

CANCER INCIDENCE STUDY

**Cancer Incidence Statistical Review
For Moab (including Spanish Valley), Grand County, Utah
Covering the Period from 1975 to 2009**

April 15, 2013

Prepared by the

Utah Department of Health
Division of Disease Control and Prevention
Bureau of Epidemiology
Environmental Epidemiology Program

TABLE OF CONTENTS

ACKNOWLEDGMENT.....	3
EXECUTIVE SUMMARY	4
INTRODUCTION	6
DATA AND METHODS	7
FINDINGS.....	14
DISCUSSION.....	17
CONCLUSIONS AND RECOMMENDATIONS	34
AUTHORSHIP, REVIEW AND CITATION	36
CERTIFICATION	37
REFERENCES	38
FIGURES	50
TABLES	55
DEFINITIONS.....	97

ACKNOWLEDGMENT

Cancer data used for this investigation was obtained from the Utah Cancer Registry (UCR). The UCR is funded by contract N01-PC-35141 from the National Cancer Institute (NCI)'s Surveillance, Epidemiology and End Results (SEER) Program with additional support from the Utah Department of Health (UDOH) and University of Utah.

Other data and analytical tools used for this investigation were obtained from the Utah Environmental Public Health Tracking Network (UEPHTN). In addition, the UEPHTN provides geocoding services to UCR data. The UEPHTN is funded by a grant from the Centers for Disease Control and Prevention (CDC), Environmental Public Health Tracking Branch. The current UEPHTN award is number 1U38EH000954.

EXECUTIVE SUMMARY

Cancer is a dominating environmental public health concern. A function of epidemiology is to investigate cancer incidence starting with a statistical review of cancer cases. This report presents a statistical review of cancer incidence among residents of Moab. The Environmental Epidemiology Program (EEP), within the Utah Department of Health (UDOH) conducted this statistical review by comparing the cancer incidence of seven sequential 5-year time periods for 42 anatomical site-specific cancer categories to expected counts derived from the state age-adjusted cancer rate for the corresponding site and time period.

The EEP considers the incidence of cancer to be meaningfully elevated when two or more sequential time periods have statistically elevated cancer incidence counts, or when the ratio of the observed incidence count to the expected count is greater than five. The EEP found that lung cancer was elevated in the first six (1974-2004) of seven five-year analytical periods suggesting the presence of a temporal cluster. The risk ranged from 2.5 to 3.7 times higher than expected for men and 2.3 to 3.2 times higher than expected for the total population. This finding suggests the presence of a temporal cluster of lung and bronchial cancer in Moab. Lung and bronchial cancer found to be elevated for women for two analytical periods but not following a pattern suggesting a temporal cluster for women.

Cervical cancer was found to be 5.8 times higher than expected for the 1980-1984 analytical period. This finding is meaningful for that period. Lung cancer and cervical cancers are preventable cancers. For persons developing these cancers, early detection and early intervention for these cancers improve the prognosis for recovery and quality of life experience. These findings suggest that action should be taken to assist the Moab population in understanding the risks of these cancers and ways to reduce their individual risk.

Cancers of the oral cavity and pharynx, rectum and recto-sigmoid junction, and non-melanoma skin cancers were statistically elevated during one five-year analytical period, but not to a level that would suggest a cluster. Prostate cancer, non-Hodgkin lymphoma and cancers of the kidney and renal pelvis were not elevated but showed a pattern of increasing trend through time. The known risks and epidemiology of each of these cancer sites are discussed.

The EEP recommends that Southeastern Utah District Health Department (SEUDHD) work with the Utah Cancer Control Program for screening and health education services that could be made available to the study area communities. In addition, the EEP recommends that SEUDHD request a follow-up cancer statistical review after three to five years (2010 to 2014) of additional cancer data become available to EEP.

INTRODUCTION

Cancer Incidence Statistical Reviews: A core function of epidemiology is to track and evaluate disease patterns. This function helps public health officials and policy makers identify and assess communities with public health challenges, define public health priorities, monitor and evaluate public health actions, and discover knowledge about public health concerns (Clapp 2000; Dicker 2002; Stanbury et al. 2012; Thacker 2000; Thacker et al. 2012). Cancer is a dominating environmental public health concern. Public fear of cancer resulting from environmental hazards is reinforced by U.S. environmental regulatory actions that use cancer as a mechanism for making regulatory decisions (Clapp 2000; Morrone 2011; Trumbo 2000; Trumbo et al 2008). Public concerns about excess cancer risk often result in requests made to public health agencies to conduct investigations (Trumbo 2000; Warner and Aldrich 1988).

Public health conducts investigations of cancer incidence using one of several methods. The first is a cancer incidence statistical review. This method focuses on determining if a particular community is experiencing more cancer than would be expected. A cancer statistical review is usually conducted by linking cancer registry and population data and evaluating trends. From the public health perspective, cancer incidence statistical review is most useful in identifying community needs about cancer related health education and awareness building, public health screening services and other public health interventions. For the community, these kinds of studies empower the community to make improvements in governmental policymaking and health-care services (Anderston et al 2012; Bell et al. 2006; Bender et al 1990; Caldwell 1990; Kingsley et al. 2007; Frumkin and Kantrowitz 1987; Thun and Sinks 2004; Warner and Aldrich 1988).

Another method available to public health practitioners is a cancer cluster investigation. Cancer cluster investigations focus on characterizing the size and extent of a population with known cancer excess and on determining potential causal factors. The cancer cluster methodology involves linking many causal variables, usually collected by medical record review and individual surveys or interviews, followed by complex statistical analysis to identify the few variables that seem to explain the risk (Anderson et al 2012; Bender et al 1990; Caldwell 1990; Kingsley et al. 2007; Thun and Sinks 2004; Warner and Aldrich 1988). Cluster investigations rarely result in important discoveries of causality (Goodman et al. 2012; Kingsley et al. 2007).

Study Area Background and Current Status: Moab is a rural community of about 5,000 permanent residents, located just south and east of the Colorado River on the Colorado Plateau at an elevation around 4,000 feet and in Grand County. Because of its close proximity to Arches National Park, Canyonlands National Park, and Dead Horse Point State Park, Moab is a hub for tourism. For this study, Moab includes Spanish Valley, an unincorporated residential area along State Highway 191 adjacent to and south of Moab. Spanish Valley extends south into San Juan County.

In 1956, the Uranium Reduction Company constructed and started operation of the Moab mill. This mill was sold to Atlas Minerals Corporation in 1962. The mill operated until 1984. During the time the mill operated, the mill processed about 1,400 tons of ore a day and generated approximately 16 million tons or 12 million cubic yards of mill tailings and tailings-

contaminated soil. These tailings exist in a tailings pile located north of Moab. The tailings have an average radioactivity of 665 picocuries or radium-226 per gram of soil. Decommission activities occurred between 1988 and 1995. As part of those activities, an interim cover was placed over the tailings pile. In 1998, Atlas Minerals Corporation relinquished through bankruptcy. At that time, the Nuclear Regulatory Commission appointed a trustee to initiate site reclamation. In 2001, the site was designated a Uranium Mill Tailings Remedial Action (UMTRA) site. The U.S. Department of Energy (U.S. DOE) became responsible for remediation of the site. In 2005, the U.S. DOE published an Environmental Impact Statement (EIS) documenting its investigation of the site, describing various remediation alternatives, and presenting the preferred alternative. The preferred alternative was to remove the mill tailings and tailings-contaminated soil to Crescent Junction, approximately 40 miles north of Moab. In 2003, the U.S. DOE established a number of extraction and injection wells to remediate leakage of ammonia, a mill tailing contaminate, into the Colorado River. Between 2008 and 2009, the U.S. DOE constructed the necessary infrastructure to remove the tailings to the disposal cell at Crescent Junction. Movement of material started in April 2009. In February 2012, approximately 31 percent of the material had been removed. The U.S. DOE estimates that the project will be completed in 2025 (GJEM 2013; MoabTailings 2011).

Statement of Public Health Concern: Because of concurrent American Recovery and Reinvestment Act (ARRA) of 2009 funds (\$104.9 million) made available to the U.S. DOE for the Moab UMTRA, the activity between 2009 and 2012 was high (USDOE 2010). With the end of ARRA funding, activity at the Moab UMTRA have slowed considerably. Some residents fear that the removal of the cap that had been installed by Atlas in 1988 will result in renewed exposure of Moab residents to contaminated materials migrating from the UMTRA site into Moab (EEP 2012, 2013).

Request: On December 4, 2012, David Cunningham, Health Officer for the Southeastern Utah District Health Department (SEUDHD) forwarded a request from the Grand County Council and Mayor of Moab that a cancer statistical review be conducted. Staff from EEP met with the UMTRA steering committee and presented a study plan for conducting the requested statistical review on January 8, 2013 (EEP 2012, 2013).

Study Objectives: This report presents a statistical review of cancer incidence among residents of Moab. The Environmental Epidemiology Program (EEP), within the Utah Department of Health (UDOH) conducted this statistical review by analyzing periodic rates and trends in rates of cancer incidence in the study area, compared to corresponding rates of the state of Utah. The objective of a statistical review is to identify significantly elevated cancer incidence rates. The statistical review methodology does not quantify the linkage of cancer rates to possible causal risk factors. Specific hazardous chemicals of concern and exposure risk are not addressed by this report.

Authority and Funding: This study was requested by David Cunningham, Health Officer, SEUDHD on behalf of Dave Lee, Mayor of Moab and authorized by the UDOH Executive Director's Office. Cancer, population, and geographic data for this investigation are collected, maintained and made available by the Utah Environmental Public Health Tracking Network (UEPHTN). The UEPHTN also funds the SAS[®] and ArcGIS[®] analytical software application

licenses that were used to conduct this investigation. The UEPHTN is funded by a grant from the Centers for Disease Control and Prevention (CDC) (UEPHTN 2012). Personnel time used to conduct this investigation was charged against state-funded Environmental Health Administrative funds. No federal funds were directly used to conduct this investigation.

DATA AND METHODS

Study Design: This investigation is a retrospective statistical review of cancer incidence among residents of the study area (defined below). Statistical reviews are not cancer cluster investigations, and lack the power to link cancer incidence to putative risk factors (dos Santos Silva 1999; Esteve et al. 1994; Jekel et al. 1996; Kingsley et al. 2007; Mann 2003). Statistical reviews are a tool used by the EEP to review the health status of a population and assess public health activities.

The incidence of cancer, quantified in sequential 5-year incidence rates for each cancer category among residents of the study area, is compared to cancer incidence rates for the state of Utah. The study's null hypothesis is that the incidence of cancer in study area is not significantly different from the incidence of cancer for the state of Utah.

Cancer Data: Cancer incidence data on people diagnosed with primary invasive cancer between 1973 and 2009 were obtained from the Utah Cancer Registry (UCR). The UEPHTN within the EEP receives cancer data for all invasive cancers on an annual basis. The UCR completes a rigorous data review for completion and data quality before data are released to the UEPHTN. The most recent years of data are not made available to the UEPHTN until they have been finalized. The UCR data includes diagnostic information, patient demographics, and residential addresses of the cases, as well as information about the behavior of the cancer. The residential address information provided by the UCR includes the city and ZIP code (UCR 2012). The UEPHTN geocodes each cancer case's residential address data to obtain an x- and y-coordinate for that address. Using those coordinates the UEPHTN is able to geo-reference cancer case data to their respective U. S. 2000 census block group areas (USCB 2004, UEPHTN 2012).

Individuals with multiple primary invasive cancers have multiple records in the data set in sequential order. These cancers are distinguished by unique cancer registry tracking numbers and a cancer sequence number. The sequence number allows discrimination between the first cancer diagnosis and subsequent diagnoses (UCR 2012). Diagnostic coding of cancers includes the International Classification of Disease Oncology, 3rd Edition (ICD-O-3) codes for site, histology and behavior (WHO 2012). When conducting a population-based statistical review, it is convenient to group similar cancers together, usually by location in the body. The UCR groups cancer into 42 major cancer types by site following the guidance provided by the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) Program (Adamo et al 2011; NCI 2012a; Thorton 2012; UCR 2012). These 42 UCR site codes are a convenient grouping for conducting surveillance analyses and allow the comparison of findings of this report with national and state cancer patterns (Copeland et al 2011).

Certain kinds of medical treatment for cancer and other diseases, such as radiation therapy, increase an individual's risk for developing subsequent leukemia, particularly myeloid leukemia (sometimes known as therapy-induced leukemia) (Godley and Larson 2008; Leone et al. 1999, 2011; Sill et al. 2011; Wilkins and Woodgate 2008). Myeloid leukemia cases that were the first of any sequence of cancers for an individual were included for this investigation. Myeloid leukemia cases that were subsequent to a previous cancer and could be therapy-induced leukemia were excluded.

Statewide between 1975 and 2009, 169,973 invasive primary cancer incidence reports among 148,943 individuals were registered by UCR. Within the study area, 878 persons experienced 990 cancer incidences between 1975 and 2009. Approximately 33% of cases were first diagnosed at an out-of-state health care facility and 67% of cases were first diagnosed at an in-state health care facility.

Population Data: The 2000 U.S. census divides Utah into 1,481 census block groups (USCB 2004) with a median population of 1,364 persons per census block group in the year 2000. Commercially available U.S. census population data for Utah for the 1970, 1980, 1990, 2000 and 2010 censuses (Geolytics 2002a, 2002b, 2002c; Geolytics 2012a, 2012b) were used to estimate annual age-group and sex population counts for each census block group for each intercensal year. These estimates were made by applying annual population growth rates derived from the previous and subsequent decennial data. This method follows national population estimation guidelines (USCB 2012). The UEPHTN prepares population data for the EEP (UEPHTN 2012).

Analytical Periods: Seven five-year analytical periods: 1975-1979, 1980-1984, 1985-1989, 1990-1994, 1995-1999, 2000-2004 and 2005-2009 were evaluated for temporal cancer incidence trends.

Study Population: The study population was defined as all residents living in Moab and Spanish Valley. Cancer cases living in Spanish Valley are recorded in the UCR as living in Moab. Of those records, 79% have a street address that can be geocoded. Thus cases were identified by having the term "Moab" as the residential city in the record.

