

CANCER INCIDENCE STUDY

**A FOLLOW-UP STUDY OF CANCER INCIDENCE
IN MONTICELLO CITY, UTAH – 1973-2004**

MONTICELLO CITY, SAN JUAN COUNTY, UTAH

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Prepared by the

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EXECUTIVE SUMMARY

Monticello, Utah is a city with a population of 1,958, located in San Juan County in southeastern Utah near the border of Colorado. From 1943 through 1960 an active uranium and vanadium processing mill was located immediately adjacent to the town. Due to chemical and radioactive contaminants from mill activities, the mill site and affected surrounding properties were put on the National Priority List in 1986 and 1989, respectively. Remediation was completed in 2004. Since the remediation, completed exposure pathways no longer pose a public health hazard in the city of Monticello. However, potential exposure pathways still continue to exist but are monitored by the U.S. Department of Energy (DOE) and the U.S. Environmental Protection Agency (EPA).

Current and former residents of Monticello felt that they had experienced a substantial amount of harmful exposures to the contaminants emanating from the former mill, which was located just outside the city limits of Monticello. Residents perceived that the cancer rate in Monticello was increasing. In response to these concerns, the Utah Department of Health (UDOH), Environmental Epidemiology Program (EEP) conducted a cancer incidence study (completed in May 2006) to determine if the cancer rates were elevated. This study was based on a zip code (84535). The geography of the zip code, approximately 2,200 square miles in area, well exceeded the geography of the city of Monticello, approximately 2.5 square miles of land space. The zip code of residency was used to conduct the investigation because the majority of the persons from the city of Monticello, in the Utah Cancer Registry (UCR) provided a post office (P.O.) box rather than a street address. A P.O. box number is insufficient to determine whether someone lived within the city limits of Monticello or outside of the city in a more rural area.

The 2006 cancer incidence study applied the most recent data from the UCR. For that study, data were available for the years from 1973 through 2003. All cancer types with one or more cases during the study period were analyzed. However, the report particularly focused on cancer types that have risk factors associated with exposures to uranium and radon. These cancers include: bladder, gallbladder, kidney, leukemia, liver, lung, multiple myeloma, stomach, and thyroid. The state of Utah's population was used as the comparison population.

The 2006 study did not find conclusive evidence that the rates in the city of Monticello and surrounding area (zip code 84535) were increasing at a greater frequency than the rest of the state of Utah. However, the study was limited by the small population size and by the possibility that residents sought care out-of-state or permanently migrated out of the area. Those limitations affected the ability to correctly count all cancers that might have resulted from exposure in Monticello.

This report presents the findings of a follow-up study to the 2006 study. As part of this follow-up study, efforts were made by the EEP to correctly count all the cancers that occurred in Monticello from 1973-2004. To accomplish that task, the EEP used contact information from two surveys conducted by the Monticello Victims of Mill Tailings Exposure committee (VMTE) to identify self-reported cancers. The surveys were conducted in 1993 and 2005, respectively.

The EEP gathered contact information from the two surveys with assistance from the VMTE. The information was then sent to the UCR. The UCR conducted a survey study to collect detailed cancer and other pertinent information from residents, former residents, or next of kin. The information was used to validate the cancers that were self-reported. The survey study was conducted from November 2006 through June 2007.

The UCR confirmed 107 persons in the city of Monticello that were diagnosed with cancer from 1973-2004 from the survey study. An additional 49 cancer cases were also included from the Registry's database that were not part of the survey study. Persons who were diagnosed with cancer prior to 1973 and after 2004 were not used in this follow-up study for several analytical and data collection reasons. This report applied the same demographic variables and statistical methods from the 2006 study as explained below.

A cancer case was included in this follow-up study if it was the first diagnosis of cancer in an individual or if it was a second diagnosis, but the first diagnosis was an in situ cancer. Data from the UCR regarding the 156 persons, with a first primary cancer, who reside in or had resided in Monticello at the time they were diagnosed with cancer, were compared to the cancer data for the state of Utah. Due to the small number of cancer cases by type, only cancer types with three or more cases occurring during an analytical period/interval were analyzed.

The population of interest was defined as the city of Monticello. Population estimates for the state of Utah were obtained from the Utah Governor's Office of Planning and Budget. Population estimates for the city of Monticello from 1990-2004 were obtained from the U.S. Census; earlier populations were estimated based on these data and a population growth slope to determine the rate.

This report, as did the 2006 study, particularly focuses on cancer types that have risk factors associated with the contaminants of concern in the city of Monticello. As mentioned earlier, these include: bladder, gallbladder, kidney, leukemia, liver, lung, multiple myeloma, stomach, and thyroid.

Standardized incidence ratios (SIR) for each cancer type (with three or more cases) were calculated from 1973 through 2004, by five-year analytical periods, and a final seven-year analytical period starting with 1998. The cumulative study period 1973-2004 was included in the analysis for cancer types (cases) with three or more occurrence in any of the non-cumulative periods evaluated. The time periods used are as follows: 1973-1977, 1978-1982, 1983-1987, 1988-1992, 1993-1997, 1998-2004, and cumulative from 1973 through 2004. The cancer rates for each cancer type for each analytical period was evaluated to determine whether the Monticello population had a greater risk or a lower risk of developing cancer as compared to the comparison population (Utah's population).

Ninety-five percent confidence intervals (CI) were used to determine whether the SIR was statistically significant. Age-adjusted cancer incidence rates were also calculated based on the 2000 U.S. standard population. Cases were analyzed by cancer type regardless of the age at diagnosis. Due to the small number of cancers in persons less than 18 years old, it was not possible to analyze those cases separately with meaningful results.

The cancers that were found to be statistically significantly elevated in at least one analytical period were lung and bronchial (1993-1997, 1998-2004, and 1973-2004) and stomach cancer (1998-2004). Lung and bronchial cancer was significantly elevated in three analytical periods. Both of these cancers, particularly lung and bronchial, have risk factors associated with exposure to the contaminants from the former uranium mill. Unlike lung and bronchial cancer, stomach cancer had a small number of observed cases (and expected number) reducing the reliability of the significant SIR.

This study had several limitations. The small size of the Monticello population limited the ability of this study to reliably detect statistically significant elevations in cancer rates. This limitation is referred to as low statistical power. This study was not able to evaluate cancer rates prior to 1973. Additionally, this study did not evaluate or adjust for individual factors such as smoking and family history, which also have an impact on cancer rates.

With the assistance of the UCR, the EEP evaluated cancer cases that were specifically limited to the city of Monticello from 1973-2004. This study did find evidence of elevated risks for lung and bronchial cancer in residents of Monticello. Stomach cancer was also elevated, but in only one analytical period (with small observed numbers) reducing the reliability to detect a true difference in the observed cases. Therefore, chance may be a plausible explanation for the significant outcome. However, the significant elevated outcomes for stomach cancer, and particularly lung and bronchial cancer, are consistent with the known exposures and are biologically plausible with prolonged exposures to the contaminants from the former mill; which may limit the significant occurrence by chance.

It must also be noted that this study does not provide evidence that common environmental exposures in the city of Monticello resulted in the significant elevations of lung and bronchial cancer, and possibly stomach cancer. However, the significant elevations, particularly in lung and bronchial cancer, do warrant further investigation and/or monitoring.

RECOMMENDATIONS

Potential exposure pathways in Monticello are currently monitored by the DOE and EPA. The EEP recommends continuation of monitoring program for potential exposure pathway until cleanup goals are met by DOE and the EPA.

The EEP recommends re-evaluating the cancer rates in Monticello when three years of cancer data has been collected by the UCR, to determine if the elevated cancer types identified by this 2007 cancer incidence investigation continue to be elevated.

The EEP recommends a more comprehensive study of the hazards and health effects of the Monticello Vicinity Properties (MVP) and the Monticello Mill Tailings Site (MMTS) in the form of a follow-up public health assessment (PHA).

The EEP recommends that further education be provided to the Monticello community and surrounding populations on the causes of cancer, the exposures the community has experienced, cancer prevention, and cancer screening activities.

The community of Monticello has requested that a dose reconstruction be conducted to specifically characterize the exposure levels associated with the contaminants from the MVP and MMTS that were experienced by the community. The EEP does not have the capability or resources to conduct a dose reconstruction. The EEP recommends that a dose reconstruction be conducted to augment the PHA and that the DOE, EPA, and the Agency of Toxic Substances and Disease Registry (ATSDR) provide assistance to complete this recommendation.

The EEP recommends discussing the results of this 2007 cancer incidence investigation with the residents of Monticello (current report).

PUBLIC HEALTH ACTION PLAN

Actions Undertaken:

In March 2006, the EEP contacted DOE, EPA, and ATSDR to discuss the feasibility of conducting a dose reconstruction and an assessment of the exposures. Due to funding issues and other concerns, no decision has been finalized by the DOE, EPA, or ATSDR.

In May 2006, the EEP presented the results of the 2006 cancer incidence investigation to the Monticello community and also assembled a panel consisting of representatives from DOE, EPA, and ATSDR to respond to community concerns regarding the feasibility of a dose reconstruction and past exposures associated with the MVP and MMTS.

In 2006 and 2007, the EEP collaborated with the Southeast Utah Health District to provide information to the Monticello community on the causes/risk factors associated with the development of cancer (particularly the cancers of concern), on cancer prevention, and on the contaminants from the MVP and MMTS. Cancer screening programs are also available to qualifying residents of Monticello.

In 2006, the EEP contacted the UDOH Native American liaison to assist in or provide information on the causes and prevention of cancer and information on the contaminants from the MVP and MMTS to the Native American populations in the surrounding area of Monticello. Cancer screening programs are available on the reservation to the Native American population.

In 2007, the EEP completed a community health assessment in Monticello to ascertain the concerns of the residents and develop additional health education programs and information.

In 2007, the EEP assisted the UCR in completing a survey study of self-reported cancers in Monticello to ascertain cancer cases that were diagnosed specifically in Monticello. The survey was completed in June 2007.

December 2007, the EEP completed the 2007 cancer incidence investigation that evaluates the cancer incidence in Monticello from 1973 through 2004.

Actions Underway:

The EEP is in the process of completing the PHA report that will characterize the contaminants from the MVP and MMTS and their potentially harmful exposures. The EEP will also incorporate this 2007 cancer incidence investigation into the PHA. That PHA report will also be made available to the community along with discussion of the results and implications. Anticipated completion date is February 2008

Actions Planned:

The EEP will provide the community with a copy of this 2007 cancer incidence investigation and the 2008 PHA and discuss the results and implications with the residents of Monticello and other interested parties. The EEP will also invite representatives from the DOE, EPA, and ASTDR to discuss a dose reconstruction that has been requested by the Monticello community.

The EEP will re-evaluate the cancer rates in Monticello when three years of cancer data has been collected by the UCR to determine through a small area analysis, if the elevated cancers identified by this 2007 cancer incidence investigation continue to be elevated.

The EEP will continue to collaborate with the Southeast Utah Health District to provide information to the Monticello community on the causes/risk factors associated with the development of cancer, on cancer prevention, and on the contaminants from the MVP and MMTS. The continuation of cancer screening programs will also be requested.

The EEP will continue to collaborate with the UDOH Native American liaison to assist in or provide information on the causes and prevention of cancer and information on the contaminants from the MVP and MMTS to the Native American populations in the surrounding area of Monticello.

The EEP will also provide additional health educational activities applying the 2007 community health assessment as a guide for addressing the community needs.

INTRODUCTION

Monticello, Utah is a city with a population of 1,958 (Census 2000) located in San Juan County in southeastern Utah near the border of Colorado. From 1943 through 1960 an active uranium and vanadium processing mill was located immediately adjacent to the town. Due to chemical and radioactive contaminants from mill activities, the Monticello Vicinity Properties (MVP) and the Monticello Mill Tailings Site (MMTS) and were put on the National Priority List (NPL) in 1986 and 1989, respectively. Remediation of the MVP completed in 1999 and the MMTS was completed in 2004 (DOE 2007a and DOE 2007b). Since the remediation, completed exposure pathways no longer pose a public health hazard. Because potential exposure pathways continue to exist, the U.S. Department of Energy (DOE) and the U.S. Environmental Protection Agency (EPA) continue to monitor both sites.

The residents of the city of Monticello have been concerned for almost 15 years that exposures associated with the former mill operations and remediation activities are resulting in elevated cancer rates. In response to these concerns, the Environmental Epidemiology Program (EEP) of the Utah Department of Health (UDOH) conducted a cancer incidence study to determine if the cancer rates were elevated. That study was completed in May 2006. That study was based on a zip code (84535) geography that included the city of Monticello. The geography (over 2,200 square miles) of that zip code was considerably larger than the geography (about 2.5 square miles) of the city boundaries of Monticello. That study did not find conclusive evidence that the rates in the city of Monticello and surrounding area were increasing at a greater frequency than the rest of the state of Utah.

This report presents the findings of a follow-up study conducted by the EEP utilizing methodology, to correctly count and evaluate all the cancers that occurred specifically in the city of Monticello from 1973-2004. The EEP collected contact information from two surveys conducted by the city of Monticello's Victims of Mill Tailings Exposure (VMTE) committee. This information was used to identify self-reported cancers that occurred in the city of Monticello. This self-reported cancer information was sent to the Utah Cancer Registry (UCR).

The UCR conducted a survey study to collect additional information from residents, former residents, or next of kin. The information was used to validate the cancers that were self-reported specifically for the city of Monticello. The survey data were compiled and evaluated from November 2006 through June 2007.

