HEALTH CONSULTATION

AN INVESTIGATION OF CANCER INCIDENCE IN MONTICELLO, UTAH

MONTICELLO, SAN JUAN, UTAH

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SUMMARY

Monticello, Utah, located in San Juan County in southeastern Utah is a city with a population of 1,958 (Census, 2000). From 1943 through 1960 a uranium and vanadium processing mill operated immediately south of the town. The mill site and surrounding properties were considered public health hazards and were both National Priorities List (NPL) sites. Remediation of contaminated soils from the mill site and affected areas off of mill property was completed in 2000. Although the contamination at the former mill site and vicinity properties posed a public health hazard prior to remediation, since the sites have been remediated the site is no longer a public health hazard.

Current citizens of the city feel that they are experiencing substantially elevated cancer rates due to exposures associated with the mill. In response to these concerns, the Utah Department of Health Environmental Epidemiology Program was asked to evaluate the incidence of cancer in Monticello.

Data from the Utah Cancer Registry were used for this investigation. The data received from the Registry covered the years from 1973 – 2003. All cancer types with one or more cases during the study period were analyzed, however this report particularly focused on cancer types that have been associated with the contaminants of concern in Monticello. These cancers include: bladder, gallbladder, kidney, leukemia, liver, lung, multiple myeloma, stomach, and thyroid. A cancer case was included 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. The population of interest was defined as the zip code 84535, which includes the city of Monticello and the surrounding area. Only cancer diagnoses received while an individual was a resident of the 84535 zip code were included in this analysis. The 84535 zip code will be referred to as Monticello. The state of Utah was the comparison population. Population estimates for the state of Utah were obtained from the Utah Governor's Office of Planning and Budget. Population estimates for the 84535 zip code from 1990-2003 were obtained from the U.S. Census; earlier populations were estimated based on these data.

Standardized Incidence Ratios (SIR) for each cancer type were calculated for five five-year periods and one six-year period starting with 1973 to evaluate whether the Monticello population had a greater risk or a lower risk of developing cancer as compared to the comparison 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 extremely small numbers of cancers in persons less than 18 years old, it was not possible to analyze those cases separately.

When cancers of all types were analyzed together, cancer actually occurred at rates lower than expected based on comparison with the remainder of Utah. In examining site-specific cancer rates, we similarly found little evidence of increased risk. A statistically significant increase in site-specific cancer was found only for lung cancer during only one time period (1993-1997). For other cancer types, elevated cancer rates were observed during some time periods but these

were based on small numbers of cancers and analysis indicated these elevations were not statistically significant, that is, they could have easily occurred by chance alone.

This study had several limitations. The small size of the Monticello population limited the ability of this study to detect statistically significant elevations in cancer rates, which is also referred to as low statistical power. It is possible that some cancer cases were not included in this analysis since residents may have gone to neighboring states for medical care and received a diagnosis there. This study was not able to analyze cancer rates prior to 1973. Additionally, this study did not evaluate individual factors such as smoking and family history, which also have an impact on cancer rates.

This study did not find elevated risk for cancer in residents of Monticello and the surrounding area. 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, limiting the ability to correctly count all cancers that might have resulted from exposure in Monticello. Therefore, this study was not able to conclusively determine whether or not cancer rates in the Monticello area are or were truly elevated.

RECOMMENDATIONS

The Environmental Epidemiology Program (EEP) recommends that further analysis of cancer rates in Monticello be performed. Specifically, an attempt should be made to validate cancer cases that were identified through surveys done by the Monticello Victims of Mill Tailings Exposure committee. These surveys may have identified cancer cases that might be missing from the Utah Cancer Registry due to being diagnosed in another state.

The EEP recommends that the feasibility of further epidemiologic studies, be explored. Such studies would need to collect information on individual behaviors such as smoking, migration and exposure to non-mill associated environmental contaminants. Additionally, the specific focus of such studies should be carefully considered. Statistical power and sample size will remain an issue that may not be surmountable by using different study designs. More in depth epidemiological studies are very time and resource intensive and may not provide clear answers to the concerns of the community. The availability of resources to perform such studies should also be considered.