The smallest population geography that tabulates the population in age and sex groups is the census block group. The populated area of Moab (including Spanish Valley) falls into eight census block groups (using the 2000 census geography). These census block groups are: 49.019.000100.1, 49.019.000100.2, 49.019.000100.3, 49.019.000200.1, 49.019.000200.2, 49.019.00020.3, 49.019.000200.4, and 49.037.978100.1. See Figure 1 in Appendix. Seven of these block groups (those starting with 49.019 where 49 is the state FIPS code for Utah, and 019 is the county FIPS code for Grand county) are in Grand county and the last (where 037 is the county FIPS code for San Juan county) is in San Juan county. The smallest census geography is the census block. The eight census block groups include 1,465 census blocks, 251 of which are within Moab. The population of census blocks inside and outside of Moab for each census block group, were used to estimate the proportion of the population for each census block group.

Census Block Group	Study Area Block Group Population in 2000	Moab Population (from Blocks) in 2000	Percentage of Block Group Population in Moab	Percentage of Block Group Level Population in Voting Age	Percentage of Block Level Population in Voting Age
49.019.000100.1	1,199	866	72.23%	72.71%	72.40%
49.019.000100.2	1,206	1,206	100.00%	71.72%	71.72%
49.019.000100.3	1,723	1,264	73.36%	77.07%	77.14%
49.019.000200.1	1,044	1,021	97.80%	74.71%	74.14%
49.019.000200.2	1,348	1,348	100.00%	70.03%	70.03%
49.019.000200.3	923	901	97.62%	69.45%	69.48%
49.019.000200.4	1,040	1,040	100.00%	74.04%	74.04%
49.037.978100.1	1,168	176	15.08%	72.92%	75.00%
Total	9,650	7,822	81.06%	72.08%	72.82%

These adjustments assume that the age and sex distribution of the population among the census blocks that comprise a census block group are uniform. Because the population tallies of the census blocks are not broken down into age and sex specific strata, it is not possible to test this assumption. However, both geographies include tabulation of voting age (adults 18 years and older). Comparing the ratio of voting age to total population indicates that the errors resulting from this assumption are likely small (GeoLytics 2012b). These adjustments also assume that the age and sex distribution is consistent through time from 1973 through 2009. It is not possible to test this assumption.

Cancer cases and population data were aggregated into six age group strata: 0-19 years of age, 20-34 years of age, 35-49 years of age, 50-64 years of age, 65-74 years of age, and 75 years and older. The cancer incidence by cancer types and population counts for each age group, sex and analytical period strata for each of the study area census block groups were added together to generate the age group, sex and analytical period cancer incidence and population counts for the study population.

Comparison Population: The comparison population for this investigation was defined as the state population excluding the study population. Similar to the process of developing the study population, the cancer incidence by cancer type and population counts for each age group, sex and analytical period for all of the census block groups in the state not included in the study population were added together to generate the comparison population. The 2010 estimated population for the state is 2,763,885 (GeoLytics 2012b).

The proportion of the population in each age group for the Moab population was found to be highly with the state population early in the study. For the 1975-1979 analytical period the Pearson Correlation Coefficient (ρ) was 0.9958 (p -value < 0.0001). The level of correlation of the population age distribution consistently decreased with each analytical period to the end of

the study. For the 2005-2009 analytical period ρ was 0.8936 (p -value = 0.0019). The population age distribution for Moab shifted towards a higher proportion of the population in the older ages.

Socio-Economic Assessment of the Study and Comparison Populations: Social determinants of health are complex, integrated, and overlapping social structures and economic systems that are now thought to affect disease morbidity and mortality. Education level is an example. A better education leads to higher income and financial stability, which in turn leads to better health care access, which leads to healthier lifestyles, and to earlier detection and better treatment options for disease (Song et al. 2011). Since 2010, census did not collect information about education, income or occupation, previously gathered in the 2000 census. Since 2000, the US Census Bureau has used the American Community Survey (ACS) to sample a small percentage of the US population each year to collect this kind of information. Data from the ACS are available for the Moab Census County Division (CCD). The ACS 5-Year estimates for 2007-2011 were used to compare selected demographic and economic characteristics that are important social determinants of health for cancer. These risk factors contribute to the burden of disease, but are not the risk of concern for this investigation. Ideally, the social determinants of health metrics for the study area should be similar to the comparison population. If the social determinants of health between the two groups are disproportionate, the social determinants of health may confound the investigation of environmental risk assessment. (USCB 2013a, 2013b, 2013c, 2013d).

Estimate	Moab, Utah	State of Utah
2011 Population (People Count)	8,956	2,763,885
Percent of Population is of Minority Race	7.4%	13.9%
Percent of Population is Hispanic or Latino	8.6%	13.0%
Median Age of Population (Years)	39.0	29.2
Percent Population are Children 0-17 Years Old	23.1%	31.5%
Percent Population are Adults 65 Years or Older	12.2%	9.0%
Households	3,553	877,692
Percent Family Households (2 or more related persons)	58.1%	75.2%
Percent Single Person Households	35.1%	18.7%
Percent Married Couple Households	46.7%	61.0%
Average Family Size	3.2	3.6
Percent Population in Family Households	73.4%	85.0%
Housing Units	4,598	979,709
Percent Housing Units Occupied	77.3%	89.6%
Percent Owned Occupied Homes	66.9%	70.4%

Demographic distribution and housing factors for the Moab CCD that were more than ten-percent different from the state of Utah were evaluated. Moab has less minority and Hispanic population, is an older population and has less of the population living in a family structure.

Estimate	Moab, Utah	State of Utah
Percent Adults with at least a High School Education	84.4%	90.4%
Percent Adults Employed	66.4%	65.9%
Percent Adults Employed in Jobs at High Risk for Chemical Exposure (Military, Agriculture, Construction, Manufacturing, Transportation, etc.)	21.0%	15.4%
Average Household Income	\$52,801	\$69,686

Moab has a higher rate of persons working in occupations that have a higher risk for occupational chemical exposures such as agricultural chemicals, glues, sealants, plastic- or petroleum-based materials. The average income in Moab is lower than the state average which may impede access to health care services.

Estimate	Moab, Utah	State of Utah
Percent Families Living Below Poverty	6.0%	7.2%
Percent Families with Children 0-18 Years Living Below Poverty	7.5%	9.8%
Percent Persons 65 Years or Older Living Below Poverty	8.5%	7.6%
Percent Population Foreign Born	5.3%	7.9%
Percent Population Not U. S. Citizens	3.4%	5.4%

Moab has a lower rate of families and children living in poverty but a higher percentage of older persons living in poverty. Since age is a risk factor for developing cancer, the potential that Moab's older population has less access to health care, screening services, or other early interventions is a concern in interpreting the findings of this investigation. Moab has a lower rate of foreign born, minority race, Hispanic/Latino and non-U.S. populations. These populations sometimes have cultural behaviors or language barriers impede access healthy life choices, access to health care, screening services, and early intervention.

This statistical review of potential socio-economic confounders found that there are significant differences with respect to these factors between Moab and the state. This investigation does not control for these potential confounders. Interpretation of the findings of this investigation should keep in mind that these confounders could have influence in the findings.

This analysis is based on a single assessment of socio-economic factors. Data is not available to determine if the status of these conditions are consistent through all of the analytical periods of this study.

Behavioral Risk Factors: Tobacco use, chronic alcohol use, and obesity are well known risk factors for many types of cancer. The UDOH conducts annual behavioral risk factors telephone surveys in Utah. These data are made available publicly on Indicator-Based Information System for Public Health (IBIS-PH) website tabulated using a small area geography known as health statistical units. The health statistical units are aggregations of one or more ZIP code areas to achieve an annual population of at least 20,000 persons. Unfortunately, for southeastern Utah the

populations of communities are small enough that it is not possible to get behavioral risk factors specific for Moab. In fact, Grand and San Juan counties are aggregated to achieve the minimum required population for reporting purposes. It is not known how well the aggregation of Grand and San Juan county behavioral risk rates reflect the behavioral risks of Moab. The Behavioral Risk Factors Survey System (BRFSS) data was queried for these behavioral risks as well as access and utilization of health care. All available years of data from 2001 through 2010 were used for the queries (UDOH 2012).

Estimate	Grand and San Juan Counties	State of Utah
Smoking Rates among Adult Men	18.8%	12.6%
Smoking Rates among Adult Women	12.9%	9.2%
Chronic Drinking (Alcohol) Rates among Adult Men	5.7%	3.5%
Chronic Drinking Rates among Adult Women	2.9%	2.3%

Estimate	Grand and San Juan Counties	State of Utah
Body Mass Index (BMI) greater than 25 among Men	62.3%	64.7%
BMI greater than 25 among Women	52.9%	47.7%
Percent of Population with No Health Coverage	27.8%	15.0%
Percent of Population not Receiving Routine Medical Checkup in Last Year	41.9%	44.6%

These data suggest that Grand and San Juan counties have higher smoking and chronic alcohol consumption rates than the state of Utah. Men are not as obese but women are more obese. Residents of the counties have less access to health care services but tend to use the service more than state residents. These are aggregated county data. It is not known how representative these data are with respect to Moab.

Indirect Age-Standardized Incidence Rates: The statistical analyses program SAS[®] version 9.2 was used to manage and analyze the data. The sex-specific and sex-nonspecific indirect age-standardized incidence rate for each cancer type and analytical period was calculated using standard methods (Anderson and Rosenberg 1998; dos Santos Silva 1999; Esteve et al. 1994; Jekel et al. 1996; Selvin 1996). This is the preferred method for analysis of disease with small case counts per analytical period. The expected incidence count and rate was computed by applying the comparison population incidence rate to the study area population for each analytical period using the indirect age-standardization method.

Standardized Incidence Ratio: The standardized incidence count of cancer for the study area was evaluated against the expected incidence count in the form of standardized incidence ratio (SIR). An SIR greater than one (1.0) indicates that the incidence of cancer in the study area population is greater than the proportional cancer incidence in the comparison population for that period of analysis. Conversely, an SIR less than one indicates that the incidence of cancer in the study area population is less than expected based on the comparison population's rate. Statistical

significance is determined by applying the Byar's 95% confidence interval for the SIR (Breslow and Day 1987; Rothman and Boice 1979, 1982; Sahai and Khurshid 1983, 1996). For statistical validity, SIRs and corresponding 95% confidence intervals were only calculated for time periods with three or more cases (Bender et al. 1990; Caldwell 1990; Thun and Sinks 2004). The EEP is required to protect confidential data from unlawful disclosure; therefore, the EEP suppresses results for analytical time periods containing three or less cases (Langeberg et al. 2004).

A SIR for a specific cancer greater than one (1.0) and a confidence interval (expressed by the lower and upper limits) that does not include one (1.0) is considered to be statistically significant. Using a 95% confidence interval is a well-established standard for interpretation of an SIR with respect to statistical significance. It should be noted that a SIR may be statistically significant using this interpretation criteria, and may be a mathematical artifact and not biologically meaningful or relevant (Bender et al. 1990; Besag and Newell 1991). When conducting multiple analyses using the 95% confidence interval to interpret the data, one would expect one in twenty (5%) of the analyses to have a statistically significant interpretation as a result of random chance. For this investigation, 784 independent analyses (35 cancer type categories x 3 sex groups x 7 analytical periods and 7 sex-specific cancer types x 1 sex group x 7 analytical periods) were conducted. This means as many as 39 (784 x 5%) of the analytical results could be due to chance. The EEP uses interpretive rules to distinguish results that are meaningfully significant from those that are not. The EEP considers the results meaningful when there are two consecutive time periods with a statistically significant result or when the SIR is greater than five (Bender et al. 1990; Caldwell 1990; Langeberg et al. 2004; Thun and Sinks 2004).

Analysis of Temporal Trend: The Kendall Tau-c (or Kendall rank correlation coefficient) test for trend was used to test for temporal trends of increasing or decreasing rates (Kendall 1938). The Kendall Tau-c statistic is an appropriate method to investigate trends when there are only a few analytical periods. The Kendall Tau-c tests the correlation between the analytical period rate and the ordered numeric designation of the analytical periods (i.e., analytical period 1975-1979 is number 1, period 1980-1984 is number 2, etc. till period 2005-2009 is number 7). The values of Tau-c range from -1 (a consistent decreasing trend) to +1 (a consistent increasing trend). Values near zero indicate no trend. Trend was indicated by statistically significant (p-value ≤ 0.05) correlation coefficients (approximately equaled to ± 0.70).

FINDINGS

The analytical results for the study area for each of the 42 cancer types and analytical periods are presented in Table 1 (see Appendix). Five cancer types were found to be elevated during at least one analytical period. Those types are: cancers of the oral cavity and pharynx, rectal and recto-sigmoid junction cancers, lung and bronchial cancers, non-melanoma skin cancers and cancers of the cervix.

Statistically Significant Cancer Results: Significantly elevated cancer incidence rates are indicated with an "S" in Table 1. Among men and among both sexes combined, lung and bronchial cancer incidence was elevated during six sequential analytical periods from 1975

through 2004. Among men the SIR ranged from 2.50 to 3.67 [the lowest and highest 95% confidence limits (CI) were 1.40 and 5.50] or two to three times higher than expected. Similarly among both sexes the SIR for lung and bronchial cancer ranged from 2.25 to 3.23 [lowest and highest CI are 1.43 and 4.73] for the same six analytical periods. Oral and pharyngeal cancer was elevated (SIR = 2.76 [CI = 1.01 – 6.01]) among men for the 1995-1999 analytical time period. Non-melanoma skin cancers were elevated (SIR = 4.06 [CI = 1.09 – 10.39]) for both sexes evaluated together during the 2000-2004 period.

For women, cancer of the lung and bronchus were elevated during 1985-1989 (SIR = 2.88 [1.24 – 5.67]) and the 1995-1999 (SIR = 3.07 [1.68 – 5.16]) analytical time periods. Cervical cancer was elevated during 1980-1984 (SIR = 5.81 [2.33 – 11.97]) and the 1995-1999 (SIR = 4.64 [1.69 – 10.09]) analytical time periods. Rectal and recto-sigmoid junction cancer incidence was elevated for the 2005-2009 (SIR = 3.19 [1.16 – 6.94]) analytical period.

