										X					
All cancers														X	X

X indicates a statistically elevated number of cancers
M = Males
F = Females
B = Both males and females combined

STATISTICAL ANALYSIS AND THE MULTIPLE COMPARISONS PROBLEM

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, with 216 independent statistical tests conducted on separate cancer sites, by gender and for several different areas, there were 12 ratios statistically higher than expected (5.6 percent of the tests compared to about 2.5 percent predicted by chance alone) and seven ratios statistically lower than expected (3.2 percent of the tests compared to about 2.5 percent predicted by chance alone). Evaluating all 520 comparisons made, 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, 40 ratios were statistically higher than expected (7.7 percent of the tests) and 13 ratios were statistically lower than expected (2.5 percent of the tests). Note that ratios based on less than three cases were not tested statistically, which likely partially accounts for the lower percentage of statistically low outcomes reported in this study.

DISCUSSION

The primary focus of this study was to evaluate the incidence of 30 different target cancer sites, previously selected for study by the RMA MMAG. Outcomes for all cancers combined, in three distinct geographic areas in the vicinity of the RMA site, were assessed as well. As described in the methods section of this report, the evaluation entailed comparing cancer incidence to expected cancer counts, based on age, gender, and race/ethnicity. This evaluation focused on cancer outcome data for the time period when on-post soil cleanup commenced at the Rocky Mountain Arsenal (1997), through 2005, the last complete year of cancer data available at the time the study began.

Cancer incidence, when compared to a standard population using statistical testing procedures, allows the identification of subpopulations with "higher than average" rates of specified categories of cancer. Due to the complex and multi-factorial nature of cancer occurrence, however, it is generally not possible to link a specific case of cancer to a specific exposure. Nor is it typically possible to distinguish between cancers resulting from chemical exposure versus other established risk factors, such as lifestyle factors (i.e., poor diet, tobacco use, excessive alcohol consumption), or other genetic, immunological and viral influences. To help interpret cancer surveillance data, however, some case-specific information may be readily available from the Cancer Registry, regarding smoking history, alcohol use, occupation, the frequency of cancer of specific anatomical sites, and the distribution of histological cell type within those anatomical sites. This type of information helps interpret cancer outcome data and inform about potential etiologies or patterns in cancer occurrence.

Case review of information recorded for primary occupation may reveal established causal factors. Certain occupations may have recognized potential for exposure to specific carcinogenic agents, and broad categories of occupation, such as farming and industrial work, may involve exposures to a variety of carcinogens. Occupational data contained in the Cancer Registry case abstract do not provide a complete picture of the life-long working experience, but can indicate the presence of a known risk factor for a specific type of cancer.

In many cases, a history of tobacco use is recorded in the Cancer Registry abstract, 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 at least one-third of all cancer cases in developed countries (ACS, 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 nonsmokers (Wynder, 1998; Doll et al., 1994). Increased cancer risk is also evident for other organ tissues including the oropharynx, esophagus, pancreas, bladder, kidney, stomach, colon and rectum, and for acute myeloid leukemia (ATSDR, 2008). Some studies have also indicated an increased risk in smokers for cancer of the uterine cervix, with some suggestive evidence for increased risk of breast cancer (ACS, 2008).

Excessive consumption of alcohol is strongly associated with cancer of the oral cavity, pharynx, larynx, esophagus, liver, and large bowel. Epidemiological evidence suggests that approximately 5 percent of cancer deaths in the U.S. are related to alcohol consumption, but other factors such as generally poor nutrition may also be involved (ACS, 2001). Information about use of alcohol is sometimes available from Cancer Registry records to help assess this potential risk factor.

Information on the type of cancer cell or cancer cell morphology for specific anatomical sites may be obtained from Cancer Registry records. Some epidemiological information is available which indicates that exposure to certain chemicals can be associated with a shift in the distribution of histology, resulting in an increased number of tumors of a specific cell type. 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 factor.

An equally important source of information for interpretation of cancer incidence data is the epidemiological literature. A substantial body of scientific and medical information has been collected describing the relationship between cancer, population incidence, and the known associated risk factors. The significance of this information is discussed below for individual cancer types where a statistically high number of cases were reported for the study area.

General Study Findings by Cancer Type

A variety of cancers were found to be statistically higher than expected within the northeast Denver study areas in the vicinity of the RMA during the time period 1997-2005 (see Table 2). The findings of this study indicate that, overall, the total number of all cancers combined was close to expected levels in Areas 1 and 2, although there were a number of individual cancer types that were statistically elevated (lung, colorectal, male bladder, gallbladder and other biliary, stomach, male leukemia and female larynx).

For Area 3, there was a trend towards a higher than expected rate of cancer in this geographic area, when compared to cancer outcomes in the rest of the Denver metropolitan area, with a statistically elevated number of all cancers combined for males and females combined (863 cancers diagnosed, compared to about 794 expected). The number of all cancers combined was also statistically high for Area 3 females, but none of the individual cancer sites were statistically high in females, except for cancer of the bladder. There was a higher than expected number of all cancers combined in males in Area 3 (423 cases diagnosed, compared to about 402 expected), but the elevation was not statistically significant. Two individual cancer sites, nasopharynx and small intestine,

were statistically higher than expected in males in Area 3.

One general observation of note was the occurrence of inconsistent groupings for many of the cancers that were significantly elevated, with statistically high incidence of many cancers being reported in only one gender or one geographic area. A residential environmental exposure to a cancer-causing agent, such as drinking contaminated water or breathing contaminated air, typically is expected to cause a similar effect in both men and women, although there are still many unknowns about specific mechanisms responsible for cancer causation and what role gender-specific factors, such as hormone levels or capacity to store fat-soluble chemicals, may play. The elevated findings in men but not women in Areas 1 and 2 may indicate an additional occupational disease burden, or other factors such as a higher proportion of smokers. In Area 3, statistically significant findings were reported in different genders for different types of cancers.

As with differences in rates of diagnosis between genders, an elevation of a particular cancer in only one race/ethnicity sub-population, tends to argue against a common causative agent or co-factor present among the entire population, whereas an elevation in more than a single race/ethnic group may suggest shared risk factors. For this study, anatomical sites, or types, of cancer for which the race/ethnicity distribution was reviewed for selected genders were colorectal, lung, bladder, gallbladder and other biliary, stomach, leukemia, nasopharyx, and small intestine. Statistically significant elevations were not observed among all racial/ethnic segments of the population. In all but two of these cases, statistically significant elevations were limited to White, non-Hispanic cases, with statistically elevated numbers of cancer reported in Hispanics only for male leukemia in Area 2 and for colorectal cancer in males and females together in Combined Area 1. This difference may be partly attributable to the size of each population segment within the study areas, with generally small numbers of cancers of other racial and ethnic groups resulting in an inability to statistically compare some race-specific observed/expected cancer ratios.

To better evaluate the statistically high findings detected in this study, additional case review and investigation of cancer-specific etiologies were conducted to help interpret the outcomes. These findings are discussed below for each specific anatomical site that was statistically elevated.

Colorectal Cancer

In the current study, colorectal cancer ratios were within expected limits in Areas 2 and 3. In Area 1b and Combined Area 1 tracts, there were statistically high numbers of colorectal cancers in males, and in males and females combined, with males accounting for about 12 of the 16 excess cases (58 diagnosed cases, compared to 42 expected). Risk factors for colorectal cancer reported in the epidemiological literature include family history of colorectal cancer or polyps, pre-existing inflammatory bowel disease, lack of physical activity, obesity, dietary characteristics such as high-fat and/or low-fiber diet, and heavy alcohol consumption (ACS, 2008). Evidence regarding environmental or workplace exposure and increased risk of colorectal cancer is generally limited or

inconsistent (Siemiatycki, 2004). There has been some suggestive evidence of increased risk of colon cancer from exposure to the solvents toluene and xylene (Lynge, 1997), and possibly high exposure to some PCB congeners. Results from NCI's Agricultural Health Study also found that pesticide applicators exposed to the herbicide dicamba were at increased risk for colon cancer (Samanic, 2006). Recent research conducted by the American Cancer Society links long-term smoking with higher risk for colorectal cancer (Chao, 2000). Studies also indicate colorectal cancer occurs earlier in smokers (Botteri, 2008). In this study, among cases with available smoking history data, 57% of the colorectal cancer cases in Combined Area 1 had a history of smoking recorded on the cancer abstract, but information about duration of smoking was not available.

It is notable that eight of the 58 colorectal cancers (14%) were diagnosed among just four individuals, each with double primary tumors during this time period. This is about twice the percentage of multiple tumors diagnosed among colorectal cancer cases in the rest of the Denver metropolitan area. These four additional cancers were sufficient to increase the O/E ratios for colorectal cancer above statistical significance for Area 1b males, males in Combined Area 1, and both sexes together in Combined Area 1.

Lung Cancer

Findings for Area 1 and Area 2 showed a statistically elevated incidence of lung cancer among males and females during 1997-2005, with statistically elevated O/E ratios ranging from 1.31 (CI=1.05-1.62) for males and females combined in Area 2, to 1.76 (CI=1.30-2.34) in Area 1b. Most cases were among persons aged 45 years and older. Because smoking is a well-recognized risk factor for lung cancer, a review of all lung cancer case records was conducted to determine smoking status. Cigarette smoking specifically is by far the most important risk factor for the development of lung cancer. According to the American Cancer Society, the risk of developing lung cancer is about 23 times higher in male smokers and 13 times higher in female smokers compared to lifelong non-smokers (ACS 2008). In this study, among cases for which information about smoking was recorded, 94 percent of the cases in Area 2, and 97 percent in Combined Area 1, had a history of smoking. There were no uncommon histological cell types recorded and the distribution of types of lung cancer in Area 1 and Area 2 during 1997-2005 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. Cases diagnosed at an unusually early age were reviewed and found to occur in individuals with either a history of smoking, or with a predisposing disease or underlying genetic condition.