BACKGROUND

Residents of the city of Monticello were exposed to numerous chemical and radioactive contaminants resulting from activities of the former uranium-processing mill. Completed exposure pathways did exist in the past. Remediation at the site and area properties was completed in 2004 and the mill site and associated contaminants no longer pose a public health hazard from the completed exposure pathways. However, the history of contamination of the former uranium mill site and surrounding properties in the city of Monticello has raised several concerns among the residents of the city. The primary concern is a perceived increase in cancer among the residents and former mill workers. These concerns have been expressed at several governmental levels.

In 2006 the EEP conducted a cancer incidence study for the population in zip code 84535, which includes the city of Monticello and surrounding farm lands in approximately 2,200 square miles. The reason this geography was used was because many of the persons from San Juan County registered in the UCR provided only a post office (P.O.) box rather than a street address. A P.O. box number is insufficient to determine whether someone lived within the city limits of Monticello or outside of the city in a more rural area.

It is also possible that the UCR may have not captured all cancer cases associated with the city of Monticello. Due to the location of the town and the lack of a full-service medical facility in the immediate area, residents may have sought medical care outside of the state (e.g., in Colorado, Arizona, or New Mexico) (See Maps, Appendix A).

The EEP 2006 cancer incidence study found inconclusive evidence that cancer rates in zip code 84535 were elevated or increasing at a greater frequency than the state of Utah as a whole. As stated earlier, the geography of the zip code significantly exceeded the city boundaries of Monticello. Because of the geographic inequality, it was not possible for this study to identify cancer risks specifically for the city of Monticello.

Site Description of Uranium Mill

There are two NPL sites in Monticello. Those are the MVP and the MMTS. The MVP and MMTS were placed on the NPL in 1986 and 1989. Both sites are associated with the Monticello Uranium Mill (ATSDR 1997).

The MVP consists of off-site residential and commercial properties located within or near the city of Monticello. The city of Monticello, private residents, and the state of Utah own various surrounding properties (ATSDR 1997). No residences are located on the mill site; however, residences are adjacent to the north and east edges of the mill site.

The MMTS is a 110-acre abandoned uranium and vanadium processing mill in the city of Monticello. The DOE owned the MMTS site until 2000; at that time remediation work on the site was almost completed and the city of Monticello was given the land through the National Park Service (DOE 2002). The remediation action for the MMTS was completed in June 2004 (DOE 2007b). A map of the area can be found in Appendix A.

Operating History

The Vanadium Corporation of America opened a vanadium ore-buying station just outside the city of Monticello in late 1940 and began mill construction of what is now the MMTS in 1941. In 1943, Vanadium Corporation began producing uranium-vanadium sludge for the Manhattan Engineer District (ATSDR 1997).

Intermediate owners and operators of the MMTS included the War Assets Office; the Atomic Energy Commission; American Smelting and Refining Company; Galigher Company; Lucius Pitkin, Inc.; National Lead Company; the Bureau of Land Management; and the DOE. Mill operations were terminated on January 1, 1960. The ore-buying station remained open until March 1962 (ATSDR 1997). The mill tailings were stabilized by grading and covering with dirt

and rock between 1961 and 1962, and the actual mill building was dismantled in 1964 (DOE 2002). Contaminated soils from the ore-buying station were removed between 1974 and 1975 (ATSDR 1997).

Remediation Activities

In 1980, the Monticello Remedial Action Project was established to remove chemical and radiological hazards from the mill site and surrounding properties (ATSDR 1997). In 1983, separate remediation projects for the MMTS and the MVP were established. The DOE had primary responsibility for remediation activities at both sites.

The MMTS was divided into three distinct operable units (OU):

| | |
|-------------------|---|
| Operable Unit I | Mill Site Tailings and Mill Site Property |
| Operable Unit II | Peripheral Properties |
| Operable Unit III | Surface Water, Groundwater and Contaminated Sediments in Montezuma Creek Canyon |

Remediation activities were completed for OU I and OU II in 2001 (DOE 2004 and DOE 2007a). Remediation action for OU III was completed in 2004 (DOE 2007b). A detailed update description of the remediation activities are available at the following link: <http://www.epa.gov/superfund/sites/fiveyear/f2007080001838.pdf>.

Remediation of the MVP began in 1984 and was completed in 1999 (DOE 2007a). All contaminated materials were placed in a permanent repository south of the mill site.

CONTAMINANTS OF CONCERN

Radioactive and Chemical Contaminants

The following section discusses radiation and specific chemicals of concern.

1. Radioactive materials

The primary radiological exposures of concern come from uranium, and the long-life products of uranium decay, radium, and radon. Elevated levels of these radioactive elements were found in the mill site, in off-site surface soils north and east of the mill site (ATSDR 1997). For an in-depth discussion of radioactivity, please see the Agency for Toxic Substances and Disease Registry (ATSDR) Public Health Assessment for Monticello Mill Tailings (DOE) CERCLIS NO. UT3890090035 and Monticello Radioactively Contaminated Properties (aka Monticello Vicinity Properties)_CERCLIS NO. UTD980667208). Website: http://www.atsdr.cdc.gov/hac/PHA/monticel/mon_toc.html

2. Non-radioactive contaminants

Based on ATSDR analyses of surface soils, groundwater and surface water, the following non-radioactive contaminants are possible causes of concern: arsenic, beryllium, chromium, copper, lead, molybdenum, nickel, selenium, and vanadium (ATSDR 1997).

Specific Contaminants

Radioactive contaminants

- 1) Uranium (U-235 and U-238)

Both U-235 and U-238 decay results in alpha particle radiation. Alpha particles consist of two protons and two neutrons bound together (essential the nucleus of a helium atom). Because of the large size and relatively lower energy of alpha particle radiation, the radiation particles do not penetrate materials well. Health effects are limited to damage to surface tissues that come in contact with alpha particle emitters such as uranium. Damage is done when the alpha particle collides with, breaks or interacts with cellular molecules. If the damaged cell molecule is involved with cell regulation and growth, cancer can result. The health effects associated with oral or dermal exposure to uranium are not related to the element's radioactive properties; these types of exposures are not associated with cancer. Inhalational exposure to uranium ore has been associated with lung cancer (ATSDR 1999a); however, there is some evidence that the association is due to exposure to radon (a product of uranium and radium radioactive decay) rather than uranium itself (ATSDR 1997 and ATSDR 1999a). The major organ most affected by uranium toxicity is the kidneys (OHS 1994).

2) Radium (Ra-223, Ra-226, R-228)

Two isotopes (R-223 and R-226) of radium are also alpha particle emitters. Decay of the R-228 isotope results in the release of a beta particle. A beta particle is essentially a free electron. With smaller size and more energy, the beta particle can penetrate further into tissues and cause damage in more layers of tissue. Similar to alpha particles, cell damage results when the beta particle breaks or interacts with cellular molecules. Radium is a decay product of uranium and is therefore found in all uranium-bearing ores (ATSDR 1997). Oral ingestion of radium has been associated with bone sarcomas and head cancers (ATSDR, 1990a). Possible associations with liver and kidney cancers have also been found (Schottenfeld and Fraumeni 1996). The primary exposure of concern for radium is through the oral route via incidental soil consumption, such as when children play outside. ATSDR calculations indicate that radium ingestion in such a setting is insufficient to cause radiation exposure beyond the maximum recommended dose (ATSDR 1997). However, no data were available regarding historical levels of radium in the soil at the time of this report.

3) Radon (R-222)

Radon (R-22) is also an alpha particle emitter. Radon is a naturally occurring radioactive gas that is odorless and tasteless. The gaseous nature of radon allows it to penetrate deeper into the lung air ways. Radon is formed from the radioactive decay of uranium or more specifically from radium. Uranium and radium are found in small amounts in most rocks and soil (ATSDR 1990b and ATSDR 1999b).

Radon also undergoes radioactive decay. The resulting decay products (sometimes called daughter products) have very short lives. The chain of decay products eventually results in the formation of non-radioactive (also called stable) lead atoms (ATSDR 1990b and ATSDR 1999b).

Radon exposure (indoors) has been associated with lung cancer (ATSDR 1990b and Schottenfeld and Fraumeni 1996).

Non-radioactive contaminants

1) Arsenic

Inhalation of inorganic arsenic has been associated with lung cancer (ATSDR 2005a). Ingestion of inorganic arsenic has been associated with skin, bladder, lung, kidney, liver (Schottenfeld and Fraumeni) and digestive tract cancer (IARC 1980 and Fukuda et al.). EPA also lists inorganic arsenic as causing skin damage and cancer (USEPA 2006a). Organic arsenic is much less toxic than inorganic arsenic, but has been associated with cancer as well. The arsenic present in Monticello is in the inorganic form (ATSDR 1997).

Current levels of arsenic in drinking water sources are insufficient to cause adverse health effects. However, it is possible that children were exposed to and ingested elevated levels of arsenic when they swam in the tailings ponds when the mill was operational. It is also possible that workers had inhalational exposure to arsenic during mill operations. It is unknown whether the levels of arsenic in these settings were high enough to cause elevated cancer risk (ATSDR 1997).

Arsenic is considered a human carcinogen by the EPA and the International Agency for Research on Cancer (IARC) (USEPA 2006a and IARC 2006).

2) Beryllium

No association or risk has been found with oral ingestion exposure to beryllium and the development of cancer. Inhaled beryllium has been associated with lung cancer in humans and animals (ATSDR 2002). The current soil concentrations of beryllium are well below the levels that have been associated with cancer (ATSDR 1997). However, no data were available regarding pre-remediation levels of beryllium in the soil at the time of this report.

Beryllium is considered a probable human carcinogen by the EPA and a known human carcinogen by the IARC (USEPA 2006a and IARC 2006).

3) Chromium

Chromium exists in several different forms, of which one, Chromium III, is an essential nutrient. Chromium VI is the more toxic form of chromium, which has been associated with respiratory system cancers when it is inhaled. Current chromium levels in the soils are below the level of concern (ATSDR 2000). However, no data were available regarding pre-remediation levels of chromium in the soil at the time of this report.

Chromium VI is considered a human carcinogen by both the EPA and the IARC. Chromium III is considered not to be a carcinogen by the EPA and is considered not classifiable regarding carcinogenicity by the IARC (USEPA 2006a and IARC 2006).

4) Lead

The primary concern with lead exposure is neurological and other types of developmental damage in children. However, lead may be associated with elevated cancer risk; the data

are not clear on this issue and do not indicate an association with any single type or group of cancers (ATSDR 2005b).

Inorganic lead is considered a probable human carcinogen by the EPA and the IARC. Organic lead is not classifiable regarding carcinogenicity by the IARC (USEPA 2006a and IARC 2006).

5) Molybdenum

Molybdenum has not been found to be associated with cancer nor classified as a carcinogen (USEPA 2006b and IARC 2006).

6) Nickel

Respiratory tract cancers have been associated with inhalation of high levels of nickel; however, these studies were conducted in occupational settings where persons were exposed to many other known carcinogens in addition to nickel (ATSDR 2005c). No adverse effects were anticipated from the reported concentrations of nickel in off-site soil. In addition, the nickel present was in a poorly soluble form and is less likely to be absorbed (ATSDR 1997).

Nickel is classified as either a probable or a known human carcinogen according to the EPA depending on the chemical form. Nickel is classified as either a possible or a known human carcinogen according to the IARC depending on the chemical form (USEPA 2006c and IARC 2006).

7) Selenium

Selenium has not been found to be associated with cancer. Selenium is an essential nutrient at low doses and may actually have a protective effect for cancer. Selenium also antagonizes the negative health effects of arsenic exposure, and vice-versa (ATSDR 2003).

Selenium is considered not classifiable with regards to human carcinogenicity by either the EPA or IARC (USEPA 2006a and IARC 2006).

8) Thallium

The relationship between thallium and cancer has not fully been studied (ATSDR 1997).

Thallium is considered not classifiable with regards to human carcinogenicity by the EPA (USEPA 2006a and IARC 2006).

9) Vanadium

Vanadium has not been found to be associated with cancer (ATSDR 1997).

Vanadium is not assigned a carcinogenicity classification by the USEPA (USEPA 2006d). It is considered a possible human carcinogen by the IARC (IARC 2006).

EXPOSURE PATHWAYS

ATSDR identified several completed exposure pathways that were of historical, and possibly, ongoing concern (ATSDR 1997). These pathways are outlined in Table 1. None of these completed exposure pathways are of current concern since they were addressed by the various remediation activities.

Table 1. Past completed exposure pathways

| Path Name | Compounds | Exposure pathway elements | | | | |
|-------------------------------|---|---------------------------|---------------|--------------------|--|--|
| | | Source | Media | Point of exposure | Route of exposure | Exposed People |
| On-Site Surface Soils | Radium-226 Radon-222 | Tailings Piles | Surface soils | On-Site | Ingestion Inhalation Dermal Absorption | Workers Residents |
| Off-Site Surface Soils | Beryllium Chromium Lead Nickel Thallium | Tailings Piles | Surface soils | Off-Site | Ingestion Inhalation Dermal Absorption | Residents Farmers Ranchers Hunters Golfers |
| Off-Site Air | Radium-226 Radon-222 | Tailings Piles | Air | Off-Site Buildings | Inhalation | Residents |

* Adapted from ATSDR PHA, 1997

These exposure pathways refer to contaminated soils and tailings at the former mill site. The removal of these materials to a permanent storage site was completed in 2000 (DOE 2003). The permanent storage repository is located adjacent to the former mill site, is regularly monitored by DOE personnel and access to the site is limited to authorized personnel by a fence and gate, which is locked at night (DOE 2003).