From 1975 through 2009, 183 incidence of lung cancer were reported to the UCR. Lung cancer can be further defined into different type based on the cancer cell histology and location. The two major types are small-cell lung cancer and non-small-cell lung cancer. Non-small-cell lung cancer are further differentiated into large cell carcinoma, adenocarcinoma and adenosquamous. Fifty-seven cases of lung cancer were not sufficiently characterized to determine which type of lung cancer it was. Of the 126 characterized cases, 20 (15.9%) were small-cell lung cancers, 103 (81.7%) were non-small-cell lung cancers, and 3 (2.4%) were other types (i.e., broncho-alveolar cancers). The most common types of non-small-cell lung cancer were adenocarcinoma and squamous cell carcinoma. There were 51 (49.5% of non-small-cell lung cancers) cases of adenocarcinoma cancers and 38 (36.9%) cases of squamous cell carcinoma.

Meaningful Cancer Results: Lung cancer was significantly elevated for men and both sexes combined for six sequential time periods from 1975 through 2004. This finding suggest temporal cancer cluster in Moab. Among women, lung cancer was statistically elevated during two non-sequential analytical periods during the same time as the temporal cluster for men and both sexes combined. During the 1980-1984 analytical period, the incidence rate for cervical cancer exceed 5.0 suggesting a short term cancer cluster.

Trends: Analysis of the changes of the rate of cancer incidence through time (trend analysis) identified types of cancer with increasing or decreasing trends. Not all cancer types with a significant trend have significantly elevated cancer incidence. However, it is possible, that cancer types with a significant trend of increasing incidence will eventually reach a time where the incidence is significantly elevated. Not all cancer types that were elevated during one or more analytical periods present a significant trend.

For this study, a significant increasing trend in cancer incidence was found among Moab men for prostate cancer ($p = 0.02$) and non-Hodgkin lymphoma ($p = 0.03$). For women, an increasing trend of cancer incidence was found for kidney and renal pelvis cancer ($p = 0.04$). This finding was also true for kidney and renal pelvis cancer among both sexes in Moab ($p = 0.02$). None of these cancer site categories were found to be elevated, but this finding suggests that they could become elevated in the future.

DISCUSSION

Cancer: Five basic types of cells make up the body. These cell types include epithelial cells, connective tissue cells, muscle cells, nerve cells and blood cells. These cells arise from stem cells or progenitor cells that divide and specialize (“differentiate”) to become different kinds of tissues that form organs and organ systems. Rapid cellular division and differentiation occurs throughout fetal development and juvenile maturation. Once adulthood is achieved, cellular division and differentiation is regulated to replace damaged or dying cells. For example, the adult body replaces white blood cells every thirty days and red blood cells every four months. The process of cell division and differentiation (the process of specializing into a tissue cell) is highly regulated. Uncontrolled cellular division can lead to non-functional growths. These non-functional growths are called neoplasms, or more commonly called cysts, polyps, or tumors. Most neoplasms are benign, meaning that they lack the ability to invade surrounding tissues or metastasize (spread to other parts of the body) and can usually be treated or removed. Other neoplasms are malignant meaning they have the ability to invade surrounding tissues or metastasize (King and Robins 2006; Weinberg 2006).

Cancer is a broad group of more than 100 diseases that involve uncontrollable cell replication and growth. Cancer arises when damage occurs to cellular genes that control cell replication, tissue growth and differentiation. On average, every cell in the body experiences some kind of genetic damage each day. Each cell in the body conducts about 5,000,000 repair checks per day. Almost all damaged genes are repaired. When the damaged gene cannot be repaired, in most cases, the cell dies. The average adult human experiences 50-70 billion cell deaths per day. In a small percentage of cases where a cell is damaged, cannot be repaired, and does not die, the cell loses some functions, but continues as a non-replicating tissue cell. When the genetic damage is specific to one of the genes controlling cellular replication, the damage may result in the formation of a cancerous cell.

Cancer cells do not undergo the aging process that normal cells undergo. Often these cells are “undifferentiated,” meaning they have lost their tissue-specific characteristics. Unlike normal cells, cancer cells lose the need to be anchored with other cells of the same tissue type. Cancer cells become insensitive to growth control mechanisms. Because cancer cells are physiologically more active than normal cells, cancer cells are able to promote angiogenesis causing nearby blood vessels to produce branches that grow into the cancerous tissue (King and Robins 2006; Weinberg 2006).

As cancer cells grow to form tumor tissue, they invade nearby healthy tissue or spread through metastasis to other tissues. This invasion or spread disrupts the functions of the affected healthy tissues. Cancer cells may also produce metabolic products that can be transported to other parts of the body resulting in adverse health effects. Different kinds of cancer have different physiological characteristics, causal risk factors, prognoses, and treatment (King and Robins 2006; NCI 2012a, 2012b; Weinberg 2006).

Cancers are classified as “carcinoma in-situ (CIS)”, meaning that the cancer cells are found at the site of origin and are not invading the surrounding tissues; “invasive,” meaning the cancer cells

are found at the site of origin and are invading the surrounding tissue; or “metastatic,” meaning the cancer cells originated elsewhere, have migrated (usually through the blood or lymphatic system) to a new location, and are invading the surrounding tissues (King and Robins 2006; NCI 2012a; Weinberg 2006). The American Cancer Society (ACS) estimates that about one in two men and one in three women will develop cancer (all invasive sites) sometime in their life (lifetime risk)(ACS 2009; NCI 2011a, 2011b). In the U. S., cancer is the second leading cause of death (CDC 2012; Jemal et al 2008). Among all causes of death, approximately, one in four men and one in five women will die of cancer (ACS 2009; NCI 2011a, 2011b). On average, about one in nine people will develop two or more cancers in his or her lifetime (Wilkins and Woodgate 2008).

Risk factors that contribute to the development of cancer include both inherent and external factors. Inherent factors include a variety of genetic susceptibilities. External factors include life choices and behaviors (e.g., tobacco use, alcohol use, poor diet, obesity, lack of physical activity, excessive sunlight exposure, and sexual behavior), medical conditions and medications, oncogenic pathogens, and chemical or radiological environmental exposures. Cancer may be the result of several factors interacting together with a triggering event (NCI 2012b; Stein and Colditz 2004).

The incidence of lung and bronchial cancer were found to be meaningfully elevated for multiple periods of time suggesting the existence of a temporal lung cancer cluster. Cervical cancer was also found to be meaningfully elevated for one analytical period. The epidemiology and known or common risk factors associated with these cancers are discussed below.

Cancer of the oral and pharyngeal cancer, rectal cancer, kidney cancer, non-melanoma skin cancer, prostate cancer, and non-Hodgkin lymphoma were found to be elevated during at least one analytical period or to have a significant increasing trend of incidence. While these findings were not found to have public health relevance, based on the criteria for meaningful cancers, they also are discussed briefly below to provide public health education about cancer.

Lung and bronchial cancer: Lung cancer is the leading cause of cancer-related mortality in the United States (ACS 2013; Alberg and Samet 2003; Alberg et al. 2007; Molina et al. 2009; NCI 2012b). It is also one of the few types of cancer that has been linked to environmental exposure to alpha-emitting radiation (Coggle et al. 1986; Mould 2001; Nermina 2005; Shottenfeld and Fraumeni 1996; Tomasek et al. 1993). Approximately 1 in 13 men and 1 in 16 women will develop lung cancer during their lifetime and 1 in 15 men and 1 in 20 women will die of lung cancer (ACS 2009). There are several different kinds of lung cancer. The four major types include squamous cell (also called epidermoid) carcinoma, adenocarcinoma (cancers of the glands of the lung), large cell carcinoma, and small cell undifferentiated carcinoma. Together these four types of lung cancer account for more than 90 percent of lung cancer cases in the United States (ACS 2013; Alberg and Samet 2003; Field et al. 2004; NCI 2012b). This investigation does not differentiate the different kinds of lung cancer.

The most important risk factors for all types of lung cancer include smoking, exposure to secondhand smoke, alcohol use, the presence of certain smoking-related lung diseases (e.g., chronic obstructive pulmonary disease), poor diet, lack of physical activity, a family history

(genetic susceptibility) of lung cancer, respiratory exposure to radon gas, respiratory exposure to asbestos, respiratory exposure to polycyclic aromatic hydrocarbons, and respiratory exposure to certain metals such as arsenic, chromium or nickel (ACS 2013; Alberg and Samet 2003; Alberg et al. 2007; Armstrong et al. 2004; Brenner et al. 2011; Bronson et al. 2002; Darby et al. 2001; Molina et al. 2009; NCI 2012b; Samet and Eradze 2000; Samet et al. 2009). Smoking and exposure to second hand smoke represents approximately 84% of all lung cancer deaths in the United States (Giovino 2002). Smoking is associated with types of lung cancer. Squamous cell carcinoma is the type of lung cancer most associated with smoking. Adenocarcinoma is the type most often found in never-smokers (Blot and Fraumeni 1996).

Cervical cancer: The cervix is the opening to and lower part of the uterus (womb) and connects the body of the uterus to the vagina. The cervix consists of the exocervix lined with a squamous cell layer and the endocervix lined with glandular cells. The place where these two parts meet is called the transformation zone. Most cervical cancers start in the transformation zone. Most (80-90%) of cervical cancers are squamous cell carcinoma. The remainder are adenocarcinoma or a mixed adenosquamous carcinoma (ACS 2013). Although cervical cancer incidence and mortality rates have declined approximately 50 percent in the United States over the past three decades, the disease remains a serious health threat. Incidence rates for Hispanic women are higher than those for non-Hispanic women. Even though the mortality rate for African-American women has declined more rapidly than the rate for white women, the African-American mortality rate continues to be at least double that of whites. Geographic and socioeconomic disparities in cervical cancer mortality also exist (NCI 2012b).

The most important risk factor for cervical cancer is infection by human papilloma virus (HPV). HPV is a group of more than 100 related viruses, some of which cause a type of growth called a papilloma (or more commonly known as warts). High risk types of HPV are HPV-16, -18, -31, -33 and -45. About two-thirds of all cervical cancers are caused by HPV-16 and -18. Other infections such as human immunodeficiency virus (HIV) or chlamydia also increase the risk for cervical cancer. Women who smoke are about twice as likely as non-smokers to develop cervical cancer. Women with diets low in fruits and vegetables may be at an increased risk for cervical cancer. Weight, weight gain and obesity increase the risk for adenocarcinoma of the cervix. Poverty has also been associated with the risk of cancer. Poverty may be a signal for other risks such as poor diet or inadequate health care services. Birth control including the use of certain oral contraceptives or intrauterine devices has an increased risk. Diethylstilbestrol (DES) is a hormone that was used to help some women to prevent miscarriage between 1940 and 1971. After that time, DES was no longer used. Women whose mothers took DES are at a slightly (1 in 1,000) higher risk for developing adenocarcinoma of the vagina or cervix. Women who have three or more full-term pregnancy or have their first full-term pregnancy at an age younger than 17 years have a higher risk for cervical cancer. Women who have a family history of cervical cancer have two to three times higher risk than women who do not have a family history for cervical cancer (ACS 2013, NCI 2012b).

Oral and pharyngeal cancer: Cancers of the oral cavity and pharynx include cancers of the lips, the inside lining of the lips and cheeks (buccal mucosa), the gums, the tongue, the floor and roof of the mouth, the top part of the throat just behind the mouth, and the tonsils. There are a number of different kinds of cancers that can develop depending on the tissue type. Oral and

oropharyngeal cancers are about twice as common in men as in women. This might be because men have been more likely to use tobacco and alcohol in the past. While this is changing, the recent rise in HPV-linked cancers has been mainly among younger men, so the difference in occurrence in genders is likely to remain in the near future. The use of tobacco, tobacco-like products (e.g., betel quid or gutka) and alcohol are among the strongest risk factors for oral cavity and oropharyngeal cancers. Most people with oral cavity and oropharyngeal cancers use tobacco, and the risk of developing these cancers is related to how much and how long they smoked or chewed. Tobacco smoke from cigarettes, cigars, or pipes can cause cancers anywhere in the mouth or throat, as well as causing cancers of the larynx (voice box), lungs, esophagus, kidneys, bladder, and several other organs. Pipe smoking is a particularly significant risk for cancers in the area of the lips that touch the pipe stem. Oral tobacco products (snuff or chewing tobacco) are linked with cancers of the cheek, gums, and inner surface of the lips. Using oral tobacco products for a long time poses an especially high risk. These products also cause gum disease, destruction of the bone sockets around teeth, and tooth loss. Drinking alcohol increases the risk of developing oral cavity and oropharyngeal cancers. The risk goes up even more for people who use both tobacco and alcohol. A diet low in fruits and vegetables is linked with increased risk for oral cancer. Oral human papillomavirus (HPV) infections increase the risk for oral and pharyngeal cancers. Prolonged unprotected exposure to ultraviolet light (e.g., sunlight) accounts for about half of cancers of the external lip. Immunosuppressive medical treatment (radiation therapy to the head or neck, medications) or diseases contribute to the risk for oral and pharyngeal cancer. Other risk factors include certain genetic conditions (e.g., dyskeratosis congenita or Fanconi anemia) and chronic medical conditions (graft-versus-host disease or lichen planus) (ACS 2013; NCI 2012b).

Rectal cancer and cancer of the recto-sigmoid junction: Colorectal cancer is cancer that starts in the colon or the rectum. Because cancers of the colon, rectum and rectal-sigmoid junction share common features and risk factors, they are often discussed together. There are a number of different kinds of colorectal cancers. More than 95% of colorectal cancers are a type of cancer known as adenocarcinomas. These cancers start in cells that form glands that make mucus to lubricate the inside of the colon and rectum. Other kinds of cancers include gastrointestinal stromal tumors, carcinoid tumors (arising from specialized hormone-producing cells) and sarcomas (arising from the blood vessels, muscles and connective tissues in the wall of the colon and rectum). Colorectal cancer is the third leading cause of cancer-related deaths in the United States when men and women are considered separately, and the second leading cause when both sexes are combined. The death rate (the number of deaths per 100,000 people per year) from colorectal cancer has been dropping in both men and women for more than 20 years. There are a number of likely reasons for this. One is that polyps are being found by screening and removed before they can develop into cancers. Screening is also allowing more colorectal cancers to be found earlier when the disease is easier to cure. In addition, treatment for colorectal cancer has improved over the last several years (ACS 2013; NCI 2012b).