Other known environmental risk factors for lung cancer include exposure to asbestos, arsenic, radon, mustard gas, and other forms of air pollution. Other air pollutants that may be carcinogenic to the lung include diesel exhaust, soot, pitch and tar, dioxin, chromium, cadmium and nickel compounds, and other combustion by-products, such as polycyclic aromatic hydrocarbons (Boffetta, 2004). Some studies have also linked increased risk of lung cancer to high dose exposure to the solvents benzene and toluene, and to natural fibers, such as silica, and wood dust (Clapp, 2007). Recent evidence from

the Agricultural Health Study found a significantly increased incidence of lung cancer in pesticide applicators exposed to the organochlorine pesticide dieldrin, in contrast to previous mortality studies which did not detect significant outcomes (Purdue, 2006). Animal studies have also shown that exposure to N-nitroso-dimethylamine (NDMA), through ingestion or inhalation, can increase the risk of developing lung and liver cancer (ATSDR 1999), with some suggestive findings in studies in humans. NDMA was formerly used in the production of liquid rocket fuel, pesticides, dye manufacturing and a variety of other uses. Exposure to NDMA can also occur through ingestion of smoked or cured meats or fish, or from inhaling cigarette smoke. It has been identified by EPA as an emerging drinking water contaminant because of its presence as an unintended byproduct of chlorination of drinking water and wastewater (EPA, 2008).

Smoking is likely the predominant cause of the elevated lung cancer findings in this study, with very few cases occurring in non-smokers. The possibility of some causal or combined effect from exposure to other factors, such as exposure to carcinogens in an occupational setting or other chemical exposure indoors or in the outdoor ambient air environment, cannot be ruled out by this analysis, but any such effect would likely be small compared to the smoking effect.

Laryngeal Cancer

Cancer of the larynx was statistically high in one of the three geographic areas evaluated in this study (Area 2), with ratios statistically elevated in females only (4 cases reported versus 1 expected). Three of four cases (75%) had a positive history of smoking. Risk to smokers for developing cancer of the larynx is about 20-35 times higher than for non-smokers, depending on the level of tobacco use. Some studies have reported an increased risk of developing laryngeal cancer in people with diets high in meat fat, or low in fruits and vegetables (De Stefani, 1995; Riboli, 1996). Work place exposures associated with an increase in cancer of the larynx include exposure to mustard gas, wood dust, metal-working fluids and mineral oils, and exposure to reactive chemicals, such as sulfuric acid mists (Siemiatycki, 2004). An elevation in cancer of the larynx was also found in Area 2 in a previous study, for males and females combined, for the time period 1979-1988 (see Table 3). A strong association with smoking was evident in that finding as well.

Bladder Cancer

Bladder cancer was statistically elevated in males in Area 1b, and in females and males combined, as well as females alone, in Area 3, during the period 1997-2005. The elevation in Area 1b was restricted to males, with no cases diagnosed in females from 1997-2005. In Area 3, there was a higher than expected number of bladder cancers in males (20 compared to 14 expected). This elevation was not statistically high, but did contribute to a statistically high finding for males and females combined in Area 3.

The distribution of cases by age showed that, in Area 1b, most cases occurred in individuals age 55 and over (see Table A16), with a statistically high number of cases occurring only in the 65-74 age group (7 cases compared to about 2 to 3 expected). The

median age at diagnosis for bladder cancer is 68 years (ACS, 2008). In Area 3, there was an elevation in the 35-44 age group, with a ratio of 7.54, which was statistically high (six cases compared to about one case expected). Six of seven cases in individuals under age 45 had a history of smoking, with smoking status unknown for one case.

Smoking is recognized as a primary risk factor for bladder cancer, accounting for as many as 60 percent of all cases. Cancer Registry case abstracts showed that 82 percent of the individual cases diagnosed from 1997-2005 in both Area 1b and Area 3 occurred in smokers, compared to 74 percent in the comparison population. This indicates some of the increased risk above that expected is likely due to the higher rate of smoking among individuals from the study area who were diagnosed with bladder cancer.

Table 3. Comparison of statistically elevated cancer incidence from 1997-2005 with outcomes from earlier CDPHE incidence studies.

TIME PERIOD	ELEVATED CANCER SITE BY STUDY AREA				
	Combined Area 1	Area 1a	Area 1b	Area 2	Area 3
1997-2005	Lung (M,B) Colorectal (M,B) Other Biliary (B)	Other Biliary (M,B)	Bladder (M) Lung (M,B) Colorectal (M,B)	Lung (M,B) Larynx (F) Stomach (M,B) Leukemia (M)	Bladder (F,B) Nasopharynx (M,B) Small intestine (M,B) All cancers (F,B)
1997-2000	Lung (M,F,B) Pancreas (B)	Lung (M,B) Pancreas (B)	Lung (M,F,B)	Lymphoma (M)	Brain (F,B)
1979-1996	Bladder (M,B) Lung (F,B) Stomach (M)		Bladder (M,B) Lung (B) Other oral/ Pharynx (M)	Lung (B) Cervix (F)	Salivary gland (F,B)
1989-1996	Lung (M,B) Stomach (M,B)		All cancers (B) Lung (M,B) Larynx (M) Kidney (F) Stomach (M,B)	Leukemia (F,B)	Salivary gland (F,B)
1979-1988		Lung (F) Kidney (M)	Lung (B)	Lung (M,F,B) Larynx (B) Cervix (F)	Multiple Myeloma (F)

M= Statistically elevated number of cancers in males
F= Statistically elevated number of cancers in females
B= Statistically elevated number of cancers in males and females combined

One-fourth of bladder cancer cases in the United States are estimated to be associated with occupational exposures. Occupations that have been reported in the literature to increase the risk of developing bladder cancer include the rubber, textile, painting and printing industries, and the manufacture of aluminum, dyestuffs, pigments, and certain pesticides. Other recognized risk factors for bladder cancer include individual exposure to inorganic arsenic, especially in smokers; exposure to aromatic amines in dyes (i.e., benzidine and beta-naphthylamine); exposure to coal tar and PAHs in combustion products and diesel engine exhaust (Siemiatycki, 2004). Finally, some studies have provided suggestive evidence of an increased risk of bladder cancer from exposure to chlorination by-products, including chloroform (Villanueva, 2006). History of bladder stones, bladder infections, and other diseases of the urinary tract; inflammation from long-term catheter use; and use of certain weight-loss products have also been identified as risk factors for bladder cancer (ACS, 2008). 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 get the disease. Bladder cancer is rare in individuals under age 40 (NCI, 2008).

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 statistically significant elevation was observed for the period 1981-1985; the ratio for a longer time period, 1979-1988 was elevated, but not statistically high. Subsequent to the 1993 report, a case-control 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. 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.

Subsequent studies published by CDPHE in 2003 also showed a statistically significant elevation in bladder cancer in Area 1b. Elevations have not been reported for bladder cancer in Areas 2 or 3 in the past (see Table 3). With the exception of the high number of bladder cancers diagnosed in Area 3 from 1997-2005, statistically elevated numbers of bladder cancer cases have been accompanied by an elevation of lung cancer, which may indicate a smoking effect. In past studies, statistical elevations in the incidence of bladder cancer have occurred predominantly in males, a finding consistent with an expected stronger influence from smoking and occupational exposure from blue-collar jobs. The finding in Area 3 females in the current study is somewhat of an anomaly in this regard, although the findings are based on relatively few cases (9 diagnosed compared to about 4 expected), and there was still a strong (60%) connection with smoking. The finding reported in the current study of a statistically higher than expected number of cases in a relatively young age group (35-44) is likely largely smoking related, with a history of smoking reported in all cases where smoking status was available.

Gallbladder and Other Biliary Tract Cancer

Other biliary tract cancer, (defined here as cancer of the extrahepatic bile duct and gallbladder) is a relatively rare disease, affecting about 4,600 people in the U.S. in 2007, with men being affected more than women (ACS 2008). Rates in individuals from Asia, Latin America, and the Middle East are often high, due to common parasitic infections in the bile duct. Other risk factors identified for biliary tract cancer include presence of stones in the bile duct or gallbladder, history of ulcerative colitis or other long-standing inflammatory disease (sclerosing cholangitis), and being overweight or obese (Randi, 2009).

Similarly, presence of gallstones and obesity are recognized as strong risk factors for gallbladder cancer. The presence of certain chronic infections (*Salmonella typhi and S. paratyphi*) and *Helicobacter bilis* and *H. pylori* bacteria may also contribute to risk of developing gallbladder cancer. Apart from obesity, no other dietary factors have been identified as risk factors for developing gallbladder cancer. Risk of developing gallbladder cancer is generally higher in females than males and significantly higher in Hispanic, American Indian, and Alaska Native populations compared to other race/ethnic groups in the U.S. (Randi, 2006). The potential biological mechanism for an association between obesity and increased risk of gallbladder cancer may be via a direct role (increase in hormones, insulin, insulin-like growth factors) or may have an indirect role due to the increased risk of gallstone formation for individuals who are obese (Larsson, 2007).

Chemicals that have been associated in some studies with increased risk of gallbladder and biliary cancers include dioxin, nitrosamines in the diet, dibromochloropropane, polychlorinated biphenyls (PCBs), and possibly exposure to dinitrotoluene in munitions workers (Clapp 2007; Stayner 1983). There is suggestive evidence of an association with trichloroethylene (Siemiatycki, 2004). However, many of these studies do not clearly distinguish the anatomical site of the tumor within the biliary tract (i.e., intrahepatic versus extrahepatic). Smoking has been shown to contribute to an increased risk of developing bile duct cancer in individuals with sclerosing cholangitis, a chronic inflammatory condition. In the current study, three of the six cases (50%) had a history of smoking documented in Cancer Registry abstracts, but it is not known if these individuals had a history of other underlying inflammatory disease that could contribute to increased risk from smoking.

A statistically high number of other biliary tract cancers were diagnosed in males and in males and females combined, in Area 1a. The number of cases detected in Area 1a was high enough to result in a statistical elevation for both males and females combined, for Combined Area 1, with only one case (in a female) being diagnosed in Area 1b for the time period 1997-2005.

Four of the six cases in Area 1 Combined were cancers of the gallbladder, which is strongly related to the presence of gallstones, a condition linked with obesity. Past surveys of northeast Denver residents indicate there may be some unquantifiable impact

from higher rates of obesity reported in the study area boundary than in the comparison population. Results from the state Behavioral Risk Factor Surveillance Study (BRFSS) indicate a consistently higher percentage of north Denver residents are obese (BMI>30), compared to overall state rates for the years data are available (2003-2008).

Elevations of gallbladder and other biliary tract cancer have not been seen in any of the previous time periods studied.[7] Additional case investigation was conducted to further investigate the statistical elevation in other biliary cancers reported in Area 1a in the current study. No obvious risk factors were identified.[8]

It should be noted that the elevated cancer incidence ratio of 2.79 reported for gallbladder/other biliary tract cancer in males and females in Combined Area 1 is based on a small number of cases (6 diagnosed versus about 2 expected) with a relatively unstable 95 percent confidence interval (CI) of 1.02-6.08. The weak statistical association for this finding, in combination with large population fluctuations, makes it difficult to interpret this finding with much certainty.