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

PUBLIC HEALTH ACTION PLAN

The EEP will provide the Monticello community with a copy of this health consultation and will meet with the community and other interested parties to discuss the results of this health consultation.

The EEP is working on validating the cancer cases identified through surveys performed by the Monticello Victims of Mill Tailings Exposure committee. This is a lengthy and involved process that may take considerable time to complete. Upon completion of this process, the EEP will repeat the analyses presented in this report including the additional, validated cases where the inclusion is appropriate based on the study methods.

The EEP will collaborate with ATSDR to determine appropriate methods for further evaluating hazardous exposures which have occurred in the Monticello community.

The EEP will explore the feasibility of performing a more in-depth epidemiologic study and whether such a study could overcome some of the limitations presented in this report.

INTRODUCTION

Monticello, Utah is a city with a population of 1,958 located in San Juan County in southeastern Utah (Census, 2000). 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**.**

City residents have expressed concern that they are experiencing substantially elevated cancer rates due to exposures associated with mill operations and remediation activities. In response to these concerns, the Environmental Epidemiology Program of the Utah Department of Health was requested to evaluate the incidence of cancer in Monticello.

BACKGROUND

SITE/FACILITY DESCRIPTION AND BACKGROUND

Monticello is located geographically in the southeast part of the state of Utah in San Juan County. Its elevation is 7069 feet, and its population, according to the 2000 census, is 1,958 (Census, 2006).

There are two National Priorities List (NPL) sites in Monticello the Monticello Mill Tailings Site (MMTS) and the Monticello Vicinity Properties (MVP). The MMTS and MVP were placed on the NPL in 1989 and 1986 respectively. Both sites are associated with the Monticello Uranium Mill.

Site Description

The Monticello Mill Tailings Site is a 110-acre abandoned uranium and vanadium processing mill in the city of Monticello. The Monticello Vicinity Properties (MVP) are off-site residential and commercial properties located within or near the city of Monticello. The United States Department of Energy (DOE) owned the site until 2000; at that time, remediation work on the site was completed and the city of Monticello was given the land through the National Park Service. The city of Monticello, private residents and the state of Utah own various surrounding properties. No residences are located on the mill site; however, residences are adjacent to the north and east edges of the mill site. A map of the area can be found in Appendix A.

Operating History

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

Construction of the Monticello plant, in addition to the mill proper, included the development of an adequate water supply, installation of a power plant, and construction of two large housing projects for workers. The staff town site, on the hill opposite the mill to the south, consisted of a staff house for 12 men, a manager's house, and 14 four-room family dwellings. The other

housing project consisted of 32 two-room family houses and a bunkhouse and boardinghouse for 32 men (ATSDR, 1997).

Intermediate owners and operators of the Monticello Mill Tailings Site included the War Assets Office; the Atomic Energy Commission (AEC); American Smelting and Refining Company; Galigher Company; Lucius Pitkin, Inc.; National Lead Company; the Bureau of Land Management (BLM); and the DOE (ATSDR, 1997). 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).

Milling processes used at Monticello during the 11 years of AEC operation included raw ore carbonate leach, low-temperature roast/hot carbonate leach and salt roast/hot carbonate leach until 1955, acid leach resin-in-pulp and raw ore carbonate leach from 1955 to 1958, and a carbonate pressure leach resin-in-pulp process from 1958 until mill closure in 1960 (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 Monticello Mill Tailings Site (MMTS) and the Monticello Vicinity Properties (MVP) were established. The DOE has primary responsibility for remediation activities at both sites. The MMTS and the MVP were added to the National Priority List in 1989 and 1986 respectively.