African Americans have the highest colorectal cancer incidence and mortality rates of all racial groups in the United States. The reasons for this are not yet understood. Major chronic health or genetic risk factors include personal history of inflammatory bowel disease such as ulcerative colitis or Crohn's disease, family history of colorectal cancer, and certain inherited syndromes

such as familial adenomatous polyposis (FAP), Lynch syndrome, or Peutz-Jeghers syndrome (ACS 2013).

People with type 2 (usually non-insulin dependent) diabetes have an increased risk of developing colorectal cancer. Both type 2 diabetes and colorectal cancer share some of the same risk factors (such as excess weight). But even after taking these factors into account, people with type 2 diabetes still have an increased risk. They also tend to have a less favorable prognosis (outlook) after diagnosis (ACS 2013).

Lifestyle-related risk factors include diet, physical inactivity, obesity, smoking and heavy alcohol use. A diet that is high in red meats (beef, lamb, or liver) and processed meats (hot dogs and some luncheon meats) can increase colorectal cancer risk. Cooking meats at very high temperatures (frying, broiling, or grilling) creates chemicals that might increase cancer risk. Diets high in vegetables, fruits, and whole grains have been linked with a decreased risk of colorectal cancer, but fiber supplements do not seem to help (ACS 2013; NCI 2012b).

Long-term smokers are more likely than non-smokers to develop and die from colorectal cancer. Smoking is a well-known cause of lung cancer, but some of the cancer-causing substances in smoke dissolve into saliva and if swallowed, can cause digestive system cancers like colorectal cancer. Long-term smokers are more likely than non-smokers to develop and die from colorectal cancer. Smoking is a well-known cause of lung cancer, but some of the cancer-causing substances in smoke dissolve into saliva and if swallowed, can cause digestive system cancers like colorectal cancer (ACS 2013; NCI 2012b).

Kidney and renal pelvis cancer: Kidney cancer is among the ten most common cancers in both men and women. For reasons that are not totally clear, the rate of people developing kidney cancer has been rising steadily since the 1990s. This may be in part, due to better diagnostic tools such as modern imaging tests. There are number of different kinds of kidney cancer. Renal cell carcinoma is the most common type and account for about 90% of kidney cancers (ACS 2013; NCI 2012b).

The most important risks factors for kidney cancer are smoking and obesity. Men have about twice the risk as women, and African Americans have a slightly higher risk. Other risk factors include hereditary conditions (e.g. von Hippel-Lindau disease or Birt-Hoog-Dube syndrome, etc.), high blood pressure, the use of phenactin pain reliever or diuretics, or advanced kidney disease (ACS 2013; NCI 2012b).

Non-melanoma skin cancer: Skin cancer is by far the most common type of cancer. There are a number of different kinds of skin cancer. The most common types of skin cancer are basal cell carcinoma, squamous cell carcinoma, melanoma and lymphoma of the skin. The SEER site categorization separates melanoma and lymphoma from the basal cell and squamous cell skin cancers. In this investigation non-melanoma skin cancers were found to be elevated (ACS 2013).

Basal cell carcinoma is the most common type of skin cancer and the most common type of cancer (all types) in humans. About 80% of skin cancers are basal cell carcinoma. This cancer arises from the lowest layer of the epidermis (hence the name basal cell). If left untreated, basal

cell carcinoma can invade the underlying muscle and bone tissues. After treatment, recurrence of basal cell carcinoma occurs in about 50% of the patients. Squamous cell carcinoma accounts for about 20% of skin cancers. Squamous cell cancer arises from squamous cells in the outer layer (the epidermis) of the skin. This kind of cancer is most common on the face, ears, neck, lips and backs of the hand. Other, rare skin cancers include keratoacanthomas, Merkel cell carcinoma, and Kaposi sarcoma. These types of cancers account for less than 1% of non-melanoma skin cancers (ACS 2013).

Excessive exposure to sun light or other ultraviolet light source is the most important risk factor for non-melanoma skin cancers. Persons with light colored skin have a higher risk than persons with more natural skin pigment. People who smoke are more likely to develop squamous cell carcinoma, especially on the lip. Smoking is not known to be a risk factor for basal cell carcinoma. Non-melanoma skin cancers typically arise in older people, although they are arising more frequently in younger persons than in the past. Men have about twice the risk for basal cell cancers and three times the risk for squamous cell cancers. This probably due to a higher level of sun exposure. Exposure to certain chemicals such as arsenic, some pesticides, tar, coal, paraffin and some types of petroleum-based oils increase risk. Exposure to penetrating radiation is also a risk factor. Rare inherited skin conditions (e.g., xeroderma pigmentosum), sun damage (actinic keratosis), scars and chronic skin sores also increase risk. Merkel cell carcinoma and Kaposi sarcoma are associated with certain kinds of viral diseases (ACS 2013).

Prostate cancer: Prostate cancer is now one of the most serious oncological diseases in men. Nationally, the 2008 incidence rate is 156.0 cases of prostate cancer per 100,000 men, based on data reported to the SEER program. Prostate cancer is the second leading cause of cancer mortality. The death rate is 24.4 deaths due to prostate cancer per 100,000 men. Utah has higher rates than the United States. In Utah, the incidence rate for 2008 is 165.6 cases per 100,000 men and the mortality rate is 27.0 deaths per 100,000 men. From 1975 until the early-1990s, annual prostate cancer incidence and mortality rates had been increasing. Since the 1990 rates of have been declining (NCI 2012b).

The risk for prostate cancer increases with age. African American men have a higher risk than other races, and men with a family history of prostate cancer have a higher risk. High animal fat diets and vitamin D and/or E deficiency may increase the risk for prostate cancer. Exposure to pesticides, particularly organophosphate and triazine pesticides has been associated with increased risk for prostate cancer. Higher incidence of prostate cancer has been noted among men working in agricultural occupations and this may be related to increased risk of exposure to agricultural chemicals such as pesticides. Both arsenic and cadmium are significant environmental contaminants and exposure to these contaminants has been associated with a number of carcinogenic outcomes, including the development of prostate cancer. On the other hand, selenium and possibly zinc have been shown to be preventive agents for prostate cancer. Natural compounds known as carotenoid lycopenes, found in tomatoes and tomato-based products are also thought to be protective (ACS 2013; NCI 2012b).

Non-Hodgkin lymphoma: Non-Hodgkin's lymphoma is a type of lymphoma, a cancer that starts in white blood cells called lymphocytes. Lymphocytes are part of the body's immune system. There are two kinds of lymphomas: Hodgkin's lymphoma (named after Dr. Thomas Hodgkin,

who recognized it in 1832), and non-Hodgkin's lymphoma. These two main types of lymphomas differ in how they behave, spread, and respond to treatment. Lymphomas are cancers that arise in lymphocytes. Lymphocytes are a type of white blood cell that help the body fight infections. There are two major types of lymphocytes, known as B-cell lymphocytes and T-cell lymphocytes. T-cell lymphocytes are involved in producing substances that help other kinds of white blood cells fight infections or respond to injuries. B-cell lymphocytes make antibodies against germs. Lymphocytes are found throughout the body, but tend to collect in certain kinds of tissues including the lymph nodes (hence their name), spleen, bone marrow, and thymus. Lymphoma can start anywhere lymphocytes are found, but most often start in the lymph nodes in the upper part of the body. Lymphoma occurs when lymphocytes are produced in an out-of-control excessive rate (ACS 2013; NCI 2012b).

There are many types of non-Hodgkin's lymphoma and classifying them (even for doctors) can be confusing. Non-Hodgkin's lymphoma can start with either B-cell or the different kinds of T-cell lymphocytes. Overall, the risk of non-Hodgkin lymphoma is higher in men than in women, but there are certain types of non-Hodgkin lymphoma that are more common in women. In the United States, whites are more likely than African Americans and Asian Americans to develop non-Hodgkin lymphoma. Non-Hodgkin's lymphoma is more common in older people (ACS 2013).

People with weakened immune systems have an increased risk for non-Hodgkin's lymphoma. For example, people who receive organ transplants (kidney, heart, liver) are treated with drugs that suppress their immune system to prevent it from attacking the new organ. These people have a higher risk of developing non-Hodgkin's lymphoma. The human immunodeficiency virus (HIV) can also weaken the immune system, and people infected with HIV are at increased risk of non-Hodgkin's lymphoma. Some genetic (inherited) syndromes can cause children to be born with a deficient immune system. Along with an increased risk of serious infections, these children also have a higher risk of developing non-Hodgkin's lymphoma. These inherited immune deficiency diseases can be passed on to children, but people with non-Hodgkin's lymphoma who do not have these inherited diseases do not pass an increased risk of lymphoma onto their children (ACS 2013; NCI 2012b).

Some autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus (SLE, or lupus), celiac sprue (gluten-sensitive enteropathy), and others have been linked with an increased rate of non-Hodgkin's lymphoma. In autoimmune diseases, the immune system sees the body's own tissues as foreign and attacks them, as it would a germ. Lymphocytes (the cells from which lymphomas start) are part of the body's immune system. The overactive immune system in autoimmune diseases may cause lymphocytes to grow and divide more often than normal. This may increase the risk of them developing into lymphoma cells (ACS 2013).

In addition to HIV infections, other types of infections, for example human T-cell leukemia/lymphoma virus (HTLV-1) and the Epstein-Barr virus (EBV) may raise the risk of non-Hodgkin's lymphoma. These viruses infect lymphocytes and can directly affect the DNA of infected cell, helping to transform them into cancer cells. Almost all people living in the United States have been infected by EBV (the cause of mononucleosis), usually in their early childhood (ACS 2013; NCI 2012b).

Exposure to chemicals such as benzene and certain herbicides and insecticides (weed- and insect-killing substances) may be linked with an increased risk of non-Hodgkin's lymphoma. Some chemotherapy drugs used to treat other cancers may increase the risk of developing non-Hodgkin's lymphoma many years later. For example, patients who have been treated for Hodgkin's lymphoma have an increased risk of later developing non-Hodgkin's lymphoma. Human herpes virus 8 (HHV8), hepatitis C virus (HCV) and *Helicobacter pylori* are also known to increase the risk for developing non-Hodgkin's lymphoma (ACS 2013).

Studies of survivors of atomic bombs and nuclear reactor accidents have shown they have an increased risk of developing several types of cancer, including leukemia, thyroid cancer, and non-Hodgkin's lymphoma. Patients treated with radiation therapy for some other cancers, such as Hodgkin's lymphoma, have a slightly increased risk of developing non-Hodgkin's lymphoma later in life. This risk is greater for patients treated with both radiation therapy and chemotherapy (ACS 2013).

Some studies have suggested that being overweight or obese may increase your risk of non-Hodgkin's lymphoma. Other studies have suggested that a diet high in fat and meats may raise your risk. More research is needed to confirm these findings (ACS 2013).

Limitations: The public often wants public health investigations to determine if cancer risk can be linked to a putative environmental concern. The methodology (indirect standardized incidence ratio) used in this investigation does not have the capability to definitively link the study population's elevated cancer rates to any inherent or external risk factors including environmental exposures (dos Santos Silva 1999; Esteve et al. 1994; Jekel et al. 1996; Kingsley et al. 2007; Mann 2003). These kinds of cancer statistical reviews are based on annual incidence data reported to the UCR. The incidence of cancer per year is dependent on diagnosis of clinically manifested cancer. There are a number of limitations that impede this linkage. There is seldom any knowledge about the frequency, duration, or intensity of cancer victims to putative environmental concern. Cancer can have a variable length latency period between the time of exposure to the actual manifestation and diagnosis of cancer. Cancer can be present for some period before an individual seeks medical assistance that leads to diagnosis (Bray and Parkin 2009; Izquierdo and Schoenbach 2000; Parkin and Bray 2009; Thoburn et al. 2007). Cancer risk is thought to be the result of complex interactions between individual factors (e.g., genetics, behaviors, socio-economics, etc.) and environmental exposures (e.g., occupational exposures, domestic exposures, etc.). There is seldom sufficient information available to statistically control for the many non-environmental factors that contribute to cancer risk, or the exposure to other potential environmental risks that are not the putative environmental concern (Chaix et al. 2010; Merlo et al. 2012; Peterson et al. 2006; Prentice and Thomas 1993). For small populations, the incidence of cancer has a tendency to manifest arbitrary clusters. This tendency is a common phenomenon encountered when investigating the rate of rare diseases in a small population. Often, a few types of cancer may be statistically elevated for disparate periods, but that conclusion may change if the analytical periods are changed (Greenland et al 1986, 2000). Overcoming these limitations usually requires a comprehensive assessment of individual risk supported by a clear and consistent trend of elevated rates for a population.

This investigation used data from the UCR and US Census. In Utah, the diagnosis of cancer for all site categories is reportable to the UCR. When a Utah resident seeks diagnosis, a report is generated. The UCR will follow-up on the report to confirm information and collect additional factors about the case. This process occurs when cases are diagnosed in Utah, but may not occur if a case is diagnosed outside of Utah. The UCR may contain records of incidence of cancer in persons who recently moved to the study area prior to their diagnosis. The UCR may lack records on individuals who lived for most of their life in the study area but moved elsewhere before seeking diagnosis and treatment. These situations create ascertainment biases. For the purposes of diagnosis, the EEP assumes that the ascertainment bias is non-systematic, meaning that the “move-in” and “move-out” situations balance each other. It is highly unlikely that this assumption is true in all cases and can be a significant limitation when the study population is small.

The EEP uses US Census data purchased from a commercial vendor of the data. The vendor as re-tabulated 1980, 1990, and 2010 data for the 2000 census block groups in Utah. Re-tabulation involves population distribution weighting based on census blocks that may not be consistent through time. The EEP estimates intercensal population counts using linear regression between the know census tabulations. This methodology does not account for short-term population growth dynamics such as the zoning and development of a new subdivision, which can occur in just a few years.