Stomach Cancer

Stomach cancer is more common in men than women, with particularly high rates in Asian and Pacific Islanders. Stomach cancer is most often diagnosed in individuals over age 55 and rarely occurs in individuals under age 40. Stomach cancers are more strongly associated with dietary factors and gastric health conditions than with environmental

---

7 – The Agency for Toxic Substances and Disease Registry (ATSDR) conducted an extensive public health assessment (PHA) in 1995, as part of the regulatory review process for the Rocky Mountain Arsenal Superfund Site (ATSDR, 1995). The PHA discussed a finding of a statistically elevated number of biliary tract/ gallbladder cancers in an occupational study of 2,384 workers at RMA pesticide production facilities (Amoateng-Adjepong, 1995). The 1995 mortality study reported a statistically high SMR of 386 (CI = 125 to 900) for hepatobiliary cancers among a subcohort of white men. Detailed exposure status was lacking, however, for many workers, and 3 of the 5 hepatobiliary cancers occurred in workers with short exposure history (2 years or less). Results reported in the 1995 study were also based on very few cases (5 cases compared to 1.3 expected) and results did not display duration-response trends. The retrospective mortality study was conducted to evaluate health outcomes in workers exposed to a variety of pesticides produced at the plant site from 1952-1982. The authors concluded that the finding of an increase in mortality for hepatobiliary cancer was not an outcome with a known association with any of the pesticides produced at the plant.

8 - No data were readily available on individual risk factors or residence history for the individual cases reported to the Cancer Registry. However, a review of geographic location for place of residence at time of diagnosis for the six cases of other biliary cancer, reported from 1997 to 2005 in Area 1 Combined, indicated residences for all cases were located fairly distant from the RMA. Of the 6 cases diagnosed with other biliary cancer, one case lived approximately ¾ of a mile from the north fence line of the RMA, and all others lived more than a mile from the RMA boundary. Residences for five of the 6 gallbladder cases were not served by well water. One of the 6 cases lived in a residence with a history of domestic well water use, however no elevations were detected in that well during routine sampling events in 1993 and 1997.

factors. Risk factors recognized by the American Cancer Society include: infection with the *H.pylori* bacteria; chronic gastritis; exposure to nitrates and nitrites from a diet high in cured meats, salted fish, smoked foods, or pickled vegetables; low intake of fresh fruit and vegetables; individuals with Type A blood; and presence of **c**ertain inherited conditions or disease states. The rate of stomach cancer in smokers is about double that for non-smokers, particularly for tumors that develop in the upper portion of the stomach (ACS, 2008).

Certain occupations, such as work in the coal, metal, and rubber industries have been associated in the epidemiological literature with a higher risk of stomach cancer. Some occupational studies have provided suggestive evidence of increased stomach cancer in workers exposed to PCBs during the manufacturing of electrical capacitors (Prince, 2006) and an increased risk of developing stomach and esophageal cancer in aerospace workers exposed to certain mineral oils (Zhao, 2005). In addition, a 2006 IARC monograph identified an association between occupational exposure to lead and increased rates of stomach cancer in 5 of 6 occupational cohorts studied (IARC, 2006). No residential environmental exposure studies were identified with a statistically high incidence of stomach cancers.

Findings from the 1997-2005 cancer incidence study revealed statistically high ratios for stomach cancers in males (O/E of 2.12) and in males and females combined (O/E of 1.79) in Area 2. Sixty-two percent of stomach cancer cases (eight out of 13) were smokers, a recognized risk factor for gastric cancer. One case was a non-smoker and smoking status was unknown for the other four cases. A statistically significant elevation in stomach cancer in males and in males and females combined was reported previously for the 1979-1996 time period (see Table 3), however this finding occurred in a different geographic area.

Leukemia

The findings in this study showed a statistically high incidence of leukemia in males in Area 2 only (O/E ratio of 1.93), with a statistically high number of cases occurring in Hispanics. Leukemias were also elevated in Area 2 in a previous report for the time period 1989-1996, however the elevation occurred in females and not in males (see Table 3). There was no obvious time trend over the period 1997-2005, with an average occurrence of about two cases per year, with each year having from none to three cases diagnosed.

Leukemias are cancers of white blood cells (leukocytes). In the United States, leukemias constitute approximately 3 percent of all cancers. Leukemias are classified as either acute or chronic, with a strong dependence on age for all subtypes. Acute lymphocytic leukemia (ALL) is the most common type of leukemia in young children, representing approximately 75 percent of all pediatric leukemias. This disease can also affect adults, especially those ages 65 and older. Incidence of a different subtype, acute myleloid leukemia (AML) generally increases with age, and is the predominant form after age 25 (ACS, 2008).

No single factor has been shown to cause all leukemias. Genetic factors, drugs, and environmental and occupational exposures have each been implicated in both children and adults. Occurrence of multiple cases of leukemia within the same family, particularly among siblings, is one source of evidence of a genetic etiology for ALL and AML. Studies of twins with leukemia also suggest a genetic factor, but do not rule out an intrauterine event or exposure. Other evidence of a genetic predisposition to develop acute leukemia is suggested by the increased leukemia incidence associated with a number of hereditary or congenital disorders. Most notable among congenital disorders is Down syndrome. Both childhood acute leukemia and Down syndrome have similar risk factors (prenatal radiation exposure, older maternal age at birth, abnormal maternal reproductive history). Individuals of Hispanic origin, particularly those born in Mexico, are also at increased risk of developing leukemia. In this study, two cases occurred in Mexican nationals. One individual had previously had another tumor removed (within the past five years).

Nonionizing electromagnetic radiation from overhead power lines, home electrical wiring and home appliances has been weakly associated with childhood leukemia in some studies, although study results have been somewhat inconsistent. Interpretation of these studies is difficult because of limitations in exposure assessment, other methodological problems, and the small numbers of cases (Belson, 2007).

An increased incidence of AML has been reported in patients who have received chemotherapy for other disorders. Exposure to a combination of chemotherapy and radiotherapy further increases affected patient's risk of developing leukemia (Smith et al., 1996). Exposure to even moderate doses of ionizing radiation, either weapons or therapy related, appears to be associated with an increased risk for developing leukemia (Stevens et al., 1990; Adamson and Seiber, 1981).

A recent review of the findings of NCI's ongoing Agricultural Health Study confirmed a previously reported finding in pesticide applicators between exposure to the organochlorine insecticides chlordane and heptachlor and an increased risk of leukemia (Purdue, 2006).

There is a well established causal association between benzene and other combustion by-products such as 1,3-butadiene, which commonly occur in ambient air from air pollution and other industrial sources, and increased risk of leukemia. Other important non-occupational sources of benzene exposure may be cigarette smoke and consumer products. Heavy cigarette smoking has been associated with the development of certain histologic types of leukemia (ALL and AML) in adults, and small excesses have been reported among children of mothers who smoked during pregnancy (Severson et al. 1992). In this study, sixty-seven percent of males diagnosed with leukemia in Area 2 for the time period 1997-2005 had a history of smoking.

Childhood ALL has been associated with parental occupation in chemical and other industries. Non-specific prenatal occupational exposure to pesticides and maternal

employment in agriculture have been associated with increased risk of childhood ALL, as has postnatal residential exposure to pesticides (Belson, 2007).

Occupational exposure to benzene from diesel exhaust and other sources is recognized as an important risk factor for increased incidence of AML (Richardson, 2008). In the current study, six of 19 male leukemia cases in Area 2 had AML and five of the six were truck drivers, with likely exposure to benzene from diesel exhaust. Review of medical abstracts indicated that only twelve of the 19 cases had smoking status listed, but eight of the twelve were smokers. Studies have indicated that smokers have about a 30-40% increased risk of developing leukemia (Brownson, 1993).

Cancer of the Small Intestine

A statistically significant elevation in cancer of the small intestine was reported for 1997-2005 in Area 3 only. All except one of the eight cases diagnosed in Area 3 occurred in males, with an O/E ratio of 3.93 (7 cases diagnosed compared to 1 or 2 expected cases). Two cases were diagnosed in individuals under age 45, but the number of cases was too small to conduct significance testing. The youngest male case (in the age group 25-34) had a family history of colon cancer. There is some evidence that small bowel tumors have some risk factors in common with colon cancer (Chow 1996). Presence of either colon cancer or adenocarcinoma of the small intestine is associated with an increased risk of developing the other type of cancer. Adenocarcinomas of both sites are associated with familial adenomatous polyposis (FAP). The average age at diagnosis is about 60 (ACS, 2008).

Incidence of this relatively rare disease is believed to be unrelated to tobacco or alcohol consumption (Chow, 1993). People with celiac disease, Crohns disease and certain other inherited conditions, such as FAP and Lynch syndrome, are at increased risk for cancer of the small intestine. No established risk factors for cancer of the small intestine related to environmental or occupational exposure were identified in the epidemiological literature.

Nasopharyngeal Cancer

Cancer of the nasopharynx is a rare disease in the general U.S. population and most cases have an unknown etiology. Nasophayyngeal cancer is known to occur at much higher rates in Southeast Asia, parts of southern China, and in Inuit natives of Alaska and Greenland. Early childhood exposure to N-Nitrosodimethylamine in salt-cured fish and meat has been proposed as the likely causative agent in this high-risk subpopulation (Cheng, 2001). The Epstein-Barr virus is also a proven causative agent for nasopharyngeal cancer, and genetic susceptibility may play a role in a small number of cases (ACS, 2008). In addition, several occupations have been associated with higher rates of nasopharyngeal cancer, including workplace exposure to formaldehyde, certain aromatic hydrocarbons, and dust or smoke particulate. Nasopharyngeal cancer has also been associated in some occupational studies with metal work. Risk factors may include exposure to metal dust (grinding) or exposure to cutting oils (Siemiatycki, 2004).

A significant history of alcohol consumption is associated with 80-85% of all head and neck cancers. However, according to recent data from the National Cancer Institute (NCI), tobacco smoke and alcohol are not believed to be significant risk factors for cancer of the nasopharynx (NCI, 2008).

In the current study, cancer of the nasopharynx was statistically high in one area, Area 3, in both males and males and females combined, with 5 of the 6 diagnosed cases occurring in males. Additional case review determined that two cases occurred in individuals from Southeast Asia, a strong risk factor for nasopharyngeal cancer, and two of the remaining adult cases had automobile service jobs, an occupation associated with increased risk of nasopharyngeal cancer (Siemiatycki, 2004). Two cases occurred in a relatively young age group (ages 10-14). One child was listed as having fetal alcohol syndrome: however, no association was identified in the literature between FAS and increased risk of developing cancer. The mothers of both of the two younger cases had a past history of cancer, which could indicate a family predisposition to cancer.