Past exposures from the former mill site have been addressed by remediation by the DOE and EPA; however, individuals who worked and lived at the mill or played on the former mill site were also exposed to compounds through the completed pathways. Available data on the level and types of exposures are limited (ATSDR 1997).

The potential for other pathways continue to exist. The DOE and EPA are actively monitoring both the MVP and MMTS for contaminants that may result in future exposures through those potential pathways.

Groundwater and Surface Water (potential exposure pathways)

Radioactive and chemical contaminants from tailings piles have leached into the shallow alluvial aquifer, which is contiguous with Montezuma Creek. The alluvial aquifer is not currently used for drinking water, irrigation, or livestock. Potential exposure pathways to chemical contaminants may occur through incidental or recreational contact with the contaminated water. Watering and institutional controls are in place to prevent it from being used for these purposes in the future (DOE 2004). Remediation of the shallow alluvial aquifer and Montezuma Creek, as

part of OU III, was completed in 2004 (DOE 2007b). These activities addressed contaminants of concern in shallow groundwater and surface water.

The deeper Burro Canyon Aquifer, which is used as a drinking water source, is hydrologically isolated from the shallow alluvial aquifer by various shale layers. It is unlikely that contaminants would be able to reach this aquifer. However, there is ongoing monitoring of the aquifer to monitor for this possibility (DOE 2004).

FOOD CHAIN

There was the possibility of bioconcentration of contaminants in animal food products. In 1996, Utah Department of Environmental Quality (UDEQ) and EPA conducted a study examining the levels of contaminants in deer and cattle. The most commonly consumed parts of the animals (eatable soft tissues) were tested for concentrations of metals and radionuclides. The levels of metals and radionuclides in the Monticello deer and cattle were similar to levels in the reference animals (Everett et al. 1998).

For plant products, the primary sources of contamination are trace amounts of soil on the surface of the plant. Washing food prior to preparation, as is standard hygiene practice, should be sufficient for removing potential contaminants from the food (ATSDR 1997).

DEMOGRAPHICS

The city of Monticello is located just to the northeast of the geographic center of San Juan County, Utah. The population, according to the 2000 census, is 1,958. In contrast to the predominantly Native American population of San Juan County as a whole, 83% of residents in the city of Monticello are white. A high percentage of the population is under the age of 18 (37%), however this is consistent with the county as a whole (40%) and with Utah's overall population. Almost 90% of residents over 25 had a high school degree or GED; this is consistent with the Utah population (Census 2000).

Nearly 79% of households are owner-occupied in the city of Monticello. The median household income was almost \$36,000 compared to \$28,000 for the county as a whole (Census 2000).

LITERATURE REVIEW - CANCERS OF CONCERN

A review of the literature was conducted to review the data on cancer types associated with uranium milling in general and with the specific contaminants of concern discussed earlier. The following cancers have been associated with uranium milling (and its compounds), mining, and radon exposure: gallbladder, kidney, leukemia, liver, lung, multiple myeloma, stomach, and thyroid (Schottenfeld & Fraumeni 1996, Tomasek et al. 1993, Nermina 2005, OHS 1994), and Shpagina et al. 2005). In addition, the major organ most affected by uranium toxicity is the kidneys (OHS 1994). Please see Appendix B – Cancer Epidemiology, for a discussion of other common risk factors for these cancers.

UTAH CANCER REGISTRY (UCR) SURVEY STUDY

The primary objective of the UCR survey study was to verify cancers reported to the EEP by the VMTE committee in the city of Monticello. The Registry compared cancer cases identified by

the surveys to the existing cancer registry database. This study received Institutional Review Board (IRB) approval from the UDOH and the University of Utah. The survey review occurred between November 2006 and June 2007.

UCR Survey Study Procedures

The data collected by the Registry was stored in a Microsoft (MS) ACCESS database, which served as the administrative tracking database (i.e., tracked who was sent a survey, when surveys were mailed, the date the survey was returned to the Registry, etc.). All personal information collected by the Registry is confidential.

The respondents received a letter describing the Registry and the study; it provided instructions on what to do with the survey when complete; and information on who to call for questions or concerns. The survey packet contained requests for:

- (a) An Authorization to Release Information
- (b) Respondent Information
- (c) Patient Information, and
- (d) Cancer Information

The Registry conducted two major survey mailings. The first mailing totaled 579 survey packets that were sent to patients or their next-of-kin. The second attempt to reach patients or next-of-kin totaled 344 survey packets. The total of survey packets mailed out by the end of the survey was 613. The final response rate from former and current residents of the city of Monticello was 54.6% (335 responses/613 total sample).

UCR Survey Results

The disposition of reported cases (verification outcome) is presented in Table 2 (below):

Table 2. Final outcomes for the city of Monticello cancer survey study, November 2006 through June 2007.

| Utah Cancer Registry Survey Study Outcomes | Number | Percentage (%) |
|---|---------------|---------------------------|
| Initial Mailing | 579 | 94.5 |
| Additional Surveys Received (Not part of initial mailing) | 34 | 5.5 |
| Total Sample Size | 613 | 100 |
| | | |
| Total Respondents and/or Surveys Received | 335 | 54.6 |
| | | |
| Total Linked with UCR Database | 174 | 51.9 |
| | | |
| Out-of-State Resident at Diagnosis (non-Utah case) | 72 | 21.5 |
| Did not have cancer (per survey) | 47 | 14.0 |

Table 2 continued.

Final outcomes for the city of Monticello cancer survey study, November 2006 through June 2007.

| | | |
|---|-----|------|
| Diagnosed before registry began collecting data | 12 | 3.6 |
| Not a State-designated reportable cancer | 12 | 3.6 |
| Not yet reported (diagnosed in 2006 or later) | 6 | 1.8 |
| Unverifiable | 9 | 2.7 |
| Missing | 1 | 0.3 |
| Refused to complete survey | 1 | 0.3 |
| Other | 1 | 0.3 |
| Total | 161 | 48.1 |
| | | |
| Undeliverable | 55 | 19.8 |
| No Response (letter not returned) | 223 | 80.2 |
| Total Non-Responses | 278 | 45.4 |
| | | |
| Total Monticello respondents identified with cancer from 1973-2004. | 107 | 17.5 |

Source: Utah Cancer Registry Monticello Survey Study, 2007.

A total of 174 case matches were determined for the survey respondents to the cases registered in the UCR databases. From the 174 cases, a total of 107 respondents were identified as residents of the city of Monticello at the time they were diagnosed with cancer from 1973-2004. These are the cases included for this cancer incidence study in the city of Monticello from the UCR.

CANCER DATA, STUDY PROCEDURES, AND METHODS

A cancer cluster is defined as three or more cases occurring within a certain location or geographical area and time period (Aldrich and Griffith 1993). Rural areas generally have a small number of cancers by specific types. Therefore, ATSDR recommends against performing statistical analysis whenever there are fewer than three cases of the same type of cancer in a population (ATSDR 1993) during a given period of time. For this reason, only those cancers occurring three or more times in the city of Monticello in any time period/interval were included in this analysis.

Data for this investigation were obtained from the UCR, which receives reports on newly diagnosed cases from Utah hospitals, radiation therapy facilities, pathology laboratories, nursing homes, and physicians. Information was available on cancer site/type, sex, age group, residence, and year of diagnosis from 1973 through 2004. The year 2004 was the most recent year for which complete data were available and 1973 was the earliest year where completed cancer information was available.

The UCR confirmed 107 persons in the city of Monticello that were diagnosed with cancer from 1973-2004 from the survey study. An additional 49 cancer cases were also included from the

Registry's database that were not part of the survey study. The study period for this report is from 1973 through 2004 and includes a total of 156 cancer cases.

The cancer cases from the registry were examined in five-year intervals (referred to as analytical periods) except for the last time period, which had seven years (1973-1977, 1978-1982, 1983-1987, 1988-1992, 1993-1997, and 1998-2004). Separate analyses were also performed on combined data (cumulative) for the study period (1973-2004). This period only evaluated cancer cases with three or more occurrence in any of the five or seven year analytical periods.

As mentioned earlier in this report, there have been numerous contaminants of concern in the city of Monticello. Those contaminants have risk factors associated with various cancer types. As recommended by ATSDR, all cancer types with three or more cases during any of the analytical periods/intervals were analyzed. However, the analysis particularly focused on cancers that have risk factors associated with the exposure to the contaminants of concern in the city of Monticello.

The cancers evaluated for elevated rates during the study period are listed in Table 3. Cancer types with an asterisk (*) have risk factors associated with the contaminants of concern. See Appendix E for a list of the International Classification of Diseases for Oncology (3rd edition) codes for cancers used in this study.

Table 3. Cancers evaluated in the city of Monticello, Utah, 1973-2004.

| Cancers in Monticello, 1973 – 2004 | |
|---|--|
| Gastrointestinal Tract *Stomach Colon Skin, Bone, Soft Tissue Cutaneous Melanoma Respiratory Tract *Lung & Bronchial | Blood and Lymph Non-Hodgkin's Lymphoma Female-specific cancers Breast Male-specific cancers Prostate |

Source: Utah Cancer Registry, University of Utah, Salt Lake City, Utah, 2007.

The following cancer types had less than three cases reported during any of the analytical periods/intervals: oral cavity and pharynx, ovary, cervix, rectum, liver and intrahepatic bile duct, gallbladder, urinary bladder, pancreas, soft tissue, uterus, testis, thyroid, Hodgkin's lymphoma, lymphocytic leukemia, myeloma, other leukemia, and "other site-not specified cancers." The category "other site-not specified" was included in the analysis of all cancers combined.

The following cancer types had no cases reported in the city Monticello during the study period: esophagus, small intestine, anus, other digestive system cancers (excluding the sites already mentioned), larynx, other respiratory tract cancers (excluding larynx and lung/bronchial), bones and joints, vagina, vulva, and other female genital cancers (excluding the sites already

mentioned), other male genital cancers (excluding prostate and testicular), eye/orbit, non-brain central nervous system cancers, myeloid leukemia, monocytic leukemia, other non-epithelial skin, and other endocrine.

Melanoma, lung and bronchial, prostate, breast, colon, and non-Hodgkin's lymphoma accounted for 60% of the cancers in the city of Monticello from 1973 through 2004.

Population of Interest

The population chosen for analysis included only residents within the city limits of Monticello. Population denominator data for the city of Monticello data for 1990 and 2000 were obtained from the U.S. Census. Earlier population estimates for the city of Monticello were calculated using the average rate of growth for intercensal years of the city of Monticello and the Monticello Census County Division. The age-specific populations as early as 1973 through 1989 were estimated, based on the estimated total population and growth slope, using the age distribution of the San Juan County population.

The state of Utah's population was selected as the comparison population for this investigation. Population denominator data for the state of Utah was obtained from the Utah's Governor's Office of Planning and Budget.

Study Inclusion Criteria

The requirement for a cancer case to be included in this study is as follows: A cancer case was included if the person was or is a resident of the city of Monticello at the time of diagnosis. Regarding the comparison population, a cancer case was included if the person was a resident of the state of Utah at the time of diagnosis. For the purpose of analysis, the city of Monticello will be referred to as *Monticello* and the state of Utah will be referred to as *Utah*, unless otherwise specified.

Other additional considerations: A cancer case was included in the analysis if it represented the first primary cancer diagnosed in an individual. For example, if a person was diagnosed with breast cancer in 1990 and lung cancer in 2000, only the breast cancer was included in the analysis. This did not apply to persons whose first diagnosis of cancer was an in situ cancer. In that circumstance, the in situ cancer was not included in the analysis, but the subsequent cancer was. Treatments for cancer, such as chemotherapy and radiation, increase the likelihood that an individual will later develop cancer; therefore, it is not possible to determine whether the development of a second primary cancer is due to exposures the individual experienced prior to their first cancer or due to the treatment of the first cancer (UOF 2006).

Statistical Analysis

The following statistical methods applied to this study are similar to the previous EEP 2006 cancer incidence report.

The observed and expected numbers of cancer cases were compared using Standardized Incidence Ratios (SIR) for each period (Kelsey et al.1986 and Aldrich and Griffith 1993). The expected number of cancer cases was calculated by applying age-specific cancer rates for Utah as a whole to the age-specific population of Monticello. Five-year age groups from 0-4 to 20-24

were used, followed by 10-year age groups from 25-34 to 75+ for the indirect standardization. A single SIR was calculated for each cancer in each single period. No sub-analyses by age-group (e.g. for persons under 18 years old) were calculated due to small sample sizes. The statistical significance of the SIR was evaluated using 95% confidence intervals. A normal estimation of a Poisson distribution was used in order to account for the small number of observed cases (Frumkin and Kantrowitz 1987). SIRs and confidence intervals were only calculated for analytical periods in which there were three or more observed cases.

Chi-square tests for linear trend were performed for cancer from all sites and for cancer types when there were cases in at least five analytical periods, excluding the cumulative analytical period 1973- 2004. Fisher's exact test was used for all trend analyses to account for the small number of cases.

Age-adjusted rates for each cancer type and cancer from all sites were calculated per 100,000 person-years.

Interpreting SIRs and Confidence Intervals

An SIR is used to evaluate whether one population has a higher number of cancers than would be expected, if that population had the same age-specific cancer rates as Utah as a whole. An SIR is calculated by dividing the number of observed cancer cases by the expected number of cancer cases. An SIR of one (1.0) indicates that age-adjusted rates were equal and there was no increased risk. A SIR greater than one (1.0) suggests an increased risk for the study group, while a SIR less than one (1.0) suggests a decreased risk for the study group. An SIR might not be 1.0 either because there is a true difference in the number of cases or because of random variation in cancer rates. The confidence interval helps determine whether a high or low SIR is likely to have occurred due to chance or due to a real difference.