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, 2005; DOE, 2004). Detailed descriptions of the remediation activities are available on the DOE Monticello website (http://www.lm.doe.gov/land/sites/ut/monticello/monticello.htm). Briefly, tailing piles on the mill site were moved to a permanent storage site adjacent to the mill property. Other contaminated soils and sediments were also moved to the permanent storage repository. A total of 30 peripheral properties, non-mill owned properties that were adjacent to the site, were remediated. Remediation of OU III was delayed until remediation of OU I and OU II was completed. The plan for OU III is detailed in the DOE 2004 Final Record of Decision for OU III and construction for the remedy was completed in 2004 (EPA, 1998; DOE, 2005).

The Monticello Vicinity Properties are off-site residential and commercial properties. Land use of most of these properties is residential housing. Throughout the operating period of the mill, tailings from the mill site were windblown into the city of Monticello and were intentionally used in various construction activities in the city. There was an estimated 156,000 cubic yards of contaminated materials present in the city of Monticello (ATSDR, 1997).

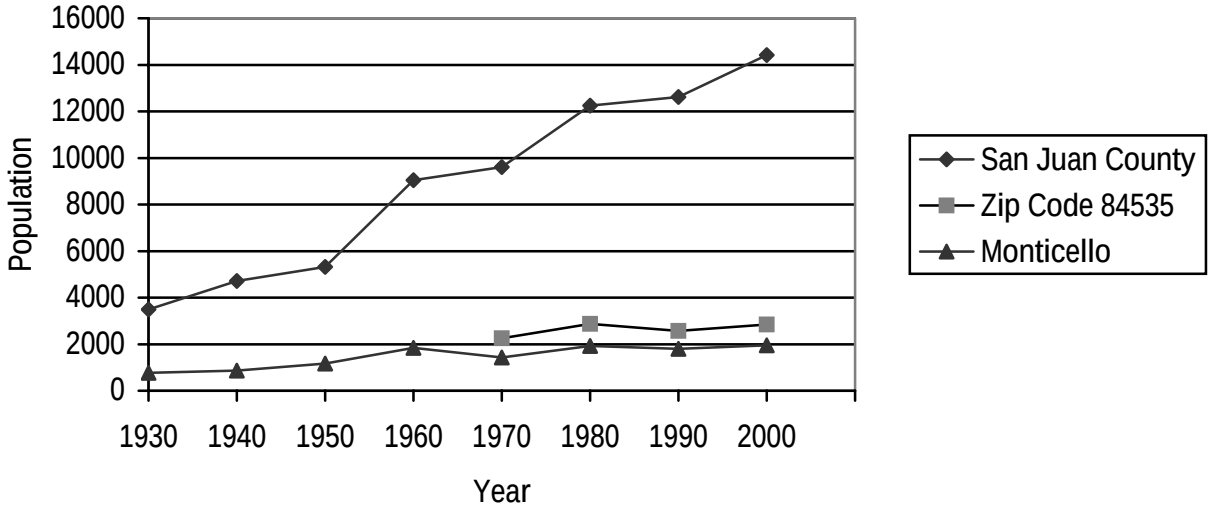
Remediation of the MVP began in 1984 and was completed in 1998 (DOE, 2005). A total of 424 properties were remediated to remove tailings from soil, concrete, brick mortar, plaster and other contaminated construction materials. All contaminated materials were placed in a permanent repository south of the mill site.

Demographics

Monticello is the geographic center of San Juan County, Utah. The population, according to the 2000 census, is 1,958 (Census, 2006). Figure 1 shows the population of the city of Monticello and of San Juan County by census year. In contrast to the predominantly Native American population of San Juan County as a whole, 83% of Monticello residents 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.

Nearly 79% of Monticello's households are owner-occupied. The median household income was almost $36,000 compared to $28,000 for the county as a whole.

Figure 1. Population of San Juan County, the zip code 84535 and the city of Monticello by Census Year



CONTAMINANTS OF CONCERN

Waste Types

The Monticello Uranium Mill produced several types of waste. The following is a list of types and content of wastes produced as they appeared during processing activities (ATSDR, 1997).