Multiple Comparisons Assessment

The evaluation of the statistical outcome of the independent multiple comparisons made in this analysis predicted that 2.5 percent of the comparisons made would be statistically significantly high, and 2.5 percent statistically significantly low. Among the independent comparisons made, 5.6 percent were high and 3.2 percent were low. Since six of the 12 statistically high ratios were among cancer sites with strong smoking connections (e.g. lung, larynx, and bladder cancer) and Cancer Registry abstracts documented positive smoking histories in most study area patients with these cancers, recalculating the percentage of remaining high ratios (six out of 210 tests) results in 2.9 percent of the ratios being statistically high. This outcome does not suggest an overall marked departure from that predicted. Note that ratios based on less than three cases were not tested statistically, which likely partially accounts for the low percentage of the number of statistically lower than expected tests found.

Study Limitations

The current cancer incidence evaluation for residents living in the vicinity of the RMA used a standard ecological study design. A well-recognized limitation of this type of study is that surveillance data are analyzed at the group level, rather than for the individual. Reliable data are typically not available, or are incomplete, for critical exposure variables, such as individual estimates or measures of exposure, individual-level data about length of residence, in- and out-migration, and other exposures inside or outside the home. Geographic area is used as a surrogate for individual exposure status, and each individual diagnosed with cancer at a residence within the study area is presumed to have resided within the study area for a sufficient amount of time to account for the time that would elapse between a given environmental exposure and the clinical diagnosis of cancer. Inherent in this study design is that information on some potential confounders may be lacking and cannot be easily controlled for with this study design. For instance, it is not possible to control for the influence of common carcinogenic exposure such as traffic-related exposure to benzene or other industrial influences within

a given study boundary.

This study was able to control for potential confounding due to population differences in age and sex, by calculating age- and sex-adjusted tumor rates. In addition, medical abstracts were reviewed for each case among each age-gender specific grouping with a statistically elevated number of cancers. For many cases, potential confounders or individual risk factors such as smoking, occupational exposure to known carcinogens, and other predisposing conditions were identified. However, the available data do not provide a complete history for each individual. For this reason, studies such as this one cannot be used to draw conclusions about causal association but are considered to be hypothesis generating and are valuable for exploring issues that may warrant additional investigation.

Comparison with Past Studies

No obvious patterns or trends in cancer occurrence over time were identified (see Table 3). Statistically high numbers of cancers reported in the previous post-remediation study for some individual cancer types (lung, pancreas, lymphoma and brain) did not persist in the current study, with the exception of lung cancer in combined Area 1. Past surveys have identified higher smoking rates in the study area than is typical for the rest of the Denver metropolitan area. Review of case abstract data for the current study confirmed that most individuals diagnosed with lung cancer were smokers.

In the current study, stomach cancer and leukemia were statistically elevated in Area 2. Both of these types of cancers were elevated previously in the baseline study for the time period 1979-1996. The findings, however, were not consistent across time periods. Stomach cancer was statistically elevated in males only during both time periods, but statistically elevated numbers occurred in different study areas (Area 2 only in the current study; combined Area 1 only in the baseline study). Leukemia findings were not consistent across gender, with statistical elevations in males in the current study and in females in the baseline study.

For the current study, a statistically high number of bladder cancer cases was reported in males in Area 1b. This finding was also reported for the baseline time period (1979-1996), but not for the previous post-remediation study (1997-2000). A 1993 follow-up study of bladder cancer in Adams County, Colorado (CDPHE, 1996), found that a history of bladder infection and smoking were significant risk factors for cases for the period 1982 through part of 1991. Smoking is recognized as a primary risk factor for bladder cancer, accounting for as many as 60 percent of all cases. Case abstract reviews confirmed a higher rate of smoking in cases diagnosed in the study area for the time period 1997-2005 than was typical for the control population, which is consistent with findings reported in previous studies.

CONCLUSIONS

Statistical elevations in cancer incidence identified in the current study varied across location, gender, race and time. Cancer incidence was not statistically elevated in both genders for any of the three geographic areas studied. A finding of statistically high ratios of cancer in only one gender is generally considered an inconsistency when investigating environmental exposures, making it less likely that cancer outcomes were caused by a common environmental agent in the ambient environment.

For nine of the 30 individual types of cancer studied (lung, colorectal, stomach, small intestine, nasopharynx, bladder, larynx, leukemia and extrahepatic bile duct/gallbladder), the number of diagnosed cases exceeded the statistical range for the expected number of cases in one gender only. Statistical elevations were reported for multiple types of cancers, without one type predominating. A common environmental cause would be more likely when several cases of the same type of cancer occur and that type of cancer is not common in the general population.

Cancer outcomes from the current study were also compared to previous data reviews, including the October 2003 post-remediation study, and no obvious patterns or historical trends were identified.

Smoking is an important contributing risk factor for many of the cancer sites where an elevated relative risk was identified (lung, bladder, larynx, colorectal, leukemia, stomach), and is likely the predominant cause of the elevated cancer findings reported in this study, particularly for lung, bladder, and larynx. The possibility of some interaction effect from exposure to other risk co-factors, such as exposure to carcinogens in an occupational setting or other chemical exposures indoors or in the outdoor ambient air environment, cannot be ruled out by this analysis, but any such effect would likely be small compared to the smoking effect for these cancer types. All cancer types that were statistically elevated in Area 2 (lung, larynx, stomach and AML leukemia) have a well-established association with increased risk in smokers. Additional review of individual case-level medical records confirmed a higher rate of smoking in individuals diagnosed with several types of smoking-related cancers than was typical in the comparison population.

For the remaining sites for which a statistically high number of cancers was identified- gallbladder and other biliary, nasopharynx, and small intestine- smoking has not been strongly associated in the scientific literature with elevated cancer risk. The results for each of these cancers are based on a very small number of cases for each site, resulting in generally weak associations with wide confidence intervals. This greatly limits the ability to make firm conclusions about the significance of the findings.

Review of other available case information (smoking history, alcohol use, occupation, predisposing genetic factors and family history of cancer) was conducted for cancer types that occurred in statistically high numbers in some subpopulations. Case investigation generally identified typical risk profiles that partially explain the slight to moderate

increased risk reported in this study for a variety of cancer types. Identification of other contributing risk factors at the individual case level cannot be fully assessed with this study, but the presence of known risk factors further weakens the likelihood of a common environmental exposure.

Inherent in this study design is the limitation that information on some potential confounders may be lacking and cannot be easily controlled for with this study design. For instance, it is not possible to control for the influence of common carcinogenic exposure such as traffic-related exposure to benzene or other industrial influences within the study boundary. Other lifestyle factors, such as poor diet, lack of physical activity or being overweight or obese, are recognized risk factors for several of the cancers that occurred in statistically high numbers in this study. Many studies have shown that exposure to asbestos, benzene, benzidine, cadmium, nickel, vinyl chloride and other chemicals in the workplace can cause cancer. People who have certain jobs (such as painters, construction workers, and those in the chemical industry) may have an increased risk of cancer which can not be accounted for. It is not possible with this study to definitively determine an association, or lack of association, with past RMA-related exposures. Review of other available case information (smoking history, occupation, histology) and the general occurrence of statistical elevations in only a single gender and in only one geographic area, suggest other factors, particularly smoking and workplace exposures.

A significant source of uncertainty for this study was the large and rapid shifts in population that have occurred in the vicinity of the RMA over time. Available population estimates indicated an increase in total population of 576% and 32% in Area 1a and Area 3, respectively, from 2000-2005. But detailed age, gender and race/ethnicity adjusted estimates were not available for all study areas. Age adjustment is particularly critical for any investigation of cancer outcomes, because cancer is largely a disease of older persons, particularly for certain types of cancers, with about 77% of all cancers being diagnosed in individuals age 55 and older (ACS, 2008). Uncertainty introduced into some of the statistical analyses performed in this study by such dynamic, but currently uncharacterized, demographic fluctuations may be particularly difficult to interpret for cancers that occur in small numbers, such as nasopharynx, other biliary tract and small intestine. Area 2 population change was not substantial, but change in demographic composition from in- and out-migration since the 2000 Census was conducted is an unknown effect, leading to uncertainty in the precision of the age, gender and race composition in this area. Although best methods available were applied at the time this study was done to calculate expected cancer rates, it will not be possible to fully evaluate this population effect until the 2010 Census data are available.

In this study, many statistical tests were carried out and it is expected that some of them would be statistically high or low by chance alone. Tumor rates are quite variable in small populations and rarely match the overall average rate for a larger area, such as the state or the greater Denver metropolitan area. For any given time period, some subpopulations have rates above the overall rate and others have rates below the overall rate, so that even when there is an excess of cancer cases reported, this may be consistent

with expected random variation. For this reason, it is important to evaluate statistically elevated findings within the broader context of overall patterns and consistency over location, time, demographic characteristics and cancer type.

The time period of this study was selected to coincide with soil cleanup activities at the RMA. Cancer cases diagnosed from 1997-2005 are not likely to be related to cleanup activities because focused air monitoring of 27 RMA-related chemicals has not shown ongoing or significant off-site release that would cause significant exposure or increased risk of cancer to surrounding communities for RMA chemicals. In addition, the time it takes for cancer to develop and be diagnosed (latency period) is generally believed to be longer than the time elapsed since cleanup began.

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 covers the period 1997-2005, a time period beginning just after the initiation of the arsenal soil remediation activities. The following activities are recommended to address the findings and uncertainties discussed in the current study:

1. Produce an addendum to the current study when the 2010 U.S. Census population data become available, to improve estimates of study area population counts, and age, race/ethnicity and gender distributions. Compile the updated surveillance report using the last complete year of cancer data available from the state Cancer Registry at the time the 2010 census population data are released.

2. Post the addendum to the current study on the state RMA web page, for consideration during the five-year site review process.

3. Communicate the findings of this report to the Comprehensive Cancer Control Section at the state health department, local health departments serving the study area and affected neighborhood groups, to provide risk prevention information and to improve cancer control strategies in the northeast Denver metropolitan area, e.g. obesity prevention and, particularly smoking cessation.

REFERENCES

Adamson RH, Seiber, SM. Chemically Induced Leukemia in Humans. Environ Health Perspect 39:93-103, 1981.

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

American Cancer Society (ACS). Cancer Facts and Figures 2008. Atlanta: American Cancer Society.