A confidence interval is used to determine statistical significance. Whenever an SIR, or other measure of association, is calculated, the result is only an estimate of the true risk. A 95% confidence interval (the numbers between the lower and upper confidence limits) gives a range of values that are more likely to include the true risk. In other words, there is a 95% chance that the true risk of the result exists somewhere in that range between the lower and upper confidence limits. If the confidence interval of an SIR includes 1.0, then the result is not statistically significant, because there is a 95% probability that the difference found is due to chance alone. If a confidence interval does not include 1.0, then the result is statistically significant because there is a 95% probability that the difference found is not due to chance alone; however, statistical significance alone does not prove that cancer risk is truly higher or lower than expected. Confidence limits are generally wide when the sample size (or the number of people in the study) is small. Wide confidence intervals indicate that the SIR is not very reliable or precise. See Appendix C for further discussion of the statistical methods used in this study.

RESULTS

The results presented below are for cancer types that were statistically significantly elevated in at least one analytical period (with the exception of cancer from all sites/types). Lung and bronchial cancer and stomach cancer demonstrated statistically significant elevations. Both of these cancer types have risk factors that have been associated with uranium milling, mining, and its products.

Cancer rates are *only adjusted* for age. Due to the small number of cancer types and the small geographical area of Monticello, all analytical periods where three or more cases occurred are depicted as ≥ 3 ; analytical periods where less than three cancers cases or zero cases occurred are depicted with a hyphen (-). This is to protect the confidentiality of cancer cases that occur in small areas, particularly small rural areas.

The results from this report may vary from the 2006 cancer incidence investigation due to new cancers reported to the UCR and this report combined related cancers for both Monticello and Utah.

Cancer from all sites

Cancer from all sites (all cancers) was examined by each five-year and seven-year analytical periods, and cumulatively from 1973 through 2004. There are three analytical periods where the SIR exceeded 1.0, most note-able the last two analytical periods/intervals (1993-1997 and 1998-2004). Although the rates for Monticello exceeded the rates for Utah in both analytical periods, they were not statically significant. The cumulative SIR (study period) was less than 1.0 (SIR = 0.97) (Table 4), but was also not statistically significant. The rates for Monticello have fluctuated from 1973 through 2004. Trend analysis did not demonstrate any statistically significant trends in either direction (increase or decrease) over the period of the study.

Table 4. Annual age-adjusted rates for all cancers combined by each analytical study period comparing Monticello to Utah – 1973-2004.

| Time Period | Monticello Rate per 100,000 | Utah Rate per 100,000 | Monticello Observed number cases ³ | Monticello Expected number cases | SIR ¹ | 95% CI ² |
|--|-----------------------------|-----------------------|---|----------------------------------|------------------|---------------------|
| 1973-1977 | 296.3 | 284.7 | ≥ 3 | 18.1 | 1.05 | 0.63 - 1.57 |
| 1978-1982 | 272.5 | 296.1 | ≥ 3 | 20.8 | 0.96 | 0.59 - 1.43 |
| 1983-1987 | 285.7 | 313.3 | ≥ 3 | 22.4 | 0.94 | 0.58 - 1.38 |
| 1988-1992 | 259.5 | 349.0 | ≥ 3 | 25.4 | 0.71 | 0.42 - 1.08 |
| 1993-1997 | 395.4 | 336.4 | ≥ 3 | 27.3 | 1.14 | 0.77 - 1.57 |
| 1998-2004 | 378.3 | 336.0 | ≥ 3 | 41.90 | 1.12 | 0.82 - 1.12 |
| 1973-2004 | 349.7 | 360.3 | ≥ 3 | 160.7 | 0.97 | 0.82 - 1.13 |
| ¹ Standardized Incidence Ratio | | | | | | |
| ² 95% Confidence interval | | | | | | |
| ³ Observed cases are presented as ≥ 3 when cases are greater than or equal to three or as – when cases are less than three in order to protect the confidentiality of the cases. | | | | | | |
| Data Source: Utah Cancer Registry, 1973-2004. | | | | | | |

Lung and Bronchial Cancer

The SIRs for lung and bronchial cancer in Monticello were statistically significantly elevated during three analytical periods: 1993-1997, 1998-2004, and 1973-2004, respectively. The SIRs for the analytical periods are as follows: 1993-1997 was 3.34 (95% CI = 1.32 - 6.27), 1998-2004 was 2.51 (95% CI = 1.07 - 4.56) and 1973-2004 was 1.94 (95% CI = 1.24 - 2.79) (Table 5). The incidence rates for lung and bronchial cancer have decreased in the analytical periods from 1993 through 2004. However, the rates for each analytical period evaluated exceeded the rates of Utah. The SIRs for analytical periods 1993-1997 and 1998-2004 exceeded 3.0 and 2.0. Due to

the small cases numbers in the analytical periods from 1978 through 1992, no statistical pattern or trend analysis was conducted. The number of lung and bronchial cases from 1993 to 2004 and cumulatively (1973-2004) were large enough to perform more reliable analysis.

Table 5. Annual age-adjusted incidence rates for lung and bronchial cancer by each analytical study period comparing Monticello to Utah – 1973-2004.

| Time Period | Monticello Rate per 100,000 | Utah Rate per 100,000 | Monticello Observed number cases ³ | Monticello Expected number cases | SIR ¹ | 95% CI ² |
|--|-----------------------------|-----------------------|---|----------------------------------|------------------|---------------------|
| 1973-1977 | 57.7 | 24.2 | ≥3 | 1.54 | 2.60 | 0.68 - 5.77 |
| 1978-1982 | - | - | - | - | - | - |
| 1983-1987 | - | - | - | - | - | - |
| 1988-1992 | - | - | - | - | - | - |
| 1993-1997 | 90.2 | 25.7 | ≥3* | 2.1 | 3.34 | 1.32 - 6.27 |
| 1998-2004 | 65.8 | 25.1 | ≥3* | 3.2 | 2.51 | 1.07 - 4.56 |
| 1973-2004 | 51.3 | 26.9 | ≥3* | 12.4 | 1.94 | 1.24 - 2.79 |
| ¹ Standardized Incidence Ratio | | | | | | |
| ² 95% Confidence interval | | | | | | |
| ³ Observed cases are presented as ≥3 when cases are greater than or equal to three or as – when cases are less than three in order to protect the confidentiality of the cases. | | | | | | |
| * Statistically significant increase (p = <0.05) from the expected number of cases. | | | | | | |
| Data Source: Utah Cancer Registry, 1973-2004. | | | | | | |

Stomach Cancer

Stomach cancer was statistically significantly elevated during analytical period 1998-2004. The SIR for this period was 6.14 (95% CI = 1.60-13.63) (Table 6). The number of observed cases and expected number were small. This accounts for the large SIR and wide confidence interval. This lessens the reliability of the significant outcome or SIR. The SIR for the study period (1973-2004) was elevated, but was not statistically significant. No statistical significant test for increased pattern or trend was performed.

Table 6. Annual age-adjusted incidence rates for stomach cancer by each analytical study period comparing Monticello to Utah – 1973-2004.

| Time Period | Monticello Rate per 100,000 | Utah Rate per 100,000 | Monticello Observed number cases ³ | Monticello Expected number cases | SIR ¹ | 95% CI ² |
|--|-----------------------------|-----------------------|---|----------------------------------|------------------|---------------------|
| 1973-1977 | - | - | - | - | - | - |
| 1978-1982 | - | - | - | - | - | - |
| 1983-1987 | - | - | - | - | - | - |
| 1988-1992 | - | - | - | - | - | - |
| 1993-1997 | - | - | - | - | - | - |
| 1998-2004 | 65.6 | 10.8 | ≥3* | 0.7 | 6.14 | 1.60 - 13.63 |
| 1973-2004 | 8.93 | 6.0 | ≥3 | 2.7 | 1.47 | 0.38 - 3.25 |
| ¹ Standardized Incidence Ratio | | | | | | |
| ² 95% Confidence interval | | | | | | |
| ³ Observed cases are presented as ≥3 when cases are greater than or equal to three or as – when cases are less than three in order to protect the confidentiality of the cases. | | | | | | |
| * Statistically significant increase (p = <0.05) from the expected number of cases. | | | | | | |
| Data Source: Utah Cancer Registry, 1973-2004. | | | | | | |

CANCERS NOT ASSOCIATED WITH URANIUM MILLING

The following cancer sites/types (with three occurrences or more) have not been found to be associated with uranium milling, mining, or its products/compounds: colon cancer, melanoma, breast cancer, prostate cancer, and non-Hodgkin's lymphoma. The results for these cancers, for which the rates were not elevated during any of the periods, are presented in Appendix D.

DISCUSSION

Cancer is a term that refers to the uncontrolled growth and spread of abnormal cells anywhere in the body. The term cancer does not refer to a single disease. It is an umbrella term for at least 100 different types of uncontrolled cell growth. Cancers of the same type and especially cancers of different types have been associated to many different causes. These include genetic predisposition, personal habits such as smoking, and environmental exposures (Schottenfeld and Fraumeni 1996).

The American Cancer Society estimates that 1,399,790 people were diagnosed with cancer in 2006 in the U.S. Approximately one out of every two men and one out of every three women will be diagnosed with some type of cancer during their lifetime. Although anyone can get cancer at any age, about 77% of all cancers are diagnosed in people age of 55 and older (ACS 2006 and SEER 2006).

The residents of Monticello have been concerned with a perceived increase of cancer in their community for almost 15 years. Completed exposure pathways, for potentially cancer-causing contaminants, have existed in Monticello in the past from the former uranium mill. In 2006 the EEP conducted a cancer incidence study in Monticello and the surrounding area (zip code 84535). The EEP did not find elevated risks for cancer in the residents of zip code 84535. This

study was limited by the small population size and by the possibility that residents sought care out-of-state or permanently migrated out of the area, limiting the ability to correctly count all cancers that might have resulted from exposures from the former mill in the Monticello area. In addition, the geographic area of the zip code 84535 significantly exceeded the geographic area of the city limits of Monticello. Although that study found elevated lung cancer rates during 1993-1997, this study was not able to conclusively determine whether or not cancer rates in the Monticello area and surrounding area were truly elevated or occurred in Monticello.

The purpose of this current (follow-up) study is to specifically evaluate cancers with three or more occurrences (in seven different periods of time) that occurred specifically within the city limits of Monticello from 1973-2004 (32-year period).

This study found two cancers that demonstrated statistically significant elevations in Monticello. Lung and bronchial cancer and stomach cancer were significantly elevated in at least one analytical period. Both of these cancers have risk factors associated with exposures to the contaminants of the former uranium mill.

Lung and bronchial cancer was significantly elevated in three analytical periods, 1993-1997, 1998-2004, and 1973-2004, respectively. The SIR for the analytical period 1993-1997 (3.94, 95% CI = 1.32 - 6.27) was just slightly under 4.0 and the SIR for analytical period 1998-2004 (2.51, 95% CI = 1.07 - 4.56) well exceeded 2.0. The SIR for the cumulative analytical period 1973-2004 (1.94, 95% CI = 1.24 - 2.79) was just slightly under 2.0 with smaller confidence limits. The SIR for analytical period 1993-1997 is much higher than the SIR found in the previous 2006 cancer incidence study. In that study, lung and bronchial cancer was significantly elevated during the same time period (SIR = 2.4, 95% CI = 1.02-4.34).

Cancer of stomach (1998-2004) was significantly elevated in only one analytical period. The scientific literature suggests that stomach cancer has causal risk factors that are associated with exposures to uranium mill contaminants. Due to the small case number of stomach cancers reported to the UCR, the confidence regarding the statistical significant outcome of the SIR is not reliably high.

In conducting cancer incidence (or cancer cluster) studies, it is important to examine the biological relevance of the results within this study. Lung and bronchial cancer was found to be significantly elevated in three of the analytical periods evaluated. This could be consistent with prolonged exposures to some of the uranium mill contaminants that include uranium, radon, arsenic, beryllium, nickel, and chromium. It is well documented in the scientific literature that the development of lung and bronchial cancer has been associated with uranium mill contaminants/product, particularly radon.

The latency period for lung cancer can be as long as 20 to 40 years before clinical manifestations are observed (Schottenfeld & Fraumeni 1996). It is reasonable to suppose that persons who were exposed to cancer-causing contaminants would not receive a cancer diagnosis until years after they had left or had limited contact in the area where they were exposed. Therefore, the significance of elevated lung and bronchial cancer in Monticello is consistent with the known exposures, the biological plausibility, and the latency period.

There are a number of other known risk factors that could account for these findings, including the use of tobacco products, other life style choices and behavior related risks, genetic predisposition, infectious disease history, and other environmental risks (MMWR 1990) not associated with the MMTS and MVP. A limitation of this study was the lack of ability (lack of data) to account for those risk factors.

Consistency of findings between studies is also an important aspect in interpreting results. Although this study period was from 1973-2004 in Monticello, ATSDR conducted a review of cancer-related mortality data in San Juan County from 1950 through 1980. ATSDR found increasing mortality over time due to lung and breast cancers; the statistical significance of these findings was not reported. Additionally, ATSDR also conducted an analysis of lung cancer mortality between 1967 and 1992 in Monticello residents. ATSDR found statistically significantly elevated odds of dying of lung cancer in Monticello residents as compared to other San Juan County residents (Odds Ratio= 2.5, 95% CI = 1.03-5.8) (ATSDR 1997). However, these findings involve data from time periods outside of the period investigated by this study and are not directly comparable to this study. Finally, the EEP 2006 cancer incidence report, although inconclusive, did find significantly elevated lung and bronchial rates during 1993-1997 (EEP 2006). This significant elevated period is consistent with one of the elevated analytical periods (1993-1997) found by this study for lung and bronchial cancer.