An investigation that uses population-based summary data rather than individual-level data is called an ecologic study by epidemiologists. This investigation is an ecologic study. An interpretation error commonly associated with ecologic investigations is to apply population-level risk findings to the individual. This kind of interpretation error is called an “ecologic fallacy.” For example, this study found the risk of lung cancer to be 2.25 to 3.67 times higher for the population in Moab. This risk metric should not be applied to individuals. An individual may have no risk or a risk several times higher than the population risk based on the individual’s genetic makeup, behaviors, exposure history, and susceptibility or resiliency to cancer (Greenland 2001; Greenland and Robins 1994; Izquierdo and Schoenbach 2000; Morgenstern 1982, 1995; Rockhill 2005).

CONCLUSIONS AND RECOMMENDATIONS

Significantly elevated cancer incidence rates were found for lung and bronchial cancer among men and the total population in Moab for the first six (1974-2004) of seven five-year analytical periods. The risk ranged from 2.5 to 3.7 times higher than expected for men and 2.3 to 3.2 times higher than expected for the total population. This finding suggests the presence of a temporal cluster of lung and bronchial cancer in Moab. Lung and bronchial cancer found to be elevated for women for two analytical periods but not following a pattern suggesting a temporal cluster form women.

Cervical cancer was found to be 5.8 times higher than expected for the 1980-1984 analytical period. This finding is meaningful for that period. Lung cancer and cervical cancers are preventable cancers. For persons developing these cancers, early detection and early intervention for these cancers improve the prognosis for recovery and quality of life experience. These

findings suggest that action should be taken to assist the Moab population in understanding the risks of these cancers and ways to reduce their individual risk.

Cancers of the oral cavity and pharynx, rectum and recto-sigmoid junction and non-melanoma skin cancers were elevated in one analytical period, but at a level that did not distinguish a cluster from random variation. The EEP does not recommend a need for action for these cancers, but does recommend that a follow-up investigation be conducted. This is particularly important for rectal and non-melanoma skin cancers which were elevated in the later part of the study period. Prostate cancer, non-Hodgkin lymphoma, and kidney and renal pelvis cancers presented an increasing change in rate and further suggest the need for a follow-up investigation.

The EEP recommends that SEUDHD work with the Utah Cancer Control Program for screening and health education services that could be made available to the study area communities. In addition, the EEP recommends that SEUDHD request a follow-up cancer statistical review after three to five years (2010 to 2014) of additional cancer data become available to EEP.

AUTHORSHIP, REVIEW AND CITATION

This report was prepared by:

Sam LeFevre
Environmental Epidemiology Program
Bureau of Epidemiology
Utah Department of Health

Mail: PO Box 142104, Salt Lake City, Utah 84114-2104
Street: 288 North 1460 West, Salt Lake City, Utah 84116
Phone: (801) 538-6191
Fax: (801) 538-6564
Email: slefevre@utah.gov

Certifying Reviewers:

Allyn K Nakashima, MD
State Epidemiologist
Utah Department of Health

Jennifer Brown, JD
Director, Bureau of Epidemiology
Utah Department of Health

Barry Nangle, PhD
Director, Center for Health Data
Utah Department of Health

Recommended Citation:

Environmental Epidemiology Program. 2013. *Cancer Incidence Statistical Review Investigating Moab, Grand County, Utah Covering the Period from 1975 to 2009*. Salt Lake City, UT: Utah Department of Health.

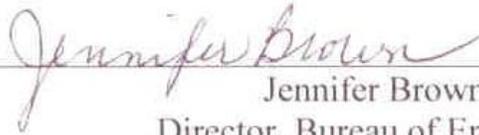
CERTIFICATION

This report titled “Cancer Incidence Statistical Review Investigating Moab, Grand County, Utah Covering the Period from 1975 to 2009” was prepared by the Environmental Epidemiology Program, Utah Department of Health. This report covers an investigation of cancer incidence using standard and approved methodology and procedures existing at the time the investigation herein reported was begun. Editorial and technical review was completed by UDOH certifying reviewers and program partners.

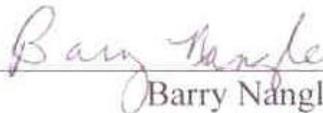
Approved by:



Allyn K Nakashima, MD
State Epidemiologist
Utah Department of Health



Jennifer Brown, JD
Director, Bureau of Epidemiology
Utah Department of Health



Barry Nangle, PhD
Director, Center for Health Data
Utah Department of Health

REFERENCES

Web links for citations of government or organizational websites may wrap onto multiple lines.

Alberg AJ, Samet JM. 2003. Epidemiology of lung cancer. *Chest* 123(Suppl 1):21S-49S.

Aberg AJ, Ford JG, Samet JM. 2007. Epidemiology of lung cancer: ACCP evidence-based clinical practice guidelines (2nd ed). *Chest* 132(Suppl 3):29S-55S.

ACS (American Cancer Society). 2009. Lifetime risk of developing or dying from cancer website. Available: <http://www.cancer.org/Cancer/CancerBasics/lifetime-probability-of-developing-or-dying-from-cancer> [accessed November 26, 2012].

ACS (American Cancer Society). 2013. Learn about cancer website. Available: <http://www.cancer.org/cancer/index> [accessed March 5, 2013]. Use this index to navigate to the relevant booklet from ACS.

Adamo MB, Johnson CH, Ruhl JL, Dickie LA. 2011. The 2011 SEER program coding and staging manual. NIH publication 11-5581. Bethesda, Maryland: National Cancer Institute. (See Appendix C - Site specific coding modules). Available: <http://seer.cancer.gov/tools/codingmanuals/index.html> [accessed March 27, 2012].

Anderson H, Blackmore C, Stanbury M, Simms E, Tai C, Tran A. 2012. A synopsis of the 2010 national assessment of state cancer cluster investigations and protocols. Atlanta, Georgia: Council of State and Territorial Epidemiologists.

Anderson RN, Rosenberg HM. 1998. Age standardization for death rates: implementation of the year 2000 standard. *National Vital Statistics Report* 47(3):1-17.

Armstrong B, Hutchinson E, Unwin J, Fletcher T. 2004. Lung cancer risk after exposure to polycyclic aromatic hydrocarbons: a review and meta-analysis. *Environmental Health Perspectives* 112(9):970-978.

Bell BS, Hoskins RE, Pickle LW, Wartenberg D. 2006. Current practices in spatial analysis of cancer data: mapping health statistics to inform policymakers and the public. *International Journal of Health Geographics* 5:49.

Bender AP, Williams AN, Johnson RA, Jagger HG. 1990. Appropriate public health response to clusters: the art of being responsibly responsive. *American Journal of Epidemiology* 132(Suppl 1):S48-S52.

Besag J, Newell J. 1991. The detection of clusters of rare disease. *Journal of the Royal Statistical Society, Part A* 154:143-155.

Blot WJ, Fraumeni JF. 1996. Cancers of the lung and pleura. In: *Cancer Epidemiology and Prevention*, 2nd Ed. (Schottenfeld D and Fraumeni JF, eds). New York, New York: Oxford University Press, 637-665.

Bray F, Parkin DM. 2009. Evaluation of data quality in the cancer registry: principles and methods. Part I: comparability, validity and timeliness. *European Journal of Cancer* 45(5):747-755.

Brenner DR, McLaughlin JR, Hung RJ. 2011. Previous lung disease and lung cancer risk: a systematic review and meta-analysis. *PloS ONE* 6(3):e17479:1-10.

Breslow NE, Day NE. 1987. Rates and Rate Standardization. In: *The Design and Analysis of Cohort Studies*, Vol 2. IARC Scientific Publication No 82. (Breslow NE, Day NE, eds). Lyon, France: International Agency for Research on Cancer.

Bronson RC, Figgs LW, Caisley LE. 2002. Epidemiology of environmental tobacco smoke exposure. *Oncogene* 21:7341-7348.

Caldwell GG. 1990. Twenty-two years of cancer cluster investigations at the Centers for Disease Control. *American Journal of Epidemiology* 132(Suppl 1):S43-S47.

CDC (Centers for Disease Control and Prevention). 2012. Leading causes of death. Available: <http://www.cdc.gov/nchs/fastats/lcod.htm> [accessed November 26, 2012].

Chaix B, Leal C, Evans D. 2010. Neighborhood-level confounding in epidemiologic studies: unavoidable challenges, uncertain solutions. *Epidemiology* 21(1):124-127.

Clapp R. 2000. Environment and health: 4 – cancer. *CMAJ* 163(8):1009-1012.

Coggle JE, Lambert BE, Moores SR. 1986. Radiation effects in the lung. *Environmental Health Perspectives* 70:26-91.

Copeland G, Lake A, Firth R, Xiao-Cheng W, Stroup A, Russell C, Boyuk K, Niu X, Schymura MJ, Hofferkamp J, Kohler B. 2011. *Cancer in North America: 2004-2008; Vol 1: combined cancer incidence for the United States and Canada*. Springfield, Ill.: North American Association of Central Cancer Registries. Available at: <http://www.naaccr.org/DataandPublications/CINAPubs.aspx> [accessed March 27, 2012].

Darby S, Hill D, Doll R. 2001. Radon: a likely carcinogen at all exposures. *Annals of Oncology* 12:1341-1351.

Dicker RC. 2002. A brief review of the basic principles of epidemiology. In: *Field Epidemiology*, 2nd Ed. (Greg MB, ed.). New York, NY: Oxford University Press.

dos Santos Silva, I. 1999. *Cancer epidemiology: principles and methods*. Lyon, France: International Agency for Research on Cancer.

EEP (Environmental Epidemiology Program). 2012. Private e-mail communications. Communications include the initial request from David Cunningham to initiate a review, EEP's response and coordinating communications to setup a community meeting.

EEP (Environmental Epidemiology Program). 2013. Private e-mail communications. Communications include additional coordinating instructions for a community meeting on January 8, 2013.

Esteve J, Benhamou E, Raymond L. 1994. Statistical methods in cancer research: IV descriptive epidemiology (IARC Scientific Publication Number 128). Lyon, France: International Agency for Research on Cancer.

Field RW, Smith BJ, Platz CE, Robinson RA, Neuberger JS, Brus CP, Lynch CF. 2004. Lung cancer histological type in Surveillance, Epidemiology and End Results registry versus independent review. *Journal of the National Cancer Institute* 96(14):1105-1107.

Frumkin H, Kantrowitz W. 1987. Cancer clusters in the workplace: an approach to investigation. *Journal of Occupational Medicine* 29(12):949-952.

Geolytics, Inc. 2002a. Census CD 1970, Release 2.0 on digital optical disk (CD). Information: <http://www.GeoLytics.com> [accessed November 26, 2012].

Geolytics, Inc. 2002b. Census CD 1990 long form in 2000 boundaries, Release 1.0 on digital optical disk (CD). Information: <http://www.GeoLytics.com> [accessed November 26, 2012].

Geolytics, Inc. 2002c. Census CD 2000 short form blocks for region 4 AK, AZ, CA, CO, HI, ID, MT, NV, NM, OR, UT, WA and WY, release 1.0 on digital optical disk (CD). Information: <http://www.GeoLytics.com> [accessed November 26, 2012].

Geolytics, Inc. 2012a. Census CD 1980 long form in 2000 boundaries, Release 1.0 on digital optical disk (CD). Information: <http://www.GeoLytics.com> [accessed November 26, 2012].

Geolytics, Inc. 2012b. Summary file 1 2010 in 2000 boundaries on digital optical disk (CD). Information: <http://www.GeoLytics.com> [accessed November 26, 2012].

Giovino GA. 2002. Epidemiology of tobacco use in the United States. *Oncogene* 21:7326-7340.

GJEM (Grand Junction Environmental Management). 2013. Moab, Utah, UMTRA project website. Available at: <http://www.gjem.energy.gov/moab/> [accessed February 19, 2013].

Godley LA, Larson RA. 2008. Therapy-related myeloid leukemia. *Seminars in Oncology* 35(4):418-429.

Goodman M, Naiman JS, Goodman D, LaKind JS. 2012. Cancer clusters in the USA: what do the last twenty years of state and federal investigation tell us. *Critical Reviews in Toxicology* 42(6):474-490.

- Greenland S. 2001. Ecologic versus individual-level sources of bias in ecologic estimates of contextual health effects. *International Journal of Epidemiology* 30(6):1343-1350.
- Greenland S, Robins J. 1994. Ecologic studies – biases, misconceptions, and counter examples. *American Journal of Epidemiology* 139(8):747-760.
- Greenland S, Schwartzbaum JA, Finkle WD. 2000. Problems due to small samples and sparse data in conditional logistic regression analysis. *American Journal of Epidemiology* 151(5):531-539.
- Greenland S, Thomas DC, Morgenstern H. 1986. The rare-disease assumption revisited. *American Journal of Epidemiology* 124(6):869-876.
- Izquierdo JN, Schoenbach VJ. 2000. The potential and limitations of data from population-based state cancer registries. *American Journal of Public Health* 90(5):695-698.
- Jekel JF, Elmore JG, Katz DL. 1996. *Epidemiology, biostatistics and preventive medicine*. Philadelphia, PA: WB Saunders Co.
- Jemal A, Siegel R, Ward E, Hao Y, Xu J, Murray T, Thun MJ. 2008. Cancer statistics, 2008. *CA: A Cancer Journal for Clinicians* 58(2):71-96.
- Kendall M. A new measure of rank correlation. 1938. *Biometrika* 30(1-2):81-93.
- King RJB, Robins MW. 2006. What is cancer? In: *Cancer biology*, 3rd Ed. (King RJB, Robins MW, eds.) San Francisco, California: Benjamin Cummings Publishing.
- Kingsley BS, Schmeichel KL, Rubin CH. 2007. An update on cancer cluster activities at the Centers for Disease Control and Prevention. *Environmental Health Perspectives* 115(1):167-171.
- Langeberg W, Contreras J, Hatch M, Kinney G, Sukhan S, Williams G. Cancer Cluster Workgroup: 2004. Protocol for investigating cancer clusters in Utah. Salt Lake City, UT: Utah Department of Health.
- Leone G, Fianchi L, Voso MT. 2011. Therapy-related myeloid neoplasms. *Current Opinion in Oncology* 23(6):672-680.
- Leone G, Mele L, Pulsoni A, Equitani F, Pagano L. 1999. The incidence of secondary leukemia. *Haematologica* 84(10):937-945.
- Mann CJ. 2003. Observation research methods. Research design II: cohort, cross sectional, and case-control studies. *Emergency Medicine Journal* 20:54-60.
- Merlo DF, Filiberti R, Kobernus M, Bartonova A, Gamulin M, Ferencic Z, Dusinska M, Fucic A. 2012. Cancer risk and the complexity of the interactions between environmental and host

factors: HENVINET interactive diagrams as simple tools for exploring and understanding the scientific evidence. *Environmental Health* 11(Suppl 1):S9.