Amoateng-Adjepong Y, Sathiakumar N, Delzell E, et al. Mortality among Workers at a Pesticide Manufacturing Plant. JOEM, 37(4):471-478. April 1995.

Agency for Toxic Substances and Disease Registry (ATSDR). Public Health Assessment for the Rocky Mountain Arsenal, Adams County, Colorado. CERCLIS No. CO5210020769. Division of Health Assessment and Consultation. September 30, 1996.

Agency for Toxic Substances and Disease Registry (ATSDR). ToxFAQs for n-Nitrosodimethylamine. CAS# 62-75-9. July 1999.

Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Cancer and Smoking.

Bailar JC, Ederer F. Significance Factors for the Ratio of a Poisson Variable to its Expectation. Biometrics 20(3): 639-643, 1964.

Belson M, Kingsley B, Holmes A. Risk Factors for Acute Leukemia in Children: A Review. Environmental Health Perspectives. Jan 2007; 115(1):138-145.

Boffetta P. Epidemiology of Environmental and Occupational Cancer. Oncogene 2004; 23:6392-6403.

Botteri E, Iodice S, Bagnardi V, et al. Smoking and colorectal cancer: a meta-analysis. JAMA. 2008 Dec 17; 300(23):2765-78.

Brownson RC, Novotny TE, Perry MC. Cigarette smoking and adult leukemia. Arch Intern Med. 1993; Feb. 22; 153(4):425-427.

Chao A, Thun MJ, Jacobs, EJ, et al. Cigarette smoking and colorectal cancer mortality in the cancer prevention study II. J Natl Cancer Inst. 2000; 92(23):1888-1896.

Cheng H. Nasopharyngeal Cancer and the Southeast Asian Patient. American Family Physician. May 1, 2001.

Chow JS, Chen CC, Ahsan H, et al. A Population-Based Study of the Incidence of Malignant Small Bowel Tumours: SEER, 1973-1990. International Journal of Epidemiology. 1996; 25(4):722-728.

Chow WH, Linet MS, McLaughlin JK, et al. Risk factors for small intestine cancer. Cancer Causes Control. 1993; Mar 4(2):163-169.

Clapp R, Howe G, Lefevre, MJ. *Environmental and Occupational Causes of Cancer, A Review of Scientific Literature.* 2005. University of Massachusetts Lowell; pg 1-46.

Clapp RW, Jacobs MM, Loechler EL. *Environmental and Occupational Causes of Cancer, New Evidence, 2005-2007*, University of Massachusetts Lowell; pg 1-41.

Colorado Department of Public Health and Environment (CDPHE). *Cancer Incidence In The Northeastern Denver Metro Area: Report Of The Ad Hoc Panel*. 1993.

Colorado Department of Public Health and Environment (CDPHE). *Analysis of Diagnosed vs. Expected Cancer Cases for the Northeast Denver Metropolitan Area in the Vicinity of the Rocky Mountain Arsenal, 1979-1996.* January 2003.

Colorado Department of Public Health and Environment (CDPHE). *Analysis of Diagnosed vs. Expected Cancer Cases for the Northeast Denver Metropolitan Area in the Vicinity of the Rocky Mountain Arsenal, 1997-2000.* October 2003.

De Stefani E, Oreggia F, Rivero S, et al. Salted meat consumption and risk of laryngeal cancer. Eur J Epidemiol. 1995 Apr; 11(2):177-80.

Doll R, Peto R, Wheatley K, et al. Mortality in Relation to Smoking: 40 years' Observations of Male British Doctors. Br Med J. 1994; 309:901-911.

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 87. Inorganic and Organic Lead Compounds. World Health Organization. International Agency for Research on Cancer. Lyon, France. 2006.

Larsson SC, Wolk A. Obesity and the risk of gallbladder cancer: a meta-analysis. British Journal of Cancer. 2007; 96:1457-1461.

Lynge E, Anttila A, Hemminiki K. Organic solvents and cancer. Cancer Causes and Control. 1997; 8:406-419.

National Cancer Institute. PDQ Fact Sheet: Nasopharyngeal Cancer. 2008.

Prince MM, Ruder, AM, Hein MJ, et al. Mortality and exposure response among 14,458 electrical capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). Environmental Health Perspectives. 2006; 114(10):1508-1514.

Purdue MP, Hoppin JA, Blair A, et al. Occupational exposure to organochlorine insecticides and cancer incidence in the Agricultural Health Study. International Journal of Cancer. 2006; 120:642-649.

Randi G, Franceschi S, La Vecchia C. Gallbladder cancer worldwide; geographical distribution and risk factors. Mini Review: International Journal of Cancer. 2006; 118:1591-1602.

Randi G, Malvezzi M, Levi F, et al. Epidemiology of biliary tract cancers: an update. Annals of Oncology. 2009; 20(1):146-159.

Riboli E, Kaaks R, Esteve J. Nutrition and laryngeal cancer. Cancer Causes and Control. 1996 Jan; 7(1):147-156.

Richardson DB. Temporal Variation in the Association between Benzene and Leukemia Mortality. Environmental Health Perspectives. March 2008; 116(3):370-374.

Samanic D, Ruisiecki J, Dosemeci M, et al. Cancer incidence among pesticide applicators exposed to dicamba in the Agricultural Health Study. Environmental Health Perspectives. 2006; 114(10):1521-1526.

Severson RK, Buckley JD, Woods WG, et al. Cigarette Smoking and Alcohol Consumption by Parents of Children with Acute Myeloid Leukemia: An Analysis within Morphorlogical Subgroups-A Report from the Children's Cancer Group. Cancer Epidemiol. Biomakers Prev. 1992; 2:433-439.

Siemiatycki J, Richardson L, Straif K, et.al. Listing Occupational Carcinogens. Environmental Health Perspectives. November 2004; 112(15):1447-59.

Smith MA, McCaffrey RP, Karp JE. The Secondary Leukemias: Challenges and Research Directions. J Natl Cancer Inst 1996; 88:407-418.

Stayner LT, Dannenberg AL, Bloom T, Thun M. Excess hepatobiliary cancer mortality among munitions workers exposed to dinitrotoluene. J Occup Med. 1983;35:291-298.

Stevens W, Thomas DC, Lyon J, et al. Leukemia in Utah and Radioactive Fallout from the Nevada test site. JAMA 1996; 264:585-591.

Talley NJ, Zinsmeister AW, Dimagno EP, et al. Gastric Adenocarcinoma and *Helicobacter pylori* Infection. J Natl Cancer Inst 1991; 83:1734-1738.

Thun MJ, Peto R, Lopez AD, et al. Alcohol Consumption and Mortality Among Middle-Aged and Elderly U.S. Adults. N Engl J Med 1997; 337:1705-1714.

U.S. Environmental Protection Agency (EPA). Fact Sheet on Emerging Contaminant NDMA. EPA505-F-07-006. April 2008.

Villanueva CM, Cantor KP, Grimwalt JO. Bladder Cancer and Exposure to Water Disinfection By-products through Ingestion, Bathing, Showering, and Swimming Pools. American Journal of Epidemiology. 2006; 165(2): 148-156.

Wynder EL. The Past, Present, and Future of the Prevention of Lung Cancer. Cancer Epidemiol Biomarkers Prev. 1998; 7:735-748.

Zhao Y, Krishnadasan A, Kennedy N, et al. Estimated effects of solvents and mineral oils on cancer incidence and mortality in a cohort of aerospace workers. American Journal of Industrial Medicine. 2005; 48:249-258.

APPENDIX 1

UPDATE OF CANCER INCIDENCE IN RESIDENTS LIVING IN THE VICINITY OF THE ROCKY MOUNTAIN ARSENAL, 1997-2005

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 1997-2005 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-A26 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, 1997-2005 – Males and Females

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	156	179.245	0.87	(0.74-1.03)
Salivary Gland	1	0.476	2.10	NC
Tongue, Mouth & Gum	3	1.687	1.78	(0.37-5.20)
Nasopharynx	1	0.177	5.65	NC
Other Oral & Pharynx	0	1.015	0.00	NC
Esophagus	1	1.703	0.59	NC
Stomach	1	2.176	0.46	NC
Small Intestine	0	0.642	0.00	NC
Colorectal	18	16.237	1.11	(0.66-1.75)
Liver	0	2.092	0.00	NC
Other Biliary	5	0.742	6.74**	(2.18-15.75)
Pancreas	4	3.603	1.11	(0.30-2.84)
Larynx	1	1.234	0.81	NC
Lung & Bronchus	19	17.309	1.10	(0.66-1.72)
Melanoma	11	12.276	0.90	(0.45-1.60)
Bladder	7	6.552	1.07	(0.43-2.20)
Kidney	6	4.671	1.28	(0.47-2.80)
Thyroid	1	4.044	0.25	NC
Other Endocrine	0	0.290	0.00	NC
Brain & Other Nervous System	4	3.097	1.29	(0.35-3.30)
Bones & Joints	0	0.459	0.00	NC
Leukemia	4	4.838	0.83	(0.23-2.12)
Multiple Myeloma	2	1.652	1.21	NC
Lymphoma	7	8.446	0.83	(0.33-1.71)
Soft Tissue	2	1.382	1.45	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 p=0.05 level. (** p=0.01 level)

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, 1997-2005– Males

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	84	90.571	0.93	(0.74-1.15)
Salivary Gland	1	0.276	3.62	NC
Tongue, Mouth & Gum	2	1.127	1.77	NC
Nasopharynx	1	0.129	7.75	NC
Other Oral & Pharynx	0	0.839	0.00	NC
Esophagus	0	1.368	0.00	NC
Stomach	1	1.478	0.68	NC
Small Intestine	0	0.392	0.00	NC
Colorectal	12	8.860	1.35	(0.70-2.36)
Liver	0	1.567	0.00	NC
Other Biliary	3	0.336	8.93**	(1.84-26.11)
Pancreas	3	1.973	1.52	(0.31-4.45)
Larynx	1	0.977	1.02	NC
Lung & Bronchus	13	9.658	1.35	(0.72-2.30)
Melanoma	7	6.806	1.03	(0.41-2.12)
Prostate	19	26.744	0.71	(0.43-1.11)
Testis	0	1.638	0.00	NC
Bladder	4	5.045	0.79	(0.22-2.03)
Kidney	2	3.118	0.64	NC
Thyroid	0	1.071	0.00	NC
Other Endocrine	0	0.185	0.00	NC
Brain & Other Nervous System	3	1.827	1.64	(0.34-4.80)
Bones & Joints	0	0.266	0.00	NC
Leukemia	3	2.875	1.04	(0.22-3.05)
Multiple Myeloma	0	1.040	0.00	NC
Lymphoma	3	4.785	0.63	(0.13-1.83)
Soft Tissue	2	0.803	2.49	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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A3 – Number of Cancer Diagnoses Compared to the Expected Number in Area 1a, 1997-2005 – Females