Relative to risk factors associated with the development of cancer of stomach. Ingestion of inorganic arsenic has been associated with cancer of stomach (digestive tract) (IARC 1980). Organic arsenic is much less toxic than inorganic arsenic, but has been associated with cancer as well. The arsenic present in Monticello is in the inorganic form (ATSDR 2005a).

Although the statistical confidence of the SIRs for stomach cancer is not reliably high, the significant outcome is consistent with the known exposures and is biologically plausible with prolonged exposures. There is an inconsistency concern, since the exposures occurred over a prolonged period of time (as early as 1943 until the completion of remediation in 2004), it is reasonable to expect significant elevations of this cancer throughout the study period, not just in one period. In a small community setting like Monticello and with small case numbers as with stomach cancer, epidemiological studies can rarely provide evidence that cancer (like stomach) is truly increasing. Therefore, chance may remain as a plausible explanation for the significant outcome. However, when looking at the latency period, stomach cancer can take from 15 to 20 years and possibly longer (NIOSH 2007) before clinical manifestations are observed. Because of this, past exposures from the mill are more relevant than current exposures as potential causes of cancer.

This study did find evidence of elevated risks for lung and bronchial cancer in residents of Monticello. Stomach cancer was also elevated, but in only one analytical period with small observed numbers, therefore reducing the reliability to detect a true difference in the observed cases. However, the significant outcomes, particularly with lung and bronchial cancer, are consistent with the known exposures and are biologically plausible with prolonged exposures to the contaminants from the former mill.

LIMITATIONS

Factors that must be considered in the development and etiology of most cancers, but could not be evaluated in this study include latency period (at point of exposure), population migration, personal habits, race, diet, familial history and other environmental exposures not related to the MMTS or MVP sites. The latency or induction period for most adult cancers can range from 10 to 30 years or more after initial exposure to a carcinogen (MMWR 1990). Therefore, to ascertain place and time of carcinogenic exposure becomes problematic. Migration into and out of Monticello also presents a problematic issue relative to exposure, latency, and case ascertainment.

Characterizing types of cancers, cancer rates, and determining causal relationships to environmental exposures without exposure measurements or data is difficult because humans live and work in many environments, and are exposed to complex mixtures of toxic pollutants at home, at work, and in the ambient environment. If cancer is diagnosed before or immediately after an individual experiences an exposure, it is unlikely that the exposure caused the cancer.

In areas with small populations (such as Monticello) the numbers of expected cases of a given cancer make it difficult to appropriately analyze small number of cancer cases with sufficient reliability. These types of cancer cluster investigations lack the statistical power to detect small or medium elevations in cancer rates. Unfortunately, there are few statistical methods available to evaluate small sample sizes to improve detection of elevated cancer rates. One method used to improve the detection is to increase the sample size. This could be accomplished by combining cancer cases from other rural areas with similar demographic characteristics and exposures as observed in Monticello. However, since most small rural areas use P.O. box numbers, this again presents problematic issues similar to the community of Monticello in identifying cases that reside in the selected community. Another method is to select one cancer that may be associated with the mill site contaminants, such as lung cancer, and conduct a retrospective case control study. A retrospective study looks backwards and examines exposures to suspected environmental risks and controls for other lifestyle factors. This method may be time consuming as it requires contacting patients who currently reside or formerly resided in Monticello or next-of-kin and requires additional resources.

Due to the long latency period of many cancers, it is possible that persons who were exposed to cancer-causing contaminants would not receive a cancer diagnosis until years after they had left or had limited contact in the area where they were exposed. Additionally, persons who have left an area/community still continue to be exposed to environmental contaminants and behavioral risk factors that can affect their cancer risk. Therefore, if a person was diagnosed with cancer, it would not be possible to determine whether their cancer was due to a recent exposure in their new environment or a past exposure. Persons who move into areas have also been exposed to environmental contaminants in their previous locations. Additionally, it is possible that people moved to Monticello and experienced different levels and different types of exposures to contaminants. The effect of this type of movement is unclear and would depend on many individual level factors and behaviors. Unfortunately, the methodology of this type of investigation is not able to analyze the effects of such migration on the community cancer risk.

An additional limitation of this study is the lack of data prior to 1973 and after 2004. The mill was operational from 1943 through 1960. The latency period of cancers associated with uranium milling is not fully established. Latency periods of 20 years have been seen in some studies. However, these studies are predominantly conducted in adult uranium workers and do not address latency time in children who are exposed to uranium milling (ATSDR 1999a). Lung and bronchial cancer, as an example, may have a latency period of up to 40 years. The lack of information on the frequency and duration of exposure, relative to the development of cancer is another limitation in this kind of study.

Other causes of cancer also play a role in determining cancer rates in a community. For example, smoking is, by far, the most common cause of lung cancer in the U.S. This study did not specifically examine smoking rates in Monticello. Currently, the smoking rates for Monticello are included in the rates for San Juan County and Grand County (combined), which significantly dilutes the actual smoking rate in Monticello (BRFSS 2007). However, if Monticello has higher rates of smoking than Utah as a whole, this may be a reason for the elevated lung and bronchial cancer rates rather than exposures associated with the mill. There are many other common risk factors for cancer that were not investigated by this study. It is not possible to draw any definitive conclusion about the cause of elevated cancer rates without also examining these other risk factors. See Appendix B for further discussion of cancer risk factors.

CHILD'S HEALTH CONSIDERATIONS

Children are at a greater risk than adults from some environmental hazards. Children are more likely to be exposed to contaminants because they play outdoors, often bring food into contaminated areas, and are more likely to make contact with dust and soil. Because children's bodies are still developing, children can sustain permanent damage if toxic exposures, to some contaminants occur during critical growth stages.

This study could not evaluate the incidence of pediatric cancers in Monticello. From 1973 through 2004 there were less than three cases of any cancer in persons between 0 and 18 years of age. Due to these extremely small sample sizes, it was not possible to analyze data for children separately from adults. Therefore, all cancer cases are analyzed together regardless of the age at diagnosis.

CONCLUSION

With the assistance of the UCR, the EEP evaluated cancer cases that were specifically limited to the city limits of Monticello, Utah, from 1973 through 2004 covering a 32-year period.

Residents of Monticello were exposed to numerous chemical and radioactive contaminants due to activities of the former uranium-processing mill for many years. The former mill began operations in 1943 and remediation of the site and other contaminated areas was not completed until 2004. Completed environmental exposure pathways did exist in the past and potential exposure pathways still exist. However, DOE and EPA continue to monitor these potential exposures to ensure that they do not become a public health hazard in the future.

This study did find evidence of elevated risks for lung and bronchial cancer in residents of Monticello. Stomach cancer was also elevated, but in only one analytical period (with small

observed numbers) reducing the reliability to detect a true difference in the observed cases. Therefore, chance may be a plausible explanation for the significant outcome. However, the significant elevated outcomes for stomach cancer, and particularly lung and bronchial cancer, are consistent with the known exposures and are biologically plausible with prolonged exposures to the contaminants from the former mill; which may limit the significant occurrence by chance.

It must also be noted that this study does not provide evidence that common environmental exposures, in the city of Monticello resulted in the significant elevations of lung and bronchial cancer, and possibly stomach cancer. However, the significant elevations, particularly in lung and bronchial cancer, do warrant further investigation and/or monitoring.

RECOMMENDATIONS

Potential environmental exposure pathways in Monticello are currently monitored by the DOE and EPA. The EEP recommends continuation of monitoring program for potential exposure pathway until cleanup goals are met by DOE and the EPA.

The EEP recommends re-evaluating the cancer rates in Monticello when three years of cancer data has been collected by the UCR, to determine if the elevated cancer types identified by this 2007 cancer incidence investigation continue to be elevated.

The EEP recommends a more comprehensive study of the hazards and health effects of the MVP and MMTS in the form of a follow-up public health assessment (PHA).

The EEP recommends that further education be provided to the Monticello community and surrounding populations on the causes of cancer, the exposures the community has experienced from the MVP and MMTS, cancer prevention, and cancer screening activities.

The community of Monticello has requested that a dose reconstruction be conducted to specifically characterize the exposure levels associated with the contaminants from the MVP and MMTS that were experienced by the community. The EEP does not have the capability or resources to conduct a dose reconstruction. The EEP recommends that a dose reconstruction be conducted to augment the PHA and that the DOE, EPA, and ATSDR provide assistance to complete this recommendation.

The EEP recommends discussing the results of this 2007 cancer incidence investigation with the residents of Monticello (current report).

PUBLIC HEALTH ACTION PLAN

Actions Undertaken:

In March 2006, the EEP contacted DOE, EPA, and ATSDR to discuss the feasibility of conducting a dose reconstruction and an assessment of the exposures. Due to funding issues and other concerns, no decision has been finalized by the DOE, EPA, or ASTDR.

In May 2006, the EEP presented the results of the 2006 cancer investigation to the Monticello community and also assembled a panel consisting of representatives from DOE, EPA, and ATSDR to respond to community concerns regarding the feasibility of a dose reconstruction and past exposures associated with the MVP and MMTS.

In 2006 and 2007, the EEP collaborated with the Southeast Utah Health District to provide information to the Monticello community on the causes/risk factors associated with the development of cancer (particularly the cancers of concern), on cancer prevention, and on the contaminants from the MVP and MMTS. Cancer screening programs are also available to qualifying residents of Monticello.

In 2006, the EEP contacted the UDOH Native American liaison to assist in or provide information on the causes and prevention of cancer and information on the contaminants from the MVP and MMTS to the Native American populations in the surrounding area of Monticello. Cancer screening programs are available on the reservation to the Native American population.

In 2007, EEP completed a community health assessment in Monticello to ascertain the concerns of the residents and develop additional health education programs and information.

In 2007, the EEP assisted the UCR in completing a survey study of self-reported cancers in Monticello to ascertain cancer cases that were diagnosed specifically in Monticello. The survey was completed in June 2007.

In December 2007, the EEP completed the 2007 cancer incidence investigation that evaluates the cancer incidence in Monticello from 1973 through 2004.

Actions Underway:

The EEP is in the process of completing the PHA report that will characterize the contaminants from the MVP and MMTS and their potentially harmful exposures. The EEP will also incorporate this 2007 cancer incidence investigation into the PHA. That PHA report will also be made available to the community along with discussion of the results and implications. Anticipated completion date is February 2008.

Actions Planned:

The EEP will provide the community with a copy of this 2007 cancer incidence investigation and the 2008 PHA and discuss the results and implications with the residents of Monticello and other interested parties. The EEP will also invite representatives from the DOE, EPA, and ASTDR to discuss a dose reconstruction that has been requested by the Monticello community.

The EEP will re-evaluate the cancer rates in Monticello when three years of cancer data has been collected by the UCR to determine through a small area analysis, if the elevated cancers identified by this 2007 cancer incidence investigation continue to be elevated.

The EEP will continue to collaborate with the Southeast Utah Health District to provide information to the Monticello community on the causes/risk factors associated with the development of cancer, on cancer prevention, and on the contaminants from the MVP and MMTS. The continuation of cancer screening programs will also be requested.

The EEP will continue to collaborate with the UDOH Native American liaison to assist in or provide information on the causes and prevention of cancer and information on the contaminants from the MVP and MMTS to the Native American populations in the surrounding area of Monticello.

The EEP will also provide additional health educational activities applying the 2007 community health assessment as a guide for addressing the community needs.

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APPENDICES

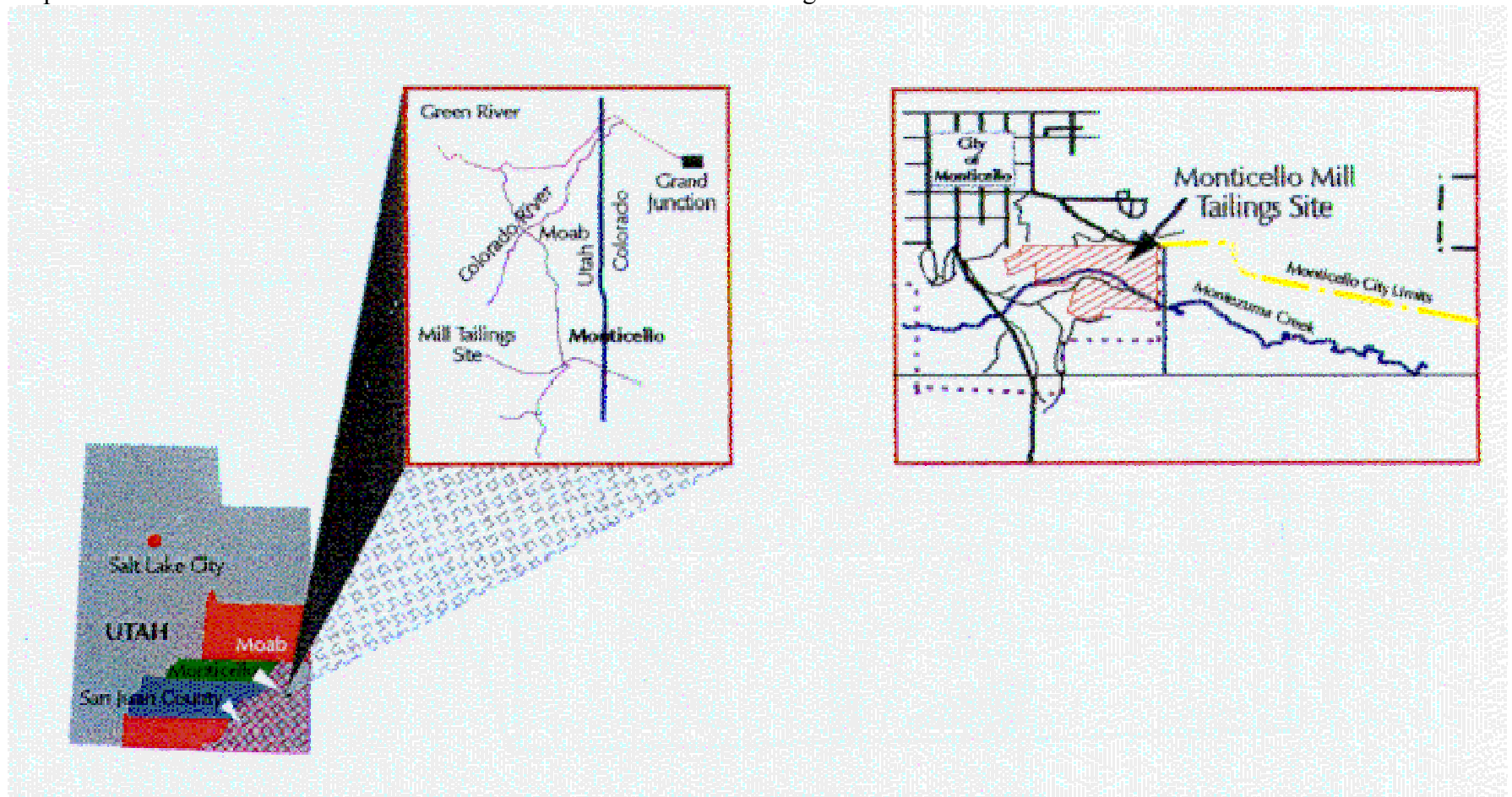
APPENDIX A – MAP OF MONTICELLO AREA

Map of Utah and location of the city of Monticello and the surrounding area.