MoabTailings. 2011. Moab UMTRA project information. Grand County, Utah. Available at: <http://www.moabtailings.org/index.htm> [accessed February 19, 2013].

Molina JR, Yang P, Cassivi SD, Shield SE, Adjei AA. 2009. Non-small cell lung cancer: epidemiology, risk factors, treatment and survivorship. *Mayo Clinic Proceedings* 83(5):584-594.

Morgenstern H. 1982. Uses of ecologic analysis in epidemiologic research. *American Journal of Public Health* 72:1336-1344.

Morgenstern H. 1995. Ecologic studies in epidemiology: concepts, principles, and methods. *Annual Reviews of Public Health* 16:16-81.

Morrone M. 2011. From cancer to diarrhea: the moving target of public concern about environmental health risks. *Environmental Health Insights* 5:87-96.

Mould RF. 2001. Depleted uranium and radiation-induced lung cancer and leukaemia. *British Journal of Radiology* 74(884):677-683.

NCI (National Cancer Institute). 2011a. SEER Cancer Statistics Review 1975-2008. Lifetime Risk (Percent) of Being Diagnosed with Cancer by Site and Race/Ethnicity: Males, 17 SEER Areas, 2006-2008 (Table 1.15) and Females, 17 SEER Areas, 2006-2008 (Table 1.16). Available: http://seer.cancer.gov/csr/1975_2008/results_merged/topic_lifetime_risk_diagnosis.pdf [accessed November 26, 2012].

NCI (National Cancer Institute). 2011b. SEER Cancer Statistics Review 1975-2008. Lifetime Risk (Percent) of Dying from Cancer by Site and Race/Ethnicity: Males, Total US, 2006-2008 (Table 1.18) and Females, Total US, 2006-2008 (Table 1.19). 2011b. Available: http://seer.cancer.gov/csr/1975_2008/results_merged/topic_lifetime_risk_death.pdf [accessed November 26, 2012].

NCI (National Cancer Institute). 2012a. Surveillance, Epidemiology and End Results (SEER) Program. Available: <http://seer.cancer.gov> [accessed November 26, 2012]. Use this index to navigate to the relevant booklet from NCI.

NCI (National Cancer Institute). 2012b. What you need to know about cancer. Available: <http://www.cancer.gov/cancertopics/wyntk/cancer> [accessed March 5, 2013]. Use this index to navigate to the relevant booklet from ACS.

Nermina O. 2005. Cancer incidence in Sarajevo region. *Medical Archives* 59(4):250-254.

Parkin DM, Bray F. 2009. Evaluation of data quality in the cancer registry: principles and methods. Part II: completeness. *European Journal of Cancer* 45(5):756-764.

Peterson ML, Sinisi SE, Van der Laan MJ. 2006. Estimation of direct causal effects. *Epidemiology* 17(3):276-284.

Prentice RL, Thomas D. 1993. Methodologic research needs in environmental epidemiology: data analysis. *Environmental Health Perspectives* 101(Suppl 4):39-48.

Rockhill B. 2005. Theorizing about causes at the individual level while estimating effects at the population level: implications for prevention. *Epidemiology* 16(1):124-129.

Rothman KJ, Boice JD. 1979. Epidemiologic analysis with a programmable calculator. NIH Publication 79-1649. Washington, DC: Government Printing Office.

Rothman KJ, Boice JD. 1982. Epidemiologic analysis with a programmable calculator, New Edition. Boston, MA: Epidemiology Resources, Inc.

Sahai H, Khurshid A. 1983. Confidence intervals for the mean of a Poisson distribution: a review. *Biometrical Journal* 35:857-867.

Sahai H, Khurshid A. 1996. Statistics in Epidemiology: Methods, Techniques and Applications. Boca Raton, FL: CRC Press, Inc.

Samet JM, Avila-Tang E, Boffetta P, Hannan LM, Olivo-Marston S, Thun MJ, Rudin CM. 2009. Lung cancer in never smokers: clinical epidemiology and environmental risk factors. *Clinical Cancer Research* 15(18):5626-5645.

Samet JM, Eradze GR. 2000. Radon and lung cancer risk: taking stock at the millennium. *Environmental Health Perspectives* 108(Suppl 4):635-641.

Schottenfeld D, Fraumeni JF. 1996. Cancer Epidemiology and Prevention, 2nd Ed. New York, NY: Oxford University Press.

Selvin S. 1996. Chapter 1: Measures of risk: rates and probabilities. In: Monographs in epidemiology and biostatistics, Vol 25: Statistical analysis of epidemiologic data. (Selvin S, ed.). Oxford, UK: Oxford University Press.

Sill H, Olipitz W, Zebisch A, Schulz E, Wolfler A. 2011. Therapy-related myeloid neoplasms: pathobiology and clinical characteristics. *British Journal of Pharmacology* 162(4):792-805.

Song R, Hall HI, Harrison KM, Sharpe TT, Lin LS, Dean HD. 2011. Identifying the impact of social determinants of health on disease rates using correlation analysis of area-based summary information. *Public Health Reports* 126(Suppl 3):70-80.

Stanbury M, Anderson H, Blackmore C, Fagliano J, Heumann M, Kass D, McGeehin M. 2012. Functions of environmental epidemiology and surveillance in state health departments. *Journal of Public Health Management and Practice* 18(5):453-460.

Stein C, Colditz G. 2004. Modifiable risk factors for cancer. *British Journal of Cancer* 90:299-303.

Thacker SB. 2000. Historical development. In: Principles and practice of public health surveillance, 2nd Ed. (Teutsch SM, Churchill RE, eds.). New York, NY: Oxford University Press.

Thacker SB, Qualters JR, Lee LM. 2012. Public health surveillance in the United States: evolution and challenges. *Morbidity and Mortality Weekly Report – Surveillance Supplement* 61(3):3-9

Thoburn KK, German RR, Lewis M, Nichols PJ, Ahmed F, Jackson-Thompson J. Case completeness and data accuracy in the Centers for Disease Control and Prevention's National Program of Cancer Registries. *Cancer* 109(8):1607-1616.

Thornton M. 2012. Standards for cancer registries, Vol II, data standards and data dictionary, 16th ed, Version 12.2. Springfield, Illinois: North American Association of Central Cancer Registries. Available: <http://www.naaccr.org/StandardsandRegistryOperations/VolumeII.aspx> [accessed March 27, 2012].

Thun MJ, Sinks T. 2004. Understanding cancer clusters. *CA Cancer Journal for Clinicians* 54(5):273-280.

Tomasek L, Darby SC, Swerdlow AJ, Placek V, Kunz E. 1993. Radon exposure and cancers other than lung cancer among uranium miners in West Bohemia. *Lancet* 341:919-923.

Trumbo CW. 2000. Public requests for cancer cluster investigations: a survey of state health departments. *American Journal of Public Health* 90(8):1300-1302.

Trumbo CW, McComas KA, Besley JC. 2008. Individual- and community-level effects on risk perception in cancer cluster investigations. *Risk Analysis* 28(1):161-178.

USCB (U.S. Census Bureau). 2004. Appendix A. Geographic terms and concepts in summary file 3: 2000 census population and housing, technical documentation. SF3/14 RV. Available: <http://www.census.gov/prod/cen2000/doc/sf3.pdf> [accessed November 26, 2012].

USCB (U.S. Census Bureau). 2012. Method for intercensal population estimates: 2000 to 2010. Available: http://www.census.gov/popest/methodology/2000-2010_Intercensal_Estimates_Methodology.pdf [accessed November 26, 2012].

USCB (U.S. Census Bureau). 2013a. 2007-2011 American Community Survey 5-Year estimates: Table DP02 Selected social characteristics in the United States. *American Fact Finder*. [The geographic location of the query included Moab CCD.] Available: <http://factfinder2.census.gov> [accessed February 20, 2013].

USCB (U.S. Census Bureau). 2013b. 2007-2011 American Community Survey 5-Year estimates: Table DP03 Selected social economic characteristics. American Fact Finder. [The geographic location of the query included Moab CCD.] Available: <http://factfinder2.census.gov> [accessed February 20, 2013].

USCB (U.S. Census Bureau). 2013c. 2007-2011 American Community Survey 5-Year estimates: Table DP04 Selected housing characteristics. American Fact Finder. [The geographic location of the query included Moab CCD.] Available: <http://factfinder2.census.gov> [accessed February 20, 2013].

USCB (U.S. Census Bureau). 2013d. 2007-2011 American Community Survey 5-Year estimates: Table DP05 ACS demographic and housing estimates. American Fact Finder. [The geographic location of the query included Moab CCD.] Available: <http://factfinder2.census.gov> [accessed February 20, 2013].

UCR (Utah Cancer Registry). 2012. 2012 cancer dataset for the Utah Environmental Public Health Tracking Network: containing public use data records for primary in-situ Utah resident cancers from 1973 to 2009. Electronic data transfer. Information: <http://ucr.utah.edu> [accessed November 26, 2012].

USDOE (U.S. Department of Energy). 2010. Department of Energy recovery act state memos: Utah. Washington, DC: US. Department of Energy, Recovery Clearinghouse. Available at: http://energy.gov/sites/prod/files/edg/recovery/documents/Recovery_Act_Memo_Utah.pdf [accessed February 19, 2013].

UDOH (Utah Department of Health). 2012. Utah Indicator-Based Information System for Public Health (IBIS-PH). [See Dataset Queries; Health Surveys; Behavioral Risk Factors Surveillance System]. Available: <http://ibis.health.utah.gov> [accessed November 26, 2012].

UEPHTN (Utah Environmental Public Health Tracking Network). Utah environmental public health tracking network. Information: <http://health.utah.gov/enviroepi> [accessed November 26, 2012].

Warner SC, Aldrich TE. 1988. The status of cancer cluster investigations undertaken by state health departments. *American Journal of Public Health* 78(3):306-307.

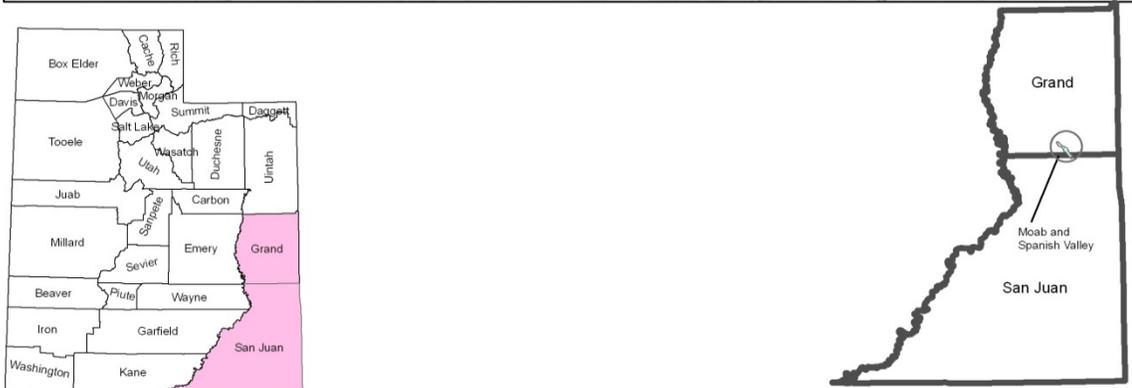
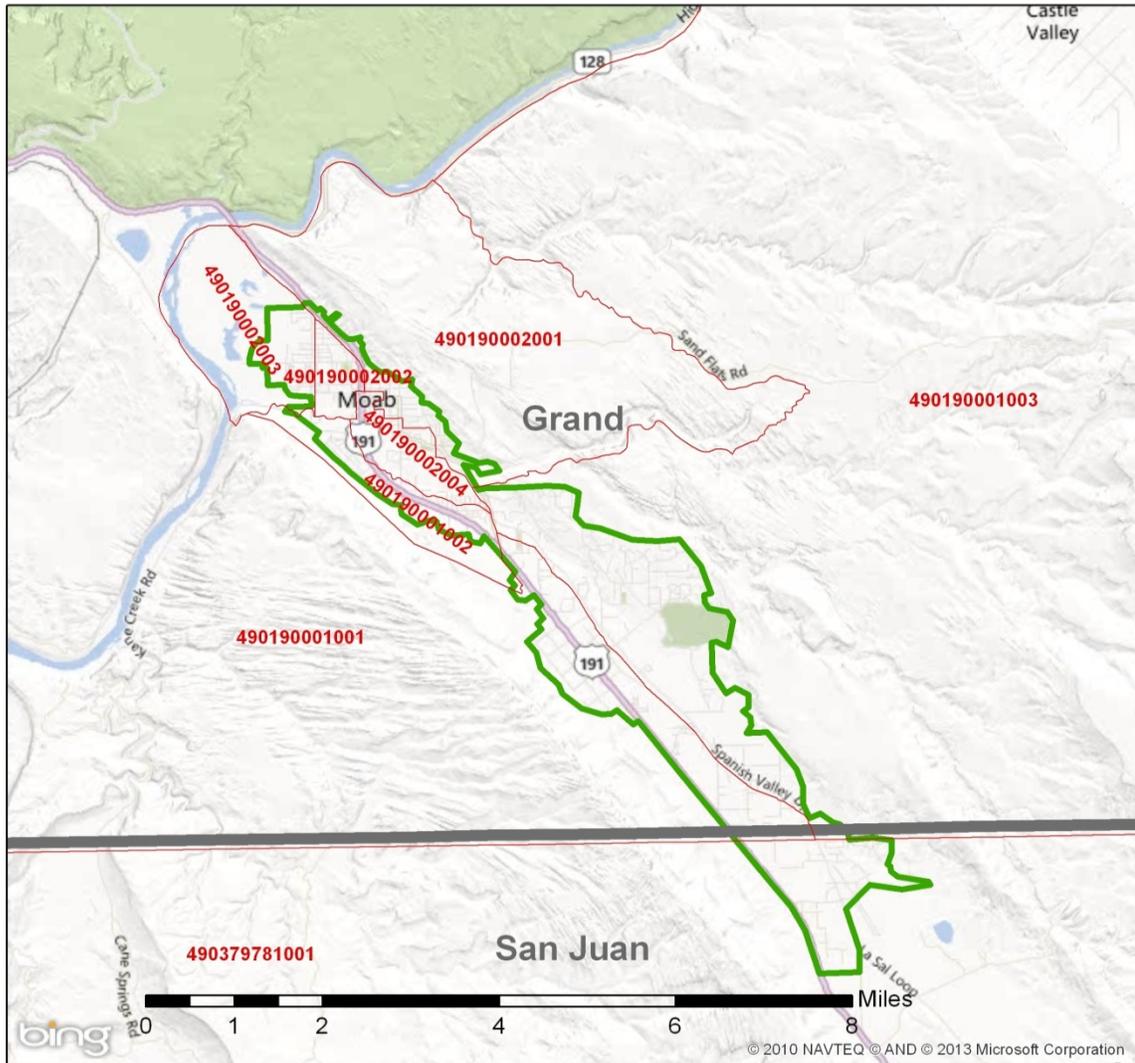
Weinberg RA. 2006. The nature of cancer. In: *Biology of cancer*. (Weinberg RA, ed.) New York, New York: Garland Science, Taylor & Francis Group.