	Cancers Diagnosed	Cancers Expected	Diagnosed/ Expected	95% C.I. for Ratio
All Cancers	72	88.674	0.81	(0.64-1.01)
Salivary Gland	0	0.200	0.00	NC
Tongue, Mouth & Gum	1	0.560	1.79	NC
Nasopharynx	0	0.048	0.00	NC
Other Oral & Pharynx	0	0.176	0.00	NC
Esophagus	1	0.335	2.99	NC
Stomach	0	0.698	0.00	NC
Small Intestine	0	0.250	0.00	NC
Colorectal	6	7.377	0.81	(0.30-1.77)
Liver	0	0.525	0.00	NC
Other Biliary	2	0.406	4.93	NC
Pancreas	1	1.630	0.61	NC
Larynx	0	0.257	0.00	NC
Lung & Bronchus	6	7.651	0.78	(0.29-1.71)
Melanoma	4	5.470	0.73	(0.20-1.87)
Female Breast	20	34.505	0.58*	(0.35-0.90)
Cervix	2	1.681	1.19	NC
Uterus	4	3.981	1.00	(0.27-2.57)
Ovary	2	3.145	0.64	NC
Bladder	3	1.507	1.99	(0.41-5.82)
Kidney	4	1.553	2.58	(0.70-6.59)
Thyroid	1	2.973	0.34	NC
Other Endocrine	0	0.105	0.00	NC
Brain & Other Nervous System	1	1.270	0.79	NC
Bones & Joints	0	0.193	0.00	NC
Leukemia	1	1.963	0.51	NC
Multiple Myeloma	2	0.612	3.27	NC
Lymphoma	4	3.661	1.09	(0.30-2.80)
Soft Tissue	0	0.579	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 p=0.05 level. (** p=0.01 level)

NC=not calculated due to less than 3 diagnoses (see text for explanation)

Table A4 – Number of Cancer Diagnoses Compared to the Expected Number in Area 1b, 1997-2005 – Males and Females

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	263	261.673	1.01	(0.89-1.13)
Salivary Gland	0	0.752	0.00	NC
Tongue, Mouth & Gum	3	2.258	1.33	(0.27-3.89)
Nasopharynx	0	0.277	0.00	NC
Other Oral & Pharynx	1	1.400	0.71	NC
Esophagus	1	2.526	0.40	NC
Stomach	4	3.819	1.05	(0.29-2.68)
Small Intestine	1	1.030	0.97	NC
Colorectal	40	26.073	1.53*	(1.10-2.05)
Liver	5	3.770	1.33	(0.43-3.10)
Other Biliary	1	1.407	0.71	NC
Pancreas	10	5.925	1.69	(0.81-3.10)
Larynx	2	1.957	1.02	NC
Lung & Bronchus	48	27.216	1.76**	(1.30-2.34)
Melanoma	8	14.295	0.56	(0.24-1.10)
Bladder	14	9.601	1.46	(0.80-2.45)
Kidney	7	7.238	0.97	(0.39-1.99)
Thyroid	3	5.247	0.57	(0.12-1.67)
Other Endocrine	0	0.417	0.00	NC
Brain & Other Nervous System	8	4.395	1.82	(0.78-3.58)
Bones & Joints	1	0.721	1.39	NC
Leukemia	8	7.182	1.11	(0.48-2.19)
Multiple Myeloma	3	2.798	1.07	(0.22-3.14)
Lymphoma	9	12.558	0.72	(0.33-1.36)
Soft Tissue	0	2.055	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 p=0.05 level. (** p=0.01 level)

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, 1997-2005 – Males

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	139	133.042	1.04	(0.88-1.25)
Salivary Gland	0	0.445	0.00	NC
Tongue, Mouth & Gum	1	1.472	0.68	NC
Nasopharynx	0	0.196	0.00	NC
Other Oral & Pharynx	1	1.139	0.88	NC
Esophagus	1	1.954	0.51	NC
Stomach	0	2.396	0.00	NC
Small Intestine	0	0.597	0.00	NC
Colorectal	23	14.099	1.63*	(1.03-2.45)
Liver	4	2.770	1.44	(0.39-3.69)
Other Biliary	0	0.603	0.00	NC
Pancreas	6	3.206	1.87	(0.69-4.08)
Larynx	2	1.529	1.31	NC
Lung & Bronchus	30	15.009	2.00**	(1.35-2.86)
Melanoma	4	8.103	0.49	(0.14-1.26)
Prostate	21	38.023	0.55**	(0.34-0.84)
Testis	1	2.166	0.46	NC
Bladder	14	7.443	1.88*	(1.03-3.16)
Kidney	6	4.625	1.30	(0.48-2.83)
Thyroid	2	1.372	1.46	NC
Other Endocrine	0	0.269	0.00	NC
Brain & Other Nervous System	5	2.555	1.96	(0.63-4.57)
Bones & Joints	0	0.400	0.00	NC
Leukemia	4	4.208	0.95	(0.26-2.43)
Multiple Myeloma	2	1.724	1.16	NC
Lymphoma	2	7.020	0.28	NC
Soft Tissue	0	1.179	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 p=0.05 level. (** p=0.01 level)

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, 1997-2005 – Females

	Cancers Diagnosed	Cancers Expected	Diagnosed/ Expected	95% C.I. for Ratio
All Cancers	124	128.631	0.96	(0.80-1.14)
Salivary Gland	0	0.307	0.00	NC
Tongue, Mouth & Gum	2	0.786	2.54	NC
Nasopharynx	0	0.081	0.00	NC
Other Oral & Pharynx	0	0.261	0.00	NC
Esophagus	0	0.572	0.00	NC
Stomach	4	1.423	2.81	(0.77-7.19)
Small Intestine	1	0.433	2.31	NC
Colorectal	17	11.974	1.42	(0.83-2.27)
Liver	1	1.000	1.00	NC
Other Biliary	1	0.804	1.24	NC
Pancreas	4	2.719	1.47	(0.40-3.76)
Larynx	0	0.428	0.00	NC
Lung & Bronchus	18	12.207	1.47	(0.87-2.33)
Melanoma	4	6.192	0.65	(0.18-1.65)
Female Breast	33	47.129	0.70*	(0.48-0.99)
Cervix	3	2.642	1.14	(0.23-3.32)
Uterus	5	5.663	0.88	(0.29-2.06)
Ovary	4	4.586	0.87	(0.24-2.23)
Bladder	0	2.158	0.00	NC
Kidney	1	2.613	0.38	NC
Thyroid	1	3.875	0.26	NC
Other Endocrine	0	0.148	0.00	NC
Brain & Other Nervous System	3	1.840	1.63	(0.34-4.77)
Bones & Joints	1	0.321	3.12	NC
Leukemia	4	2.974	1.35	(0.37-3.44)
Multiple Myeloma	1	1.074	0.93	NC
Lymphoma	7	5.538	1.26	(0.51-2.61)
Soft Tissue	0	0.876	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 p=0.05 level. (** p=0.01 level)

NC=not calculated due to less than 3 diagnoses (see text for explanation)

Table A7 – Number of Cancer Diagnoses Compared to the Expected Number in Area 1 Combined , 1997-2005 – Males and Females

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	419	440.918	0.95	(0.86-1.04)
Salivary Gland	1	1.228	0.81	NC
Tongue, Mouth & Gum	6	3.944	1.52	(0.56-3.31)
Nasopharynx	1	0.455	2.20	NC
Other Oral & Pharynx	1	2.415	0.41	NC
Esophagus	2	4.229	0.47	NC
Stomach	5	5.995	0.83	(0.27-1.95)
Small Intestine	1	1.672	0.60	NC
Colorectal	58	42.309	1.37*	(1.04-1.80)
Liver	5	5.862	0.85	(0.28-1.99)
Other Biliary	6	2.149	2.79*	(1.02-6.08)
Pancreas	14	9.528	1.47	(0.80-2.47)
Larynx	3	3.190	0.94	(0.19-2.75)
Lung & Bronchus	67	44.525	1.50**	(1.17-1.93)
Melanoma	19	26.571	0.72	(0.44-1.12)
Bladder	21	16.152	1.30	(0.80-1.99)
Kidney	13	11.909	1.09	(0.58-1.87)
Thyroid	4	9.291	0.43	(0.12-1.10)
Other Endocrine	0	0.707	0.00	NC
Brain & Other Nervous System	12	7.492	1.60	(0.83-2.80)
Bones & Joints	1	1.181	0.85	NC
Leukemia	12	12.019	1.00	(0.51-1.74)
Multiple Myeloma	5	4.450	1.12	(0.36-2.63)
Lymphoma	16	21.005	0.76	(0.44-1.24)
Soft Tissue	2	3.437	0.58	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 p=0.05 level. (** p=0.01 level)

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, 1997-2005 – Males

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	223	223.613	1.00	(0.87-1.14)
Salivary Gland	1	0.721	1.39	NC
Tongue, Mouth & Gum	3	2.598	1.15	(0.24-3.38)
Nasopharynx	1	0.325	3.08	NC
Other Oral & Pharynx	1	1.978	0.51	NC
Esophagus	1	3.322	0.30	NC
Stomach	1	3.874	0.26	NC
Small Intestine	0	0.989	0.00	NC
Colorectal	35	22.958	1.52*	(1.06-2.12)
Liver	4	4.337	0.92	(0.25-2.36)
Other Biliary	3	0.939	3.19	(0.66-9.34)
Pancreas	9	5.179	1.74	(0.80-3.30)
Larynx	3	2.506	1.20	(0.25-3.50)
Lung & Bronchus	43	24.667	1.74**	(1.26-2.35)
Melanoma	11	14.909	0.74	(0.37-1.32)
Prostate	40	64.767	0.62**	(0.44-0.84)
Testis	1	3.803	0.26	NC
Bladder	18	12.488	1.44	(0.85-2.28)
Kidney	8	7.743	1.03	(0.45-2.03)
Thyroid	2	2.443	0.82	NC
Other Endocrine	0	0.454	0.00	NC
Brain & Other Nervous System	8	4.383	1.83	(0.79-3.59)
Bones & Joints	0	0.667	0.00	NC
Leukemia	7	7.083	0.99	(0.40-2.04)
Multiple Myeloma	2	2.764	0.72	NC
Lymphoma	5	11.805	0.42*	(0.14-0.99)
Soft Tissue	2	1.982	1.01	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 p=0.05 level. (** p=0.01 level)