MAP OF STUDY AREA

Map demonstration location of Monticello and the Monticello Mill Tailings Site



APPENDIX B – CANCER EPIDEMIOLOGY

Cancer is a name applied to many diseases with many different causes. Cancers are very common. Nearly half of all men and one-third of all women in the U.S. population will develop cancer at some point in their lives and 22% of the population will eventually die of cancer (ACS 2004). It is normal for cancer rates to fluctuate in smaller communities. During some years the rates are higher than expected and during other years the rates will be lower. Over time the average of rates tends to be what is expected.

Cancer Risk Factors

A number of factors can contribute to the development of cancer. These factors include a genetic predisposition, life style choices and behaviors such as tobacco use and diet (MMWR 1990), and infectious diseases (e.g., certain parasitic or viral infections) (Diego 1990). Although exposure to radiation can increase the risk of developing cancer, it is difficult to estimate risks from radiation since most of the radiation exposures that humans receive are very close to background levels. However, information from occupational studies among radiation workers have shown some evidence of a dose-related increase in the risk of cancer (leukemia) (Cardis et al. 1995 and Muirhead et.al. 1999). Exposure to environmental pollutants, radium, and radon, have been implicated in increasing the risks for cancer (Shottenfeld and Fraumeni, 1996).

When a subset of the population is found to have an increased rate of cancer, there are no definitive tests to determine which of the cancer cases are due to the unique risk factors present in that population (such as environmental exposures) and which cases are due to the risk factors present in the general population (such as smoking rates or genetics). Therefore, if the expected rate of a particular cancer in the general population is 100 cases and a particular occupational group is found to have 120 cases, no test currently can determine which 20 individuals developed the disease due to the specific risks associated with their profession (or environmental exposures) and which 100 would have occurred anyway.

Characterizing types of cancers, cancer rates, and determining causal relationships to environmental exposures without exposure measurements or data is difficult because people live and work in many environments and are exposed to complex mixtures of toxic pollutants at home, at work, and in the ambient environment. In addition, only a relatively small percentage of cancers can be attributed to environmental factors (Klaassen 1996).

Different cancers are associated with various environmental, behavioral and genetic risk factors. The following sections present some of the more common risk factors for the major cancer types that were investigated in this study.

Cancer Incidence

Cancer rates increased nationwide until the mid-1990's when they began to decline. Despite these declines, cancer remains the second most common cause of death in the U.S. after heart disease. There are several major factors that have contributed to high cancer rates. Tobacco exposure, primarily through smoking, causes most lung cancer in the U.S. Lung cancer is the leading cause of cancer death. Another factor is the longer lifespan of the modern U.S. population. Because cancer is caused by accumulated changes in our cells, it becomes more

likely as we get older. Thus, longer life-spans nationwide increase the amount of cancer seen in the population (MDCH 2000). In addition, higher rates of obesity in the U.S. probably also contributes to higher cancer rates (NCI 2003).

In addition to the decreasing rates of cancer, the survival rate once someone has been diagnosed has increased. This increase is due to improvements in the early detection and treatment of specific types of cancers such as breast, colon, and cervical cancers (NCI 2003; MDCH, 2000).

Unexplained cancer-related health disparities remain among population subgroups. For example, blacks and people with low socioeconomic status have the highest rates of both new cancers and cancer deaths (NCI 2003).

Childhood Cancers

The most common cancers in children are leukemia, brain tumors, and lymphomas. Nearly one in 450 children will be diagnosed with cancer before the age of 15 (MDCH 2000). Although some childhood cancers are associated with specific genetic, prenatal, and environmental factors, in most cases the cause of cancer is unknown. Factors that have been implicated in childhood cancers include genetics, infectious diseases, perinatal conditions, environmental pollutants, radiation, electromagnetic fields, and use of medications (Shottenfeld and Fraumeni, 1996). Few studies have been able to show a consistent link between cancer and these factors.

Cancers of Concern

The following cancer types have been found to be associated with the contaminants of concern found in the Monticello area.

Chronic Lymphocytic Leukemia

Chronic lymphocytic leukemia (CLL) is predominantly seen in the elderly. This form of cancer is more common in males than females for unknown reasons. Risk factors for CLL are not completely understood (UCR 1996). This cancer has not been convincingly linked to any myelotoxic¹ agent. Sufficient data does exist to rule out an association with ionizing radiation. CLL has been associated with chronic exposures to butadiene, ethylene oxide, non-ionizing radiation, herbicides, asbestos and solvents (Kipen and Wartenberg 1994). Risk factors such as radiation and chemical exposures commonly linked to other types of leukemia have not been shown to increase the risk of chronic lymphocytic leukemia (UCR 1996).

Gallbladder

Gallbladder cancer is not a common form of cancer; it is the 22nd most common cancer in the U.S. Gallbladder cancer occurs more frequently in woman than in men. Increased rates have been associated with a higher number of pregnancies (Moetman et al. 1994). Other risk factors associated with gallbladder cancer include gallstones, inflammation and infection of the biliary tract, liver flukes, ulcerative colitis, obesity, alcohol consumption, tobacco use, radiation exposure, familial history, and congenital defects (Shottenfeld and Fraumeni 1996). Elevated rates have also been seen in various occupations groups including textile and metal workers, automotive workers, rubber plant workers, chemical workers, aircraft mechanics, and wood

¹ Myelotoxic means toxic to cells (called stem cells) involved in the formation of blood cells.

finishing workers. No single environmental exposure has been implicated. Studies of uranium miners in Czechoslovakia found elevated lung, liver and gallbladder cancer (Tomasek et al. 1993).

Kidney and Renal Pelvis

In the U.S., two percent of new cancers are from malignant tumors of the kidney. Kidney cancer is more common in men than in women. Since the 1970's, incidence rates for this type of cancer have been increasing. The five-year relative survival rate for patients with kidney and renal pelvis cancer is about 50 to 65%. Cigarette smoking is causally linked to this type of cancer and probably accounts for a large percentage of these cancers in both men and women. Abuse of prescription analgesics is another risk factor. Obesity has also been found to be a risk factor for renal cell cancer. Coffee, tea, alcoholic drinks, and possibly increased meat consumption, are important risk factors. In some studies, asbestos-exposed workers and coke-oven workers in steel plants have an elevated risk of dying from kidney cancer (Schottenfeld and Fraumeni 1996; McLaughlin 2003). Ingestion of inorganic arsenic has been associated with the development of kidney cancer (ATSDR 2005a). There are also some studies, mainly of the correlation type, suggesting other cancers also to be related to indoor radon, especially leukemia, kidney cancer, and malignant melanoma (Axelson 1995).

Liver

The greatest risk factor for cancer of the liver is persistent infection with the either Hepatitis B or C Virus. This accounts for over three quarters of the world's cases. The remaining cases are caused by exposures that damage the liver, such as excessive alcohol consumption, and exposures that may be directly genotoxic, such as dietary aflatoxin² and tobacco use. Exposure to diagnostic thorium dioxide has been strongly associated with an increased risk of liver cancer. Occupational exposure to inorganic arsenic, vinyl chloride, and the organic solvent trichloroethylene (TCE) are also risk factors. There is a positive association of liver cancer with diabetes mellitus (Schottenfeld and Fraumeni 1996; Adami 2002).

Lung & Bronchial

Smoking is by far the leading risk factor of lung cancer. Exposure to passive smoke is also a risk factor. Exposure to radon and asbestos are factors leading to lung cancer, however, smoking in addition to these exposures greatly increases the cancer causing effects of asbestos and radon.

Excess lung cancers of all types have been reported in military personnel exposed to nuclear weapons and nuclear weapons testing. Smoking and radiation exposure also appear to have an additive effect on lung cancer. Occupational lung cancer may result from exposure to inorganic arsenic compounds (insecticides, pesticides, smelter workers, tin miners) (Schottenfeld & Fraumeni 1996).

The risk of lung cancer, mesothelioma, and asbestosis is increased in various asbestos industries, including mining, milling, textile, gas mask, friction products, insulation, shipyard, and cement workers. A high risk of lung cancer was reported in workers exposed to bis(chloromethyl)ether

² Aflatoxin, a group of related toxins primarily produced by two species of *Aspergillus* mold and that target the liver and immune system.

(BCME). Risk appears to decrease following cessation of exposure, suggesting that the chemical may affect late as well as early stages of carcinogenesis (Shottenfeld & Fraumeni 1996).

Other risk factors implicated in lung and bronchial cancers are exposure to coal, gas, nickel, polycyclic hydrocarbons, chromium, arsenic (Shottenfeld and Fraumeni 1996), chlormethyl ethers (Gowers et al. 1993), radon (Archer et al. 1973), and occupational exposures associated with mining (arsenic, asbestos and coal) (Ames et al. 1983, McDonald and McDonald 1987 Taylor et al. 1989) and uranium (ATSDR 1997). Risk increases when exposure to these contaminants occurs in conjunction with cigarette smoking.

Lung cancer may also be connected with breathing vinyl chloride over long periods (ATSDR 1997). In a study of workers exposed to dry cleaning solvents (carbon tetrachloride, TCE, and tetrachloroethylene) an excess of lung cancer was observed (Blair et al. 1979). Some studies have suggested a possible association between respiratory cancer with 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD or Dioxin) exposures (NTP 2001).

Tuberculosis has also been identified as a risk factor for lung and bronchial cancer. Tuberculosis and some types of pneumonia often leave scars on the lung. Because of the scarring it can increase the risk of developing the adenocarcinoma type of lung cancer (Zheng et al. 1987).

Stomach

Stomach cancer is the seventh leading cause of death in the U.S. In the past 50 years the incidence and mortality rates have fallen steadily and that trend is continuing. This decline is believed to be due primarily to improved nutrition. The Utah rates have been consistently lower than the national rates. The incidence of stomach cancer is observed more frequently as age increases. Environmental risk factors associated with stomach cancer include smoking, alcohol abuse, ionizing radiation, nitrate and related compounds (Shottenfeld and Fraumeni 1996) nickel oxide (Polednak 1981) exposure to metal dust, including beryllium, chromium, and nickel (Cocco et al. 1996) uranium mining compounds, inorganic arsenic (digestive tract) (IARC 1980), chromium, and coal mining (Stock 1994). Tobacco smoking is the most important risk factor associated with stomach cancer due to the high levels of nitrosamines in cigarettes (Risch et al. 1985, Hu et al. 1988, Forman 1987, and Hecht and Hoffmann 1991).

Thyroid Cancer

Thyroid cancer is an uncommon form of cancer and accounts for only one percent of all cancers in the U.S. (NCI 1996). Thyroid cancer occurs more often in women than in men and is most often found in young adults and teenagers. In women the peak occurrence of thyroid cancer is during their reproductive years. Radiation exposure is the only known risk factor strongly associated with an increased risk of thyroid cancer. External beam radiation treatment for medical therapy, acute gamma ray exposure from environmental sources (e.g., nuclear weapons testing, nuclear power plant accidents, etc.), and ingestion of short-lived radioactive iodine isotopes are the primary sources of radiation exposure that have been associated with increased risk of benign tumors and malignant thyroid cancer. Prescription drugs such as pentobarbital, meclizine, diphenoxylate, dicyclomine, griseofulvin, bisacodyl and senna have been associated with thyroid cancer (Shottenfeld and Fraumeni 1996). Familial history has also been associated with thyroid cancer (NCI 2007).