WHO (World Health Organization). 2012. International Classification of Diseases for Oncology, 3rd Edition (ICD-O-3) website. Available: <http://www.who.int/classifications/icd/adaptations/oncology/en/> [accessed November 26, 2012].

Wilkins KL, Woodgate RL. 2008. Preventing second cancers in cancer survivors. *Oncology Nursing Forum* 35(2):E12-E22.

FIGURES

Figure 1. Map of Grand and San Juan counties showing the location of Moab and Spanish Valley, the study area for this investigation.



TABLES

Table 1. Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts ≤ 3 means the count could be 0 to 3. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
01 Oral cavity and pharynx	1975-1979	M	>3	20.7	1.44	0.39 – 3.68
		F	≤ 3			
		B	>3	10.5	1.10	0.30 – 2.83
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	>3	23.8	2.76	1.01 – 6.01 S
		F	≤ 3			
		B	7	13.9	2.20	0.88 – 4.54
	2000-2004	M	>3	21.3	2.64	0.96 – 5.75
		F	≤ 3			
		B	7	12.6	2.04	0.82 – 4.21
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts ≤ 3 means the count could be 0 to 3. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
02 Esophagus	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	> 3	12.6	3.02	0.81 – 7.74
		F	≤ 3			
		B	> 3	6.4	2.54	0.68 – 6.51

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts ≤ 3 means the count could be 0 to 3. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
03 Stomach	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	4	9.6	2.21	0.59 – 5.65
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
04 Small intestine	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
05 Colon	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	>3	21.1	1.02	0.27 – 2.61
		B	>3	12.5	0.64	0.21 – 1.48
	1985-1989	M	≤ 3			
		F	>3	30.1	1.43	0.52 – 3.11
		B	9	21.8	1.05	0.48 – 1.99
	1990-1994	M	≤ 3			
		F	>3	27.2	1.35	0.49 – 2.95
		B	9	20.0	0.96	0.44 – 1.82
	1995-1999	M	4	15.3	0.77	0.21 – 1.97
		F	5	19.6	0.96	0.31 – 2.25
		B	9	17.4	0.87	0.40 – 1.64
	2000-2004	M	≤ 3			
		F	>3	17.8	0.86	0.28 – 2.00
		B	8	14.0	0.69	0.30 – 1.35
	2005-2009	M	10	32.1	1.75	0.84 – 3.21
		F	4	13.3	0.68	0.18 – 1.73
		B	14	22.9	1.20	0.66 – 2.02

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
06 Rectum and recto-sigmoid junction	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	5	8.8	1.09	0.35 – 2.54
	2005-2009	M	≤ 3			
		F	>3	20.3	3.19	1.16 – 6.94 S
		B	9	14.9	1.88	0.86 – 3.56

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
07 Anus, anal canal and anorectum	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
08 Liver and interhepatic bile duct	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
09 Gallbladder and biliary bile ducts	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts “ ≤ 3 ” means the count could be 0 to 3. Case counts “ >3 ” means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar’s 95% confidence intervals (CI). Significance is indicated by an “S.” Sex code is “M” for male, “F” for female, and “B” for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
10 Pancreas	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	5	12.4	2.33	0.75 – 5.43
	1985-1989	M	>3	18.6	3.18	0.86 – 8.14
		F	≤ 3			
		B	>3	9.7	1.71	0.46 – 4.37
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	5	11.1	1.93	0.62 – 4.51
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	>3	13.3	1.81	0.49 – 4.64
		B	7	11.3	1.57	0.63 – 3.22

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
11 Other digestive system	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
12 Larynx	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts “ ≤ 3 ” means the count could be 0 to 3. Case counts “ >3 ” means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar’s 95% confidence intervals (CI). Significance is indicated by an “S.” Sex code is “M” for male, “F” for female, and “B” for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
13 Lung and bronchus	1975-1979	M	17	89.2	3.18	1.85 – 5.09 S
		F	4	22.4	2.77	0.75 – 7.10
		B	21	55.6	3.09	1.91 – 4.73 S
	1980-1984	M	>3	106.7	3.67	2.32 – 5.50 S
		F	≤ 3			
		B	26	61.1	3.03	1.98 – 4.44 S
	1985-1989	M	15	68.2	2.50	1.40 – 4.12 S
		F	8	38.1	2.88	1.24 – 5.67 S
		B	23	53.0	2.62	1.66 – 3.93 S
	1990-1994	M	17	72.1	2.53	1.47 – 4.05 S
		F	6	26.5	1.71	0.62 – 3.72
		B	23	49.4	2.25	1.43 – 3.37 S
	1995-1999	M	23	86.2	3.34	2.12 – 5.01 S
		F	14	53.7	3.07	1.68 – 5.16 S
		B	37	70.0	3.23	2.28 – 4.46 S
	2000-2004	M	24	82.1	3.38	2.16 – 5.03 S
		F	9	31.5	1.93	0.88 – 3.66
		B	33	57.0	2.81	1.93 – 3.94 S
	2005-2009	M	13	41.4	1.82	0.97 – 3.12
		F	7	23.0	1.23	0.49 – 2.53
		B	20	32.3	1.56	0.95 – 2.41

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
14 Other respiratory system	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
15 Bones and joints	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
16 Soft tissue (including heart)	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
17 Cutaneous melanoma	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	>3	26.7	2.44	0.79 – 5.70
		B	8	20.6	1.85	0.80 – 3.64
	1990-1994	M	>3	28.5	2.02	0.74 – 4.39
		F	≤ 3			
		B	8	19.7	1.48	0.64 – 2.92
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	5	10.6	0.70	0.23 – 1.64
	2000-2004	M	6	22.4	1.19	0.43 – 2.58
		F	5	20.1	1.28	0.41 – 2.99
		B	11	21.2	1.23	0.61 – 2.20
2005-2009	M	>3	23.8	0.86	0.35 – 1.78	
	F	≤ 3				
	B	10	17.7	0.76	0.36 – 1.40	

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
18 Other non-melanoma skin cancers	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	4	7.6	4.06	1.09 – 10.39 S
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts “ ≤ 3 ” means the count could be 0 to 3. Case counts “ >3 ” means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar’s 95% confidence intervals (CI). Significance is indicated by an “S.” Sex code is “M” for male, “F” for female, and “B” for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
19 Breast	1975-1979	F	10	55.5	0.92	0.44 – 1.70
	1980-1984	F	16	78.5	1.21	0.69 – 1.97
	1985-1989	F	20	97.3	1.23	0.75 – 1.89
	1990-1994	F	11	49.8	0.59	0.29 – 1.06
	1995-1999	F	25	99.1	1.11	0.72 – 1.63
	2000-2004	F	23	83.3	0.92	0.58 – 1.37
	2005-2009	F	20	68.5	0.75	0.46 – 1.16

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
20 Cervix	1975-1979	F	≤ 3			
	1980-1984	F	7	3.46	5.81	2.33 – 11.97 S
	1985-1989	F	≤ 3			
	1990-1994	F	≤ 3			
	1995-1999	F	6	27.5	4.64	1.69 – 10.09 S
	2000-2004	F	≤ 3			
	2005-2009	F	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
21 Uterus	1975-1979	F	5	27.5	1.28	0.41 – 2.98
	1980-1984	F	5	24.2	1.27	0.41 – 2.96
	1985-1989	F	≤ 3			
	1990-1994	F	6	26.4	1.34	0.49 – 2.91
	1995-1999	F	6	23.5	1.29	0.47 – 2.81
	2000-2004	F	6	21.4	1.20	0.44 – 2.62
	2005-2009	F	10	33.7	1.73	0.83 – 3.17

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
22 Ovary	1975-1979	F	≤ 3			
	1980-1984	F	5	24.7	2.24	0.72 – 5.23
	1985-1989	F	≤ 3			
	1990-1994	F	≤ 3			
	1995-1999	F	4	16.1	1.53	0.41 – 3.92
	2000-2004	F	5	18.4	1.78	0.57 – 4.15
	2005-2009	F	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
23 Other female genital	1975-1979	F	≤ 3			
	1980-1984	F	≤ 3			
	1985-1989	F	≤ 3			
	1990-1994	F	≤ 3			
	1995-1999	F	≤ 3			
	2000-2004	F	≤ 3			
	2005-2009	F	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts “ ≤ 3 ” means the count could be 0 to 3. Case counts “ >3 ” means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar’s 95% confidence intervals (CI). Significance is indicated by an “S.” Sex code is “M” for male, “F” for female, and “B” for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
24 Prostate	1975-1979	M	6	33.7	0.60	0.22 – 1.30
	1980-1984	M	19	90.4	1.38	0.83 – 2.16
	1985-1989	M	15	69.2	0.87	0.49 – 1.44
	1990-1994	M	15	62.9	0.47	0.27 – 0.78
	1995-1999	M	30	110.8	1.03	0.69 – 1.47
	2000-2004	M	38	128.2	1.10	0.78 – 1.51
	2005-2009	M	41	128.2	1.06	0.76 – 1.44

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
25 Testis	1975-1979	M	≤ 3			
	1980-1984	M	≤ 3			
	1985-1989	M	≤ 3			
	1990-1994	M	≤ 3			
	1995-1999	M	≤ 3			
	2000-2004	M	≤ 3			
	2005-2009	M	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
26 Other male genital	1975-1979	M	≤ 3			
	1980-1984	M	≤ 3			
	1985-1989	M	≤ 3			
	1990-1994	M	≤ 3			
	1995-1999	M	≤ 3			
	2000-2004	M	≤ 3			
	2005-2009	M	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI	
27 Bladder	1975-1979	M	≤ 3				
		F	≤ 3				
		B	≤ 3				
	1980-1984	M	≤ 3				
		F	≤ 3				
		B	≤ 3				
	1985-1989	M	>3		23.0	1.98	0.64 – 4.62
		F	≤ 3				
		B	6	14.0	1.92	0.70 – 4.19	
	1990-1994	M	≤ 3				
		F	≤ 3				
		B	≤ 3				
	1995-1999	M	≤ 3				
		F	≤ 3				
		B	4	7.6	1.12	0.30 – 2.87	
	2000-2004	M	≤ 3				
		F	≤ 3				
		B	≤ 3				
	2005-2009	M	≤ 3				
		F	≤ 3				
		B	4	6.4	1.07	0.29 – 2.74	

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
28 Kidney and renal pelvis	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	>3	1.56	2.23	0.60 – 5.71
		F	≤ 3			
		B	>3	7.9	1.37	0.37 – 3.50
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	5	8.9	1.20	0.39 – 2.79
2005-2009	M	≤ 3				
	F	>3	13.7	1.86	0.50 – 4.77	
	B	7	11.7	1.35	0.54 – 2.78	

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
29 Other urinary	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
30 Eye and orbit	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
31 Brain	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	4	10.4	1.89	0.51 – 4.84
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	4	9.0	1.57	0.42 – 4.02
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
2005-2009	M	≤ 3				
	F	≤ 3				
	B	4	8.1	1.41	0.38 – 3.60	

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
32 Other central nervous system	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts “≤3” means the count could be 0 to 3. Case counts “>3” means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar’s 95% confidence intervals (CI). Significance is indicated by an “S.” Sex code is “M” for male, “F” for female, and “B” for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
33 Thyroid	1975-1979	M	≤3			
		F	≤3			
		B	≤3			
	1980-1984	M	≤3			
		F	≤3			
		B	≤3			
	1985-1989	M	≤3			
		F	≤3			
		B	≤3			
	1990-1994	M	≤3			
		F	≤3			
		B	≤3			
	1995-1999	M	≤3			
		F	≤3			
		B	≤3			
	2000-2004	M	≤3			
		F	≤3			
		B	5	10.6	1.16	0.37 – 2.70
	2005-2009	M	≤3			
		F	≤3			
		B	≤3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
34 Other endocrine	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
35 Hodgkin lymphoma	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
36 Non-Hodgkin lymphoma	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	4	9.8	0.96	0.26 – 2.47
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	>3	18.2	1.18	0.38 – 2.76
		F	≤ 3			
		B	8	14.6	1.05	0.45 – 2.06
	2005-2009	M	>3	16.9	1.00	0.32 – 2.34
		F	≤ 3			
		B	7	11.9	0.79	0.32 – 1.64

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
37 Multiple myeloma	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
38 Lymphocytic leukemia	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	5	11.0	2.68	0.86 – 6.26
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
2005-2009	M	>3	14.3	2.23	0.60 – 5.71	
	F	≤ 3				
	B	>3	11.0	1.97	0.72 – 4.29	

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
39 Myeloid leukemia	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
40 Monocytic leukemia	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
41 Other leukemia	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	≤ 3			

Table 1 (continued). Analysis of the incidence of primary cancer diagnoses among study area residents between 1975 and 2009 reported to the Utah Cancer Registry by site code. The total number of cases is 12,695. Case counts " ≤ 3 " means the count could be 0 to 3. Case counts " >3 " means the case count was large enough to evaluate but is suppressed. Rates are indirect age-standardized incidence rate per 100,000 person-years. The SIRs are the standardized incidence ratio (SIR) with Byar's 95% confidence intervals (CI). Significance is indicated by an "S." Sex code is "M" for male, "F" for female, and "B" for both.