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, 1997-2005 – Females

	Cancers Diagnosed	Cancers Expected	Diagnosed/ Expected	95% C.I. for Ratio
All Cancers	196	217.305	0.90	(0.78-1.04)
Salivary Gland	0	0.507	0.00	NC
Tongue, Mouth & Gum	3	1.346	2.23	(0.46-6.52)
Nasopharynx	0	0.130	0.00	NC
Other Oral & Pharynx	0	0.437	0.00	NC
Esophagus	1	0.907	1.10	NC
Stomach	4	2.121	1.89	(0.51-4.82)
Small Intestine	1	0.683	1.46	NC
Colorectal	23	19.351	1.19	(0.75-1.79)
Liver	1	1.525	0.66	NC
Other Biliary	3	1.210	2.48	(0.51-7.25)
Pancreas	5	4.349	1.15	(0.37-2.69)
Larynx	0	0.684	0.00	NC
Lung & Bronchus	24	19.858	1.21	(0.78-1.80)
Melanoma	8	11.662	0.69	(0.30-1.35)
Female Breast	53	81.634	0.65**	(0.49-0.84)
Cervix	5	4.323	1.16	(0.37-2.70)
Uterus	9	9.643	0.93	(0.43-1.77)
Ovary	6	7.732	0.78	(0.28-1.69)
Bladder	3	3.664	0.82	(0.17-2.40)
Kidney	5	4.166	1.20	(0.39-2.80)
Thyroid	2	6.848	0.29	NC
Other Endocrine	0	0.253	0.00	NC
Brain & Other Nervous System	4	3.109	1.29	(0.35-3.29)
Bones & Joints	1	0.514	1.95	NC
Leukemia	5	4.936	1.01	(0.33-2.37)
Multiple Myeloma	3	1.686	1.78	(0.37-5.20)
Lymphoma	11	9.200	1.20	(0.60-2.14)
Soft Tissue	0	1.455	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 p=0.05 level. (** p=0.01 level)

NC=not calculated due to less than 3 diagnoses (see text for explanation)

Table A10 – Number of Cancer Diagnoses Compared to the Expected Number in Area 2, 1997-2005 – Males and Females

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	603	623.763	0.97	(0.89-1.04)
Salivary Gland	0	1.804	0.00	NC
Tongue, Mouth & Gum	5	5.274	0.95	(0.31-2.22)
Nasopharynx	1	0.631	1.58	NC
Other Oral & Pharynx	3	3.251	0.92	(0.19-2.70)
Esophagus	5	6.009	0.83	(0.27-1.94)
Stomach	17	9.508	1.79*	(1.04-2.86)
Small Intestine	3	2.513	1.19	(0.25-3.49)
Colorectal	71	64.251	1.11	(0.87-1.38)
Liver	8	9.073	0.88	(0.38-1.74)
Other Biliary	2	3.643	0.55	NC
Pancreas	20	14.729	1.36	(0.83-2.10)
Larynx	8	4.670	1.71	(0.74-3.37)
Lung & Bronchus	87	66.539	1.31*	(1.05-1.62)
Melanoma	16	31.849	0.50**	(0.29-0.82)
Bladder	20	22.915	0.87	(0.53-1.35)
Kidney	23	17.363	1.32	(0.84-1.99)
Thyroid	8	11.972	0.67	(0.29-1.32)
Other Endocrine	0	0.958	0.00	NC
Brain & Other Nervous System	9	10.239	0.88	(0.40-1.67)
Bones & Joints	0	1.680	0.00	NC
Leukemia	25	17.235	1.45	(0.94-2.14)
Multiple Myeloma	4	6.933	0.58	(0.16-1.48)
Lymphoma	33	29.995	1.10	(0.76-1.55)
Soft Tissue	5	4.908	1.02	(0.33-2.38)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A11 – Number of Cancer Diagnoses Compared to the Expected Number in Area 2, 1997-2005 – Males

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	319	309.474	1.03	(0.92-1.15)
Salivary Gland	0	1.054	0.00	NC
Tongue, Mouth & Gum	5	3.350	1.49	(0.48-3.49)
Nasopharynx	1	0.428	2.34	NC
Other Oral & Pharynx	3	2.607	1.15	(0.24-3.37)
Esophagus	5	4.539	1.10	(0.36-2.58)
Stomach	12	5.663	2.12**	(1.09-3.70
Small Intestine	3	1.396	2.15	(0.44-6.28)
Colorectal	39	33.411	1.17	(0.83-1.60)
Liver	6	6.493	0.92	(0.34-2.01)
Other Biliary	0	1.468	0.00	NC
Pancreas	12	7.622	1.57	(0.81-2.75)
Larynx	4	3.600	1.11	(0.30-2.84)
Lung & Bronchus	54	35.533	1.52**	(1.14-1.98)
Melanoma	10	17.932	0.56	(0.27-1.03)
Prostate	60	87.477	0.69**	(0.52-0.88)
Testis	2	4.852	0.41	NC
Bladder	14	17.451	0.80	(0.44-1.35)
Kidney	11	10.740	1.02	(0.51-1.83)
Thyroid	2	3.066	0.65	NC
Other Endocrine	0	0.604	0.00	NC
Brain & Other Nervous System	6	5.843	1.03	(0.38-2.24)
Bones & Joints	0	0.930	0.00	NC
Leukemia	19	9.843	1.93*	(1.16-3.02)
Multiple Myeloma	1	4.156	0.24	NC
Lymphoma	23	16.303	1.41	(0.89-2.12)
Soft Tissue	2	2.758	0.73	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 p=0.05 level. (** p=0.01 level)

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, 1997-2005 – Females

	Cancers Diagnosed	Cancers Expected	Diagnosed/ Expected	95% C.I. for Ratio
All Cancers	284	314.289	0.90	(0.80-1.02)
Salivary Gland	0	0.750	0.00	NC
Tongue, Mouth & Gum	0	1.924	0.00	NC
Nasopharynx	0	0.203	0.00	NC
Other Oral & Pharynx	0	0.644	0.00	NC
Esophagus	0	1.470	0.00	NC
Stomach	5	3.845	1.30	(0.42-3.04)
Small Intestine	0	1.117	0.00	NC
Colorectal	32	30.840	1.04	(0.71-1.47)
Liver	2	2.580	0.78	NC
Other Biliary	2	2.175	0.92	NC
Pancreas	8	7.107	1.13	(0.49-2.22)
Larynx	4	1.070	3.74*	(1.02-9.56)
Lung & Bronchus	33	31.006	1.06	(0.73-1.50)
Melanoma	6	13.917	0.43*	(0.16-0.94)
Female Breast	83	111.860	0.74**	(0.59-0.92)
Cervix	9	6.374	1.41	(0.65-2.68)
Uterus	21	13.602	1.54	(0.95-2.36)
Ovary	9	11.090	0.81	(0.37-1.54)
Bladder	6	5.464	1.10	(0.40-2.39)
Kidney	12	6.623	1.81	(0.93-3.16)
Thyroid	6	8.906	0.67	(0.25-1.47)
Other Endocrine	0	0.354	0.00	NC
Brain & Other Nervous System	3	4.396	0.68	(0.14-1.99)
Bones & Joints	0	0.750	0.00	NC
Leukemia	6	7.392	0.81	(0.30-1.77)
Multiple Myeloma	3	2.777	1.08	(0.22-3.16)
Lymphoma	10	13.692	0.73	(0.35-1.34)
Soft Tissue	3	2.150	1.40	(0.29-4.08)

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 p=0.05 level. (** p=0.01 level)

NC=not calculated due to less than 3 diagnoses (see text for explanation)

Table A13 – Number of Cancer Diagnoses Compared to the Expected Number in Area 3, 1997-2005– Males and Females

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	863	793.943	1.09*	(1.02-1.16)
Salivary Gland	3	2.221	1.35	(0.28-3.95)
Tongue, Mouth & Gum	2	6.739	0.30	NC
Nasopharynx	5	1.141	4.38*	(1.42-10.24)
Other Oral & Pharynx	0	5.511	0.00	NC
Esophagus	9	7.376	1.22	(0.56-2.32)
Stomach	18	12.705	1.42	(0.84-2.24)
Small Intestine	8	3.249	2.46*	(1.06-4.85)
Colorectal	90	74.333	1.21	(0.98-1.49)
Liver	10	15.748	0.64	(0.31-1.17)
Other Biliary	8	4.637	1.73	(0.74-3.40)
Pancreas	18	17.584	1.02	(0.61-1.62)
Larynx	6	6.935	0.87	(0.32-1.89)
Lung & Bronchus	88	78.675	1.12	(0.90-1.39)
Melanoma	15	26.759	0.56*	(0.31-0.93)
Bladder	29	17.731	1.64*	(1.10-2.35)
Kidney	26	23.096	1.13	(0.74-1.65)
Thyroid	21	21.930	0.96	(0.59-1.47)
Other Endocrine	0	2.444	0.00	NC
Brain & Other Nervous System	18	13.187	1.37	(0.81-2.16)
Bones & Joints	1	2.803	0.36	NC
Leukemia	27	19.792	1.36	(0.90-1.99)
Multiple Myeloma	7	14.602	0.48*	(0.19-0.99)
Lymphoma	30	36.483	0.82	(0.56-1.17)
Soft Tissue	10	8.210	1.22	(0.59-2.24)

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 p=0.05 level. (** p=0.01 level)

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, 1997-2005 – Males

	Cancers Diagnosed	Cancers Expected	Diagnosed / Expected	95% C.I. for Ratio
All Cancers	423	402.365	1.05	(0.95-1.16)
Salivary Gland	1	1.134	0.88	NC
Tongue, Mouth & Gum	0	4.848	0.00	NC
Nasopharynx	4	0.908	4.41*	(1.20-11.27)
Other Oral & Pharynx	0	4.541	0.00	NC
Esophagus	9	5.504	1.64	(0.75-3.10)
Stomach	11	8.666	1.27	(0.64-2.27)
Small Intestine	7	1.782	3.93*	(1.58-8.10)
Colorectal	49	40.256	1.22	(0.90-1.61)
Liver	7	11.505	0.61	(0.24-1.25)
Other Biliary	4	1.961	2.04	(0.56-5.22)
Pancreas	10	9.821	1.02	(0.49-1.87)
Larynx	5	5.574	0.90	(0.29-2.10)
Lung & Bronchus	44	46.300	0.95	(0.69-1.28)
Melanoma	9	12.977	0.69	(0.32-1.32)
Prostate	139	124.779	1.11	(0.94-1.33)
Testis	9	7.060	1.27	(0.59-2.42)
Bladder	20	13.783	1.45	(0.89-2.24)
Kidney	15	15.014	1.00	(0.56-1.65)
Thyroid	7	4.336	1.61	(0.65-3.33)
Other Endocrine	0	1.589	0.00	NC
Brain & Other Nervous System	9	7.563	1.19	(0.55-2.26)
Bones & Joints	1	1.657	0.60	NC
Leukemia	14	11.130	1.26	(0.69-2.11)
Multiple Myeloma	3	8.175	0.37	(0.08-1.07)
Lymphoma	16	21.253	0.75	(0.43-1.22)
Soft Tissue	5	4.190	1.19	(0.39-2.79)