Urinary Bladder

An estimated 51,000 cases of urinary bladder cancer are diagnosed each year. Urinary bladder cancer accounts for six percent of all new cases of cancer among men and two percent of cases among women. Incidence and mortality due to bladder cancer increase sharply with age (Shottenfeld and Fraumeni 1996). The most important risk factor for bladder cancer is believed to be smoking (UCR 1996). Other environmental risk factors associated with urinary bladder cancer include chronic exposure to benzidine, 2-naphthylamine, aluminum, ionizing radiation, and hair dyes. Occupations associated with urinary cancer include dye workers, miners, leather workers, metal workers, chemical workers, petroleum workers, carpenters, welders, roofers, auto mechanics, and textile workers (Shottenfeld and Fraumeni 1996).

Other Cancers

The following cancers have not been found to be associated with exposure to any of the contaminants of concern found in and around Monticello.

Breast Cancer

Female breast cancer is the most commonly occurring cancer among females in the state of Utah. Currently more than 10% of Utah women will be affected in their lifetime (UCR 1996). Age and family history are the strongest risk factors for female breast cancer. Among post-menopausal women, breast cancer risk increases with weight and body mass. Early age at menarche and later age at first pregnancy have also been associated with increased risk of developing breast cancer (Shottenfeld and Fraumeni 1996). Other risk factors associated with breast cancer include alcohol, diet, and exposure to high doses of radiation (Longnecker et al. 1988 and NCI 1996).

Cervical Cancer

Cervical cancer is the 13th most common cancer diagnosed in the U.S. It is most common in younger women and is known to be caused by exposure to the Human Papiloma Virus, a sexually transmitted disease. Other life choices and behaviors, such as tobacco use, may also play a role in cancer formation (NCI 2006).

Melanoma

In the U.S., melanoma is the most common form of cancer (excluding non-melanoma skin cancer) in men 35-44 years of age and is the second most common form of cancer for women in this same age group (preceded by breast cancer). Melanoma primarily affects the white population. The single most common environmental cause in the development of melanoma is exposure to sunlight (ultraviolet radiation). Other environmental risk factors and occupations include vinyl chloride workers, rubber workers, chronic exposure to petrochemicals, textiles workers, electronic workers, chronic exposure to printing chemicals, and radiation (Austin and Reynolds 1986; Gallagher et al. 1986; Nelemans et al. 1992; Sinks et al. 1992; and Lundberg et al. 1992). Other risk factors include age, sex, race, and family history (Shottenfeld and Fraumeni 1996). Melanoma is more common in Utah as compared to the rest of the U.S. primarily due to the light-skinned population, high average elevation and desert related meteorology and outdoor lifestyle (UCR 1996).

Hodgkin's Disease

Hodgkin's disease accounts for less than two percent of all new cancers diagnosed in the U.S. It is more common among males than females and is more common among whites than blacks. Risk factors for Hodgkin's disease vary by age group. In children infection with Epstein-Barr Virus and socioeconomic status are common risk factors.

Socio-economic indicators such as education (e.g., persons with 14 or more years of education) and family size have been shown to be associated to increased risk for Hodgkin's disease. In older adults the risk for developing Hodgkin's disease increases with a prior history of living in multiple houses as children and exposures to infectious diseases. Other non-environmental risk factors include history of tonsillectomy, nodular sclerosis, past episodes of herpes zoster and of skin and genital warts, infectious mononucleosis (i.e., the Epstein-Barr virus), other viral infections and immunological alterations. Other environmental risk factors associated include exposure to wood or wood products and occupational exposure in the rubber or plastic industries (Diego et al. 1990, Shottenfeld and Fraumeni 1996).

Prostate Cancer

Prostate cancer is the most common cancer diagnosed in men and the second leading cause of cancer death in men. Age is the most significant risk factor for prostate cancer; however diet, family history and race have been associated as well. Environmental factors associated with prostate cancer include the use of tobacco, alcohol consumption and exposure to cadmium (a non-essential trace element) (Shottenfeld and Fraumeni 1996).

Brain Cancer

In the U.S., 17,000 new primary cancers of the nervous system are diagnosed each year. These are among the most fatal of all cancers and only about half of patients are still alive one year after diagnosis. Brain cancer is the 10th most common cause of cancer death (Shottenfeld and Fraumeni 1996). Brain tumors account for over 90% of all cancers in the central nervous system (UCR 2000). Environmental agents, such as ionizing radiation, have been clearly implicated as risk factors for brain tumors. Other factors possibly associated with childhood and adult brain cancer include n-nitrosoamine compounds, exposure to low frequency electromagnetic fields, pesticides, insecticides, radiation exposure, infections, alcohol consumption, lead, hair dye and spray, barbiturate use and other medications, chemotherapy (in utero), familial history, and race. Brain cancer may also be connected with breathing vinyl chloride over long periods (Shottenfeld and Fraumeni 1996).

Pancreatic Cancer

Pancreatic cancer is one of the most rapidly fatal forms of cancer and is rarely cured. It is the ninth most common cause of cancer and is the fifth most common cause of cancer mortality. It is more common in men than women and the rates in Utah have consistently been lower than the national rate (UCR 1996). Age is the best established risk factor. Environmental risk factors associated with pancreatic cancer include smoking, diet, alcohol abuse, asbestos, ionizing radiation, and pesticides (particularly DDT-Dichloro-Diphenyl-Trichloroethane) (Hecht and Hoffmann 1991 and Shottenfeld and Fraumeni 1996).

Cancer of the Uterus

Uterine cancer does not include cervical cancer. In both white and black females the majority of cancers of the uterus are endometrial cancers (the lining of the uterus). Menstrual and reproductive factors associated with endometrial cancer include age at menarche, parity, age at first birth, age at last birth, menstrual irregularities, infertility, duration of menses, menopausal symptoms, and age at menopause. Women with elevated endogenous estrogen levels have been reported to have an increased risk for endometrial cancer. Other possible risk factors include estrogen replacement therapy, oral contraceptives, endometrial hyperplasia, obesity, diet and alcohol consumption, gallbladder disease, diabetes, hypertension, age and family history (Shottenfeld and Fraumeni 1996). Uterine cancer incidence rates are complicated by the fact that many women in older age groups have had hysterectomies and are no longer at risk for this cancer. Utah women have a reported higher prevalence of hysterectomies (UCR 1996). Therefore, the rate of uterine cancer in women at risk in Utah may be under estimated as compared to national rates.

Ovarian Cancer

Ovarian cancer is usually fatal and will affect one to two percent of women in their lifetime. Ovarian cancer occurs more frequently in the post-menopausal age group. The most frequently cited risk factor for ovarian cancer is low fertility. A higher number of pregnancies appear to be protective. The incidence and mortality rates in Utah have been lower than the national rates (UCR 1996). Environmental risk factors associated in the etiology of ovarian cancer include ionizing radiation, and diet (i.e., high cholesterol) (Shottenfeld and Fraumeni 1996).

Soft Tissue Cancer

Soft tissue cancer is a general category that includes cancer occurring in muscle, heart tissue, subcutaneous tissue and other related tissues. Because this category includes a number of different types of cancer, it is difficult to define risk factors associated with cancers of the soft tissue. Soft tissue cancers do occur more frequently in children and young adults (Shottenfeld and Fraumeni 1996).

Cancer of the Oral Cavity

The oral cavity includes the tongue, gums, salivary glands, floor and other parts of the mouth and the pharynx. Not all of these cancers share common etiologies but are simply grouped together for convenience. The most common risk factor associated with the etiology of oral cancer appears to be the use of tobacco (i.e., cigarettes, smokeless tobacco, pipe smokers) and alcohol abuse (UCR 1996). Men are more likely to develop oral cancer than women. Other risk factors associated with the etiology of oral cancer include diet, precancerous lesions, poor oral hygiene, mouthwash, viruses (Human Papilloma Virus and Epstein-Barr Virus), asbestos, textile workers, indoor air pollution (wood stoves), and familial history (Shottenfeld and Fraumeni 1996). The incidence rate of cancer of the oral cavity is lower in Utah as compared to national rates (UCR 1996).

Non-Hodgkin's Lymphoma

The cause of most of the cases of non-Hodgkin's lymphoma (NHL) remains unknown. The incidence rate of NHL is higher among males than females. There is some evidence that a majority of cases have a strong genetic basis. Individuals at increased risk for NHL include those

with primary immunodeficiency diseases, acquired immunodeficiency diseases, and patients who are immunosuppressed after organ transplantation. Increased risk for NHL has been observed for patients with testicular cancer and Hodgkin's disease. Although the data are not entirely consistent, occupations dealing with chemicals and agriculture also appear to be associated with NHL in studies of incident cases. Other industries with reported increased risks of NHL are woodworkers, meat workers, and metalworkers (Schottenfeld and Fraumeni 1996).

Colorectal Cancer

The primary risk factors for colorectal cancer include genetics (familial history), colon polyps, inflammatory bowel disease (such as ulcerative colitis) and a diet high in fat and low in fiber. Colorectal cancer rates are consistently higher in males than in females for unknown reasons (Schottenfeld and Fraumeni 1996). Colon cancer is the third leading cause of cancer-death among both men and women. Currently more than three percent of the Utah population will be affected with colorectal cancer in their lifetime (UCR 1996).

APPENDIX C – STATISTICAL DEFINITIONS AND CALCULATIONS

Definitions

Age Adjustment

Different populations have different proportions of people within the various age groups. Cancer rates increase as people get older; therefore, it is not possible to compare two populations with different proportions of older persons. The cancer rates in the two populations will look different because the age structure of the populations are different, but there may not be a real difference when you compare specific age groups (i.e., persons under 18 or persons over 65). Age adjustment techniques control for this problem by comparing cancer rates between specific age groups rather than between whole populations.

Confidence Interval

A confidence interval is used to help determine significance. A confidence interval is described by the lower and upper confidence limits. Whenever a statistical test is performed, the result is only an estimate of the true risk value. A 95% confidence interval gives a range of values that probably includes the true risk value. In other words, there is a 95% chance that the true value of the result exists somewhere in that range. If the confidence interval of an SIR (see below) includes 1.0, then the result is not statistically significant, because there is a 95% probability that the difference found is due to chance alone. If a confidence interval does not include 1.0, then the result is statistically significant because there is a 95% probability that the difference found is not due to chance alone. This finding does not prove that the cancer rates are elevated or there is a causal factor. What this finding does prove is that the rate is statistically different from what was expected. It is then reasonable to suppose that the difference is a result of some causal factor.

Generally, as the sample size (or the number of people in your study) increases, the confidence interval becomes more narrow (i.e., the lower and upper limit values are closer).

Expected number of cases

The expected number of cases is the total number of cases that would be expected if the study population (e.g., Monticello) had the same cancer rates as a comparison population (e.g., the rest of Utah). This is calculated by multiplying the cancer rate for the comparison population (e.g., the Utah population) for a specific age group (e.g., 0-4 year olds) by the number of people in that age group in the study population (in this case, Monticello). The age group specific calculations are then totaled for a population expected case count.

Because the expected number of cases is based on mathematical calculations and not real-life scenarios, the expected case count is likely to be a non-integer number (i.e., contains a fraction component) and may be less than one. However, the observed case count will always be an integer number. This difference in the types of numbers used in the calculations makes it difficult to interpret an SIR since it is possible to be elevated

even if there was only one observed case of cancer during the time period being examined. Since it is conceivable that the case happened (by chance) to have occurred in the arbitrarily determined analytical period, using larger analytical periods or different analytical periods could result in a very different interpretation of the involvement of that case in the interpretation of rates. It is important to examine the confidence interval and evaluate whether the elevation meets the criteria for significance and stability. This information can assist with deciding whether the SIR is a reliable estimate of cancer risk. This is also why the EEP does not consider analytical periods with less than three observed cases occurring during that period.

Power

Power is the ability of a study to detect a difference if that difference really exists. The concept of power is closely tied to statistical significance and generally parallels significance. If the sample size (number of people in the study) is very small, then the power of the study is low; as a result, it might not be possible to see a difference even if there really is not one there. The best way to increase the power of a study is to increase the sample size. When the population is finite, as in this report, this is done by increasing the analytical period. Power considerations are the reason that five-year and seven-year analytical periods are used in this study.

Sample Size

Sample size refers to the number of people or number of observations in the study. If a community has a population of 2000 and there are 10 cases of cancer, there are 2000 observations, of which 10 were observed to have cancer and 1990 were observed to not have cancer. In cancer cluster investigations, the population of the area being examined determines the sample size. Sample size influences the ability to determine statistical significance and power. Therefore it is important to obtain a sufficient sample size for the statistical methods used. Since the population of the community is finite, this is done by increasing the analytical period.

Significance

By convention, a finding is described as statistically significant when it can be shown that the probability of obtaining such a finding by chance alone is relatively low (commonly 5%). Therefore, if a finding is significant, 95% of the time, that result represents a true difference. The EEP uses a 95% decision level (95% confidence interval) to determine significance. One should understand that this decision level implies a one in twenty chance (a 5% error rate) of mis-representing comparison of cancer rates.

Standardized Incidence Ratio (SIR)

An SIR is used to evaluate whether a study population has a higher number of cancers than we would expect if that population had the same cancer rate as the comparison population after adjustment of age distribution differences. An SIR is calculated by dividing the number of observed cancer cases by the expected number of cancer cases.

An SIR of one (1.0) indicates rates are equal and there is no increased risk. An SIR greater than one (1.0) indicates an increased risk for the study population. An SIR less

than one (1.0) indicates a decreased risk for the study group. SIR might not be 1.0 either because there is a true difference in the number of cases or due to random chance. The confidence interval (see above) determines whether the high or low SIR is due to chance or due to a real difference.