Cancer Site	Analytical Period	Sex	Case Count	Rate	SIR	95% CI
42 Other sites/types	1975-1979	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1980-1984	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1985-1989	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1990-1994	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	1995-1999	M	≤ 3			
		F	≤ 3			
		B	≤ 3			
	2000-2004	M	4	14.1	1.79	0.48 – 4.58
		F	5	18.0	2.25	0.72 – 5.24
		B	9	16.0	2.02	0.92 – 3.83
	2005-2009	M	≤ 3			
		F	≤ 3			
		B	>3	6.6	0.78	0.21 – 2.00

DEFINITIONS

- ACS** American Cancer Society. The ACS, first established in 1913, is a nationwide voluntary health organization dedicated to eliminating cancer. The society, headquartered in Atlanta, Georgia, has over 900 offices throughout the United States. ACS funding is used for patient support services, research, prevention, detection and treatment and society operations. For more information see: <http://www.cancer.org>.
- ACS** American Community Survey. The ACS is an ongoing survey that provides annual updates to population and demographic estimates derived from census data. The ACS is operated by the USCB. For more information see: <http://www.census.gov/acs/www/>.
- AGRC** Automated Geographic Reference Center. An agency within the Utah Department of Information Technology, responsible for maintaining a repository of geographic information system (GIS) data files and GIS functionality. For more information see: <http://gis.utah.gov>.
- ArcGIS** A complete desktop GIS software application for producing maps and conducting spatial analysis. This application is developed and distributed by ESRI. EEP uses version 10.0. For more information see: <http://www.esri.com/software/arcgis>.
- ARRA** American Recover and Reinvestment Act of 2009. This act authorized \$831 billion dollars of “stimulus” funding distributed in part through grants to governmental projects. Of this money, U.S. DOE received \$6.4 billion to clean up nuclear weapons production sites.
- CIS** Carcinoma in-situ is an early form of cancer that is defined by the absence of invasion of tumor cells into the surrounding tissue. Instead the lesion is flat or follows the existing architecture of the organ. In this state CIS, seldom cause clinical systems sufficient to prompt the person with CIS to seek medical assistance and are generally undetected. CIS can progress to invasive tumors and are therefore considered a precursor or incipient form of cancer.
- EEP** Environmental Epidemiology Program. A program within the Bureau of Epidemiology, Division of Disease Control and Prevention, UDOH. The EEP was established in 1996 and is responsible for investigating diseases related to the environment. The program has two sections. One section conducts surveillance and data management activities including managing the UEPHTN. The other section conducts health hazards risk assessment, including cancer investigations. The program is staffed by personnel with experience and expertise in environmental epidemiology, environmental sciences, toxicology, statistics, public health informatics and geomatics, and health education. For more information see: <http://health.utah.gov/enviroepi/>.

- ESRI** ESRI is a leading developer and supplier of GIS software and geographically referenced data. ESRI is headquartered in Redlands, California. The EEP uses the ArcGIS software application developed by ESRI. For more information see: <http://www.esri.com>.
- FAA** The Federal Aviation Administration. The FAA is a federal government agency charged with providing aviation support to airports and U. S. air space. The FAA supports flight operations for aircraft arriving to or departing from SLCIA, including recording radar tracking of inbound and outbound aircraft. For more information see: <http://www.faa.gov> and <http://www.faa.gov/FSDP/SLC>.
- GeoLytics** GeoLytics is a commercial vendor of census and demographic data calibrated to the 2000 census boundaries. The EEP has purchased 1970, 1980, 1990, 2000 and 2010 census data from GeoLytics to be the basis for estimating intercensal population counts for each of the 1481 census block group boundaries in Utah. Population counts are aggregated into 5-year age groups for each sex. For more information see: <http://www.geolytics.com>.
- GIS** Geographic Information Systems. A GIS includes computer software and geographically referenced data. The EEP uses ArcGIS as the computer software, and obtains data from ESRI or AGRC.
- ICD-O-3** International Classification of Disease - Oncology, 3rd Edition. The ICD-O-3 is one of a number of internationally established coding standards for coding site (topography) and histology (morphology) of neoplasms (cancers). For more information see: <http://www.who.int/classifications/icd/adaptations/oncology/en/>.
- NAACCR** North American Association of Central Cancer Registries. NAACCR was established in 1987 as a collaborative professional organization for cancer registries, governmental agencies and professional associations that work with cancer registries. All central cancer registries in the United States and Canada are members. The purpose of NAACCR is to promote standards and enhance the quality of cancer registry data. The NAACCR also promotes training, epidemiologic research, public health activities and patient care improvement policies related to cancer. For more information see: <http://www.naacr.org>.
- NCI** National Cancer Institute. The NCI is one of the National Institutes of Health and part of the U.S. Department of Health and Human Services. The NCI was established under the National Cancer Act of 1937 and is primarily responsible for conducting surveillance and research about cancer incidence, diagnosis, prevention, treatment, and rehabilitation. The SEER program is operated by the NCI. For more information see: <http://www.cancer.gov>.
- SAS** SAS (originally from “Statistical Analysis System”) is a globally recognized system of integrated computer software products provided by SAS Institute Inc. The SAS system includes a large variety of data manipulation and statistical

analysis processes. The EEP uses the desktop version 9.2. For more information see: <http://www.sas.com>.

- SEER** Surveillance, Epidemiology and End Results Program. The SEER program is an agency within the NCI. The SEER program works with state cancer registries to develop and disseminate incidence and mortality statistics about cancer in the United States. The SEER program also establishes standards for the analysis of cancer data and interpretation of cancer statistics. For more information see: <http://seer.cancer.gov>.
- SEUDHD** Southeastern Utah District Health Department. One of Utah's twelve local health departments. The SEUDHD is the health authority serving Carbon, Emery, Grand, and San Juan counties. For more information, see: <http://southeastuthealth.org>.
- UBRFS** Utah Behavioral Risk Factors Survey. The UBRFS is an ongoing telephonic survey conducted by the Office of Public Health Assessment, UDOH. This survey collects data about health-related behaviors in the non-institutionalized Utah adult population. For more information, see: http://health.utah.gov/opha/OPHA_BRFSS.htm.
- UCR** Utah Cancer Registry. The UCR is operated under authority from the UDOH by the University of Utah. The UCR was established in 1966 to be a statewide population-based cancer registry. Utah administrative rule requires the reporting of cancer diagnoses to the UCR. The UCR collaborates with the NCI, SEER and the North American Association of Central Cancer Registries to implement data standards for cancer data. The UCR provide cancer to the EEP through the UEPHTN. For more information, see: <http://ucr.utah.edu>.
- UDEQ** Utah Department of Environmental Quality. The UDEQ is one of the executive agencies within Utah state government. The UDEQ strives to safeguard public health and quality of life by protecting and enhancing the environment through the implementation, compliance monitoring and enforcement of environmental laws. For more information, see: <http://deq.utah.gov>.
- UDOH** Utah Department of Health. The UDOH is one of the executive agencies within Utah state government. The UDOH strives to improve health in Utah through promoting healthy lifestyles, evidence-based interventions, creating healthy and safe communities and eliminating health disparities. The EEP is a program within the UDOH. For more information, see: <http://health.utah.gov>.
- UEPHTN** Utah Environmental Public Health Tracking Network. The UEPHTN is a data warehouse that contains health outcomes, environmental and supporting data. Data from the UCR and population data derived from the USCB is warehoused in the UEPHTN. For more information see: <http://health.utah.gov/enviroepi/activities/EPHTP/NewEPHT/ephtpnew.htm>.

UMTRA Uranium Mill Tailings Remedial Action. In 1978, the U.S. Congress passed the Uranium Mill Tailings Radiation Control Act (UMTRCA, Public Law 95-604), which required the cleanup of inactive uranium-ore processing sites. In 1983, the U.S. EPA developed regulations (Title 40 CFR Part 192) to protect the public and the environment from potential hazards at these sites. The U.S. DOE is responsible for cleaning up UMTRA sites. For more information, see: <http://www.gjem.energy.gov/moab/> or <http://www.moabtailings.org>.

U.S. DOE U.S. Department of Energy. The U.S. DOE is one of the executive agencies within the federal government. The U.S. DOE is responsible for developing energy resources and technologies, nuclear security and resolving the environmental legacy of the cold war. For more information, see: <http://energy.gov>.

U.S. EPA U.S. Environmental Protection Agency. The U.S. EPA is one of the executive agencies within the federal government. The U.S. EPA is responsible for regulatory actions that protect human health and the environment from environmental health hazards. For more information, see: <http://www.epa.gov>.

USCB U.S. Census Bureau. Officially the “Bureau of the Census,” the USCB is an agency authorized by Federal law, within the U.S. Department of Commerce that is charged with preparing and conducting regular surveys and censuses of the U. S. population. In addition to the decennial population survey, the USCB conducts a number of other surveys and has recently implemented the ACS. For more information, see: <http://www.census.gov/>.

WHO An agency of the United Nations that deals with international health concerns and policies. For more information see: <http://www.who.int/en/>.

Cancer Incidence: The term incidence refers to new cases occurring in a period of time, usually annually. Cancer incidence is the number of new cases that occurred in a year. New cancer cases occur when a diagnosis is made. The 2009 national age-adjusted incidence rate is 4.64 cancer cases per 1,000 population per year. For more information, see: <http://www.cancer.gov/statistics/glossary/incidence>.

Cancer Prevalence: The term prevalence refers to the number of cases that exist either at a moment in time or during a period of time (e.g., annual, lifetime, etc.). When using this term, the time should be included. The 2009 national lifetime cancer prevalence rate is approximately 414.65 cases of cancer among 1,000 population. Cancer prevalence is the total number of cases that exist. For more information, see: <http://www.cancer.gov/statistics/glossary/prevalence>.

Cancer Incidence Rate: This is a ratio of the cancer incidence (the number of new cancer diagnoses) over the total population. When computing a multiple year rate, the total population added from each year of the rate period is used to get the rate. For

more information, see: <http://www.cancer.gov/statistics/glossary/incidence>.

Indirect Standardized Incidence Rate. The raw (sometimes called “crude”) disease incidence rate (number of case incidences per time period divided by the person-years per period) reflects reality. The raw rate is the simplest and most straightforward summary of the population experience. Interpretation of a disease incidence rate involves a comparison of that rate with some comparison or acceptable rate to determine if the rate in question is high or low. Because rates will almost always involve comparing two populations with two different age distributions, comparison of a raw disease incidence rate with a comparison rate is problematic. It does not make sense to compare the rate of disease of a relatively young population with a relatively older population for a disease that is more common in the elderly and be able to state with confidence that the disease rate is higher or lower than expected. For this reason, when the objective is to compare two rates, age standardized rates are preferable. However, it should be noted that the rate itself, once standardized, is not the exact disease burden. The standardized rate should be of the same magnitude as the raw rate.

The indirect standardization method is the preferable method when the disease count in each age group is small or zero. A disadvantage of the indirect method is that the rate is comparable to the comparison population used in its computation, but is not comparable to other population rates. For example, for this study, the study area cancer rates are adjusted using the Utah state population and therefore are comparable to the Utah state rates. However, they are not comparable to the county rates or to national rates.

The Indirect Standardized Rate for The study area (ISR_M) is calculated by:

$$ISR_M = \frac{C_M}{\sum_{age} \left(\frac{C_{U,age}}{P_{U,age}} P_{M,age} \right)} \times \left(\frac{C_U}{P_U} \right) \times 100,000$$

Where: ISR_M is the Indirect Standardized Incidence Rate for the study area.

C_M is the total cancer incidence count for the study area for a specific analytical period (e.g., 1990 - 1994).

$C_{U,age}$ is an age-group (e.g., 0 to 19 year in age, etc.) specific cancer incidence count for the state of Utah for a specific analytical period.

$P_{U,age}$ is the age-group specific count of person-years (e.g., number of 0-19 year olds in 1990 plus number of 0-19 year olds in 1991 plus number of 0-19 year olds in 1992 ..., etc.) for the state of Utah for a specific analytical period.

$P_{M,age}$ is the age-group specific count of person-years for the study area for a specific analytical period.

C_U is the total cancer incidence count for the state of Utah for a specific analytical period.

P_U is the total count of person-years for the state of Utah for a specific analytical period.

For purposes of presentation, it is standard practice to present rates per a population of 100,000 people. For example 60 cases per 100,000 people is easier to understand than 0.00006 cases per person.

$$E_M = \sum_{age} \left(\frac{C_{U,age}}{P_{U,age}} P_{M,age} \right)$$

E_M is the expected case count of cancer incidence for the study area for a specific analytical period. This is the denominator factor of the first term of the rate formula.

Standardized Incidence Ratio. The standardized incidence ratio (SIR) is a way of comparing two rates. When using the indirect standardized rate method, the SIR is the first term of the

$$SIR = \frac{C_M}{\sum_{age} \left(\frac{C_{U,age}}{P_{U,age}} P_{M,age} \right)} = \frac{C_M}{E_M}$$

formula to compute the rate.

$$\overline{SIR} = \frac{(C_M + k)}{E_M} \times \left[1 - \left(\frac{1}{3 \cdot (C_M + k)} \right) + \left(\frac{\pm 1.96}{3 \cdot \sqrt{C_M + k}} \right) \right]^3$$

The Byar's 95% confidence limits ($Z_\alpha = 1.96$) can be calculated for the SIR by:

Where:

SIR is the standardized incidence ratio. The bar over and under means the upper and lower confidence limits of the SIR.

C_M is the total case count of cancer incidence count for the study area for a specific analytical period.

E_M is the expected case count of cancer incidence for the study area for a specific analytical period.

K is a constant for symmetry. For the upper confidence limit, $k = 1$. For the lower confidence limit, $k = 0$.

± 1.96 is the normal distribution (Z_α) function for a 95% confidence interval. For the upper confidence interval it is a positive value. For the lower confidence interval it is a negative value.