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 p=0.05 level. (** p=0.01 level)

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, 1997-2005– Females

	Cancers Diagnosed	Cancers Expected	Diagnosed/ Expected	95% C.I. for Ratio
All Cancers	440	391.578	1.12*	(1.02-1.24)
Salivary Gland	2	1.087	1.84	NC
Tongue, Mouth & Gum	2	1.891	1.06	NC
Nasopharynx	1	0.233	4.29	NC
Other Oral & Pharynx	0	0.970	0.00	NC
Esophagus	0	1.872	0.00	NC
Stomach	7	4.039	1.73	(0.70-3.57)
Small Intestine	1	1.467	0.68	NC
Colorectal	41	34.077	1.20	(0.86-1.63)
Liver	3	4.243	0.71	(0.15-2.07)
Other Biliary	4	2.676	1.49	(0.41-3.82)
Pancreas	8	7.763	1.03	(0.44-2.04)
Larynx	1	1.361	0.73	NC
Lung & Bronchus	44	32.375	1.36	(0.99-1.83)
Melanoma	6	13.782	0.44*	(0.16-0.95)
Female Breast	163	148.080	1.10	(0.94-1.28)
Cervix	17	12.080	1.41	(0.82-2.25)
Uterus	25	16.735	1.49	(0.96-2.20)
Ovary	14	12.622	1.11	(0.61-1.86)
Bladder	9	3.948	2.28**	(1.05-4.33)
Kidney	11	8.082	1.36	(0.68-2.44)
Thyroid	14	17.594	0.80	(0.44-1.34)
Other Endocrine	0	0.855	0.00	NC
Brain & Other Nervous System	9	5.624	1.60	(0.73-3.04)
Bones & Joints	0	1.146	0.00	NC
Leukemia	13	8.662	1.50	(0.80-2.57)
Multiple Myeloma	4	6.427	0.62	(0.17-1.59)
Lymphoma	14	15.230	0.92	(0.50-1.54)
Soft Tissue	5	4.020	1.24	(0.40-2.91)

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 p=0.05 level. (** p=0.01 level)

NC=not calculated due to less than 3 diagnoses (see text for explanation)

Table A16 – Number of Bladder Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 1b, 1997-2005 – Males

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	13	6.069	2.14*	(1.14-3.66)
Hispanic	1	1.223	0.82	NC
Black	0	0.038	0.00	NC
Other	0	0.113	0.00	NC
Age				
35-44	1	0.188	5.32	NC
45-54	1	0.703	1.42	NC
55-64	2	1.460	1.37	NC
65-74	7	2.616	2.68*	(1.07-5.52)
75+	3	2.406	1.25	(0.26-3.65)
Total	14	7.443	1.88*	(1.03-3.16)

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 p=0.05 level. (** p=0.01 level)
NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A17 – Number of Colorectal Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 1, 1997-2005 – Males and Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	39	31.488	1.24	(0.88-1.69)
Hispanic	16	9.109	1.76*	(1.00-2.85)
Black	0	0.216	0.00	NC
Other	3	1.363	2.20	(0.45-6.44)
Age				
25-34	1	0.389	2.57	NC
35-44	3	1.881	1.60	(0.33-4.66)
45-54	6	5.575	1.08	(0.39-2.34)
55-64	19	8.279	2.30**	(1.38-3.59)
65-74	19	13.800	1.38	(0.83-2.15)
75+	10	12.246	0.82	(0.39-1.50)
Total	58	42.309	1.37*	(1.04-1.80)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A18 – Number of Other Biliary Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 1, 1997-2005 – Males and Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	3	1.359	2.21	(0.46-6.46)
Hispanic	3	0.668	4.49	(0.93-13.13)
Black	0	0.024	0.00	NC
Other	0	0.098	0.00	NC
Age				
55-64	1	0.316	3.17	NC
65-74	2	0.700	2.86	NC
75+	3	0.819	3.66	(0.76-10.71)
Total	6	2.149	2.79*	(1.02-6.08)

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 p=0.05 level. (** p=0.01 level)
NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A19 – Number of Lung Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 1, 1997-2005 – Males and Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	56	35.378	1.58**	(1.19-2.07)
Hispanic	11	7.249	1.52	(0.76-2.71)
Black	0	0.416	0.00	NC
Other	0	1.483	0.00	NC
Age				
45-54	5	3.646	1.37	(0.44-3.21)
55-64	23	9.266	2.48**	(1.57-3.73)
65-74	27	18.103	1.49	(0.98-2.17)
75+	12	12.452	0.96	(0.50-1.68)
Total	67	44.525	1.50**	(1.17-1.93)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A20 - Number of Stomach Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 2, 1997-2005 – Males and Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	11	4.439	2.48*	(1.24-4.43)
Hispanic	5	4.481	1.12	(0.36-2.61)
Black	1	0.162	6.17	NC
Other	0	0.426	0.00	NC
Age				
45-54	1	0.964	1.04	NC
55-64	5	1.349	3.71*	(1.20-8.66)
65-74	3	2.913	1.03	(0.21-3.01)
75+	8	3.569	2.24	(0.97-4.41)
Total	17	9.508	1.79*	(1.04-2.86)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A21 - Number of Larynx Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 2, 1997-2005 –Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	2	0.607	3.30	NC
Hispanic	1	0.434	2.30	NC
Black	1	0.019	52.63	NC
Other	0	0.011	0.00	NC
Age				
45-54	1	0.192	5.21	NC
55-64	1	0.226	4.25	NC
65-74	2	0.449	4.45	NC
75+	0	0.156	0.00	NC
Total	4	1.070	3.74*	(1.02-9.56)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A22 – Number of Lung Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 2, 1997-2005 – Males and Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	69	47.850	1.44**	(1.12-1.85)
Hispanic	15	16.137	0.93	(0.52-1.53)
Black	2	1.269	1.58	NC
Other	1	1.284	0.78	NC
Age				
0- 4	1	0.005	200.00	NC
5- 9	0	0.000	0.00	NC
10-14	0	0.004	0.00	NC
15-19	0	0.010	0.00	NC
20-24	0	0.004	0.00	NC
25-34	1	0.192	5.21	NC
35-44	2	1.018	1.97	NC
45-54	6	4.036	1.49	(0.54-3.24)
55-64	12	11.776	1.02	(0.53-1.78)
65-74	31	26.035	1.19	(0.81-1.69)
75+	34	23.458	1.45*	(1.00-2.03)
Total	87	66.539	1.31*	(1.05-1.62)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A23 – Number of Leukemia Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 2, 1997-2005 – Males

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	10	6.817	1.47	(0.71-2.70)
Hispanic	9	2.737	3.29**	(1.51-6.24)
Black	0	0.111	0.00	NC
Other	0	0.178	0.00	NC
Age				
0- 4	0	0.602	0.00	NC
5- 9	1	0.226	4.43	NC
10-14	1	0.237	4.22	NC
15-19	0	0.313	0.00	NC
20-24	0	0.082	0.00	NC
25-34	1	0.508	1.97	NC
35-44	1	0.545	1.84	NC
45-54	2	0.856	2.34	NC
55-64	2	1.213	1.65	NC
65-74	5	2.297	2.18	(0.70-5.09)
75+	6	2.964	2.02	(0.74-4.41)
Total	19	9.843	1.93*	(1.16-3.02)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A24 – Number of Nasopharynx Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 3, 1997-2005 – Males and Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	2	0.196	10.20	NC
Hispanic	0	0.241	0.00	NC
Black	2	0.396	5.05	NC
Other	1	0.308	3.25	NC
Age				
0- 4	0	0.000	0.00	NC
5- 9	0	0.013	0.00	NC
10-14	2	0.028	71.43	NC
15-19	0	0.032	0.00	NC
20-24	0	0.032	0.00	NC
25-34	1	0.064	15.63	NC
35-44	0	0.242	0.00	NC
45-54	0	0.392	0.00	NC
55-64	0	0.089	0.00	NC
65-74	1	0.228	4.39	NC
75+	1	0.021	47.62	NC
Total	5	1.141	4.38*	(1.42-10.24)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A25 - Number of Small Intestine Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 3, 1997-2005 – Males and Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	4	0.733	5.46*	(1.49-13.96)
Hispanic	0	0.832	0.00	NC
Black	4	1.652	2.42	(0.66-6.19)
Other	0	0.032	0.00	NC
Age				
25-34	1	0.178	5.62	NC
35-44	1	0.360	2.78	NC
45-54	1	1.073	0.93	NC
55-64	3	0.748	4.01	(0.83-11.73)
65-74	2	0.379	5.28	NC
75+	0	0.500	0.00	NC
Total	8	3.249	2.46*	(1.06-4.85)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)

Table A26 - Number of Bladder Cancer Diagnoses by Race/Ethnicity and by Age Compared to the Expected Number in Area 3, 1997-2005 – Males and Females

Race/ Ethnicity	Cancers Diagnosed	Cancers Expected	Ratio of Diagnosed to Expected	95% C.I. for Ratio
White Non-Hispanic	14	7.516	1.86*	(1.02-3.13)
Hispanic	7	3.163	2.21	(0.89-4.56)
Black	8	6.620	1.21	(0.52-2.38)
Other	0	0.431	0.00	NC
Age				
25-34	1	0.250	4.00	NC
35-44	6	0.796	7.54**	(2.76-16.42)
45-54	6	3.048	1.97	(0.72-4.29)
55-64	2	5.046	0.40	NC
65-74	10	5.298	1.89	(0.91-3.47)
75+	4	3.208	1.25	(0.34-3.19)
Total	29	17.731	1.64*	(1.10-2.35)

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 p=0.05 level. (** p=0.01 level)

NC = not calculated due to less than 3 diagnoses (see text for explanation)