Method for Calculating Standardized Incidence Ratios

Standardized incidence ratios (SIRS) were calculated using the indirect method for age-adjusting rates. SIRs were calculated by comparing the observed number of cases as a ratio to the expected number of cases. The expected number of cases for each cancer were computed from the comparison population (the state of Utah) using age adjusting algorithms. The observed number of incidences is then compared (divided) with the expected number of incidences in the study population (Monticello) and a ratio is derived, referred to as the SIR.

The formula for this ratio = $\Sigma p_{ia} n_{ia} / \Sigma p_{is} n_{ia}$

Where: a = area chosen as the study area (Monticello)
s = area chosen as a reference standard (state of Utah)
 n_{ia} = number of individuals in ith class of study area
 n_{is} = number of individuals in ith class of reference standard area
 x_{ia} = number of cases in ith age class of area a (similarly for s)
 $p_{ia} = x_{ia} / n_{ia}$ = incidence rate in ith age class of area a (similarly for s)

(Kahn and Sempos 1989)

The confidence interval for the SIR is the range of values for a calculated SIR with a specified probability (95%) of including the true SIR value:

$$\frac{[\sqrt{n} \pm (1.96 \times 0.5)]^2}{x}$$

Where n is the Number of Observed.
 x is the Number of Expected.

(Frumkin & Kantrowitz 1987)

The confidence interval is used as a surrogate test of statistical significance (p-value). Both the p-value function and the spread of the function can be determined from the confidence interval. The difference between the observed versus the expected is considered statistically significant if the confidence interval for the SIR does not include one (1.0) and if the SIR is greater than one (1.0).

(Rothman and Greenland 1998)

APPENDIX D - (TABLES) CANCERS NOT ASSOCIATED WITH URANIUM MILLS

The following Tables are cancers that were evaluated (with three or more occurrences in any period/interval) that are not associated with exposures to uranium mills.

Colon Cancer

There were three analytical periods that demonstrated SIRs that were greater than 1.0; 1978-1982, 1993-1997, and 1998-2004 (Table 7). None of these analytical periods were statically significant. The cumulative analytical period 1973-2004 demonstrated rates (SIR=0.98) that were almost similar to the rates of Utah. No statistical significant test for increased pattern or trend was performed.

Table 7. Annual age-adjusted colon cancer incidence rates by each analytical study period comparing Monticello to Utah – 1973-2004.

| Time Period | Monticello Rate per 100,000 | Utah Rate per 100,000 | Monticello Observed number cases ³ | Monticello Expected number cases | SIR ¹ | 95% CI ² |
|--|-----------------------------------|-----------------------------|---|--|------------------|---------------------|
| 1973-1977 | - | - | - | - | - | - |
| 1978-1982 | 57.2 | 24.4 | ≥3 | 1.7 | 2.37 | 0.62 - 5.26 |
| 1983-1987 | - | - | - | - | - | - |
| 1988-1992 | - | - | - | - | - | - |
| 1993-1997 | 39.1 | 23.4 | ≥3 | 1.9 | 1.59 | 0.30 - 3.90 |
| 1998-2004 | 23.7 | 20.2 | ≥3 | 2.5 | 1.22 | 0.23 - 2.98 |
| 1973-2004 | 24.5 | 24.8 | ≥3 | 11.2 | 0.98 | 0.49 - 1.65 |
| ¹ Standardized Incidence Ratio | | | | | | |
| ² 95% Confidence interval | | | | | | |
| ³ Observed cases are presented as ≥3 when cases are greater than or equal to three or as – when cases are less than three in order to protect the confidentiality of the cases. | | | | | | |
| Data Source: Utah Cancer Registry, 1973-2004. | | | | | | |

Breast Cancer (Female)

The rates for female breast cancer exceeded the rates of Utah during analytical periods 1993-1997 and 1998-2004, but the SIRs were not statistically significant (Table 8). The study period rate (1973-2004) did not exceed the rate for Utah. Trend analysis did not demonstrate any statistically significant trends in either direction over the time period of the study.

Table 8. Annual age-adjusted female breast cancer incidence rates by each analytical study period comparing Monticello to Utah – 1973-2004.

| Time Period | Monticello Rate per 100,000 | Utah Rate per 100,000 | Monticello Observed number cases ³ | Monticello Expected number cases | SIR ¹ | 95% CI ² |
|--|-----------------------------|-----------------------|---|----------------------------------|------------------|---------------------|
| 1973-1977 | - | - | - | - | - | - |
| 1978-1982 | - | - | - | - | - | - |
| 1983-1987 | - | - | - | - | - | - |
| 1988-1992 | 78.0 | 89.8 | ≥3 | 3.4 | 0.88 | 0.17 – 2.15 |
| 1993-1997 | 98.2 | 95.7 | ≥3 | 3.9 | 1.02 | 0.27 – 2.27 |
| 1998-2004 | 118.0 | 111.1 | ≥3 | 6.7 | 1.04 | 0.41 - 1.95 |
| 1973-2004 | 74.2 | 96.4 | ≥3 | 23.5 | 0.77 | 0.45 - 1.16 |
| ¹ Standardized Incidence Ratio | | | | | | |
| ² 95% Confidence interval | | | | | | |
| ³ Observed cases are presented as ≥3 when cases are greater than or equal to three or as – when cases are less than three in order to protect the confidentiality of the cases. | | | | | | |
| Data Source: Utah Cancer Registry, 1973-2004. | | | | | | |

Prostate Cancer

The SIRs for prostate cancer were consistently lower than 1.0 in each analytical period from 1988 through 2004. Only the analytical period 1998-2004 (SIR=0.39; 95% CI 0.10-0.86) was statistically significantly decreased (Table 9). No statistical significant test for increased pattern or trend was performed.

Table 9. Annual age-adjusted prostate cancer incidence rates by each study period comparing Monticello to Utah – 1973-2004.

| Time Period | Monticello Rate per 100,000 | Utah Rate per 100,000 | Monticello Observed number cases ³ | Monticello Expected number cases | SIR ¹ | 95% CI ² |
|--|-----------------------------|-----------------------|---|----------------------------------|------------------|---------------------|
| 1973-1977 | 113.2 | 88.6 | ≥3 | 2.2 | 1.37 | 0.26 – 3.36 |
| 1978-1982 | - | - | - | - | - | - |
| 1983-1987 | 132.5 | 113.8 | ≥3 | 3.4 | 1.49 | 0.47 – 3.07 |
| 1988-1992 | 138.6 | 164.6 | ≥3 | 5.1 | 0.79 | 0.21 – 1.76 |
| 1993-1997 | 86.1 | 152.2 | ≥3 | 5.9 | 0.51 | 0.10 – 1.26 |
| 1998-2004 | 61.5 | 165.7 | ≥3** | 10.4 | 0.39 | 0.10 - 0.86 |
| 1973-2004 | 107.6 | 146.7 | ≥3 | 29.1 | 0.42 | 0.45 – 1.06 |
| ¹ Standardized Incidence Ratio | | | | | | |
| ² 95% Confidence interval | | | | | | |
| ³ Observed cases are presented as ≥3 when cases are greater than or equal to three or as – when cases are less than three in order to protect the confidentiality of the cases. | | | | | | |
| ** Statistically significant decrease (p = <0.05) from the expected number of cases. | | | | | | |
| Data Source: Utah Cancer Registry, 1973-2004. | | | | | | |

Melanoma

The rates and SIRs for melanoma during the analytical periods 1993-1997 and 1973-2004 were elevated but were not statistically significant. Period 1998-2004 demonstrated a SIR of less than 1.0, but was also not statistically significant (Table 10). The number of melanoma cases in each analytical period from 1973 through 1992 were too small to perform meaningful analysis. No statistical significant test for increased pattern or trend was performed.

Table 10. Annual age-adjusted melanoma incidence rates by each analytical study period comparing Monticello to Utah – 1973-2004.

| Time Period | Monticello Rate per 100,000 | Utah Rate per 100,000 | Monticello Observed number cases ³ | Monticello Expected number cases | SIR ¹ | 95% CI ² |
|--|-----------------------------------|-----------------------------|---|--|------------------|---------------------|
| 1973-1977 | - | - | - | - | - | - |
| 1978-1982 | - | - | - | - | - | - |
| 1983-1987 | - | - | - | - | - | - |
| 1988-1992 | - | - | - | - | - | - |
| 1993-1997 | 51.4 | 20.0 | ≥3 | 1.6 | 2.57 | 0.67 - 5.70 |
| 1998-2004 | 20.9 | 27.7 | ≥3 | 3.4 | 0.89 | 0.17 - 2.19 |
| 1973-2004 | 23.3 | 19.9 | ≥3 | 9.1 | 1.21 | 0.60 - 2.04 |
| ¹ Standardized Incidence Ratio | | | | | | |
| ² 95% Confidence interval | | | | | | |
| ³ Observed cases are presented as ≥3 when cases are greater than or equal to three or as – when cases are less than three in order to protect the confidentiality of the cases. | | | | | | |
| Data Source: Utah Cancer Registry, 1973-2004. | | | | | | |

Non-Hodgkin's Lymphoma

The number of cases in each analytical period from 1973 through 1992 were low and could not be meaningfully evaluated. Although the SIRs for periods 1993-1997, 1998-2004, and 1973-2004 were evaluated, they were not statistically significant (Table 11). No statistical significant test for increased pattern or trend was performed.

Table 11. Annual age-adjusted Non-Hodgkin's lymphoma incidence rates by each analytical study period comparing Monticello to Utah – 1973-2004.

| Time Period | Monticello Rate per 100,000 | Utah Rate per 100,000 | Monticello Observed number cases³ | Monticello Expected number cases | SIR¹ | 95% CI² |
|--|--|--------------------------------------|---|---|------------------------|---------------------------|
| 1973-1977 | - | - | - | - | - | - |
| 1978-1982 | - | - | - | - | - | - |
| 1983-1987 | - | - | - | - | - | - |
| 1988-1992 | - | - | - | - | - | - |
| 1993-1997 | 41.27 | 13.5 | ≥3 | 1.1 | 2.73 | 0.51 - 6.70 |
| 1998-2004 | 28.3 | 15.6 | ≥3 | 2.0 | 1.54 | 0.29 - 3.77 |
| 1973-2004 | 21.6 | 13.4 | ≥3 | 6.2 | 1.45 | 0.66 – 2.56 |
| ¹ Standardized Incidence Ratio | | | | | | |
| ² 95% Confidence interval | | | | | | |
| ³ Observed cases are presented as ≥3 when cases are greater than or equal to three or as – when cases are less than three in order to protect the confidentiality of the cases. | | | | | | |
| Data Source: Utah Cancer Registry, 1973-2004. | | | | | | |

APPENDIX E – CANCER CLASSIFICATIONS

International Classification of Diseases for Oncology – 3rd Edition

Cancer types with an asterisk (*) have been associated with the Monticello contaminants of concern.

Table 15. List of the International Classification of Diseases for Oncology (3rd edition) codes for cancers used in this study.

| Cancer Type | ICD-O-3 code † |
|---|--|
| Gastrointestinal Tract | |
| Oral Cavity & Pharynx | C00.0-C10.9 |
| <i>*Stomach</i> | C16.0-C16.9 |
| Colorectal | C18.0-C18.9, C26, C19.9, C20.9 |
| Liver & Intrahepatic Bile Duct | C22.0-C22.1 |
| <i>*Gallbladder & Biliary Ducts</i> | C23.9-C24.9 |
| Pancreas | C25.0-C25.9 |
| Urinary Tract | |
| <i>*Bladder</i> | C67.0-C67.9 |
| <i>*Kidney & Renal Pelvis</i> | C64.9, C65.9 |
| Other Urinary | C66.9, C68.0-C68.9 |
| Skin, Bone, Soft Tissue | |
| Bones & Joints | C40.0-C41.9 |
| Soft Tissues (including heart) | C38.0, C47.0- C47.9, C49.0-C49.9 |
| Cutaneous Melanoma | C44.0-C44.9, M8720-M8790 |
| Respiratory Tract | |
| <i>*Lung & Bronchial</i> | C34.0-C34.9 |
| Blood and Lymph | |
| Hodgkin's Lymphoma | (All Sites) M9650-M9667 |
| Non-Hodgkin's Lymphoma | M9590-9596, M9670-9719, M9727-9729, M9823, M9827 (All Sites except C024, C098-C099, C111, C142, C379, C420-C422, C424, C770-C779) |
| <i>*Multiple Myeloma</i> | M9731-9732, M9734 |
| <i>* Lymphocytic Leukemia (chronic and acute)</i> | (All Sites) M9826, M9835-M9837 / (Sites C420, C421, C424) M9823 |
| † Lymphomas were excluded from all solid-tissue cancer sites and were analyzed as a separate category | |

Table 15 continued.

List of the International Classification of Diseases for Oncology (3rd edition) codes for cancers used in this study.

| Cancer Type | ICD-O-3 code † |
|---|--|
| | |
| Head and Neck | |
| Brain | C71.0-C71.9 |
| *Thyroid | C73.9 |
| | |
| Other Endocrine | C37.9, C74.0-C74.9, C75.0-C75.9 |
| | |
| Female-specific cancers | |
| Breast | C50.0-C50.9 |
| Uterus | C54.0-C54.9, C55.9 |
| Ovary | C56.9 |
| | |
| Male-specific cancers | |
| Prostate | C61.9 |
| | |
| Other site-not specified | M9740-M9741, M9750-M9758, M9760-M9769, M9950-9989, (Sites C76.0-C76.8) M8000-M9589, C80.9 (M8000:9589), C42.0-C42.4 (M8000:9589), C77.0-C77.9 (M8000:9589) |
| † Lymphomas were excluded from all solid-tissue cancer sites and were analyzed as a separate category | |