1. Ore

Residual ore material was left along roadways and at the ore-buying station. This material came from trucking the ore to the station, segregating it and moving it to the mill. The

generic term ‘uranium ore’ refers to any type of rock deposit that contains uranium at levels that can be mined economically, typically more than 0.05% (ATSDR, 1999a). Uranium is found in many different rock types and it is difficult to determine what contaminants of concern, aside from uranium and radium, may have been in the ore that was processed at Monticello.

2. Dust

Dust was produced from crushing and grinding the ore into sand (ATSDR, 1997). Coarse dust settled out near the machines and was a hazard to operators. Uranium concentrates in the fine dust particles, which can be carried further by the wind. The size of these dust particles (1 to 10 microns) meant that they could enter and lodge inside the lungs, where there is the potential to cause cancer due to radiation. Coughing up particles and subsequent swallowing can lead to exposure to the stomach and intestines.

3. Fumes

Fumes were released from the roaster stack at the end of the production cycle. The fumes contained chlorine and hydrogen chloride, and uranium oxide and vanadium pentoxide. The oxides were in particle form and were likely small enough to penetrate deeply into lungs (ATSDR, 1997).

4. Gases

Radon gas was the primary gaseous waste from mill activities. Radon is formed by the natural radioactive decay of uranium. Humans are primarily exposed through inhalation. Radon was released from essentially all parts of the milling operation, from tailings piles and from areas where tailings were used in construction (ATSDR, 1997).

5. Liquids

Liquid tailings were the leftover processing liquids and contained various chemical and radiological contaminants such as: chlorides, sulfates, carbonates and bicarbonates of sodium and other metals including uranium, vanadium and arsenic (ATSDR, 1997). Liquids accumulated in a tailings pond and eventually flowed into Montezuma Creek (ATSDR, 1997).

6. Solids

Solid wastes (tailings) contained the original ore and the chemicals added during extraction, similar to the liquid waste, less most of the uranium and vanadium. The tailings were placed in four separate piles on the mill site. As the solid material dried, wind blew it off the mill site, contaminating some of the nearby land. In 1962, the AEC covered the piles with earth and seeded them to reduce the public health risk. However, due to erosion from rain and creek water, tailings continued to be washed downstream (ATSDR, 1997).

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 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. 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) and Monticello Radioactively Contaminated Properties (aka Monticello Vicinity Properties) (ATSDR, 1997).

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).

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 above.

Uranium milling exposure

The following cancers have been associated with uranium milling and radon exposure: gallbladder, kidney, leukemia, liver, lung, multiple myeloma, stomach, and thyroid (Schottenfeld & Fraumeni, 1996, Tomasek et al, 1993, Nermina 2005, Papathanasiou 2005, and Shpagina 2005). Please see Appendix D – Cancer Epidemiology, for a discussion of other common risk factors for these cancers.

Specific contaminants

Radioactive contaminants

1) Uranium

   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 (ATSDR, 1999a). Inhalational exposure to uranium ore has been associated with lung cancer; 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.

2) Radium

   Oral ingestion of radium has been associated with bone sarcomas and head cancers (ATSDR, 1990a). Possible associations with breast, liver and kidney cancers have also been found. 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. However, no data were available regarding historical levels of radium in the soil at the time of this report.

3) Radon

   Radon is a product of natural uranium and radium radioactive decay. It is a gas and when located outside, it disperses quickly; however, in enclosed spaces, radon gas can accumulate and may be a health hazard. Radon exposure indoors has been associated with lung cancer (ATSDR, 1990b).

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, and liver cancer. 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.

   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.

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

2) Beryllium

   No association between oral ingestion of beryllium and cancer has been found (ATSDR, 2002). Inhaled beryllium has been associated with lung cancer in humans and animals. The current soil concentrations of beryllium are well below the levels that have been associated with cancer. 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.

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.

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.

5) Molybdenum
Molybdenum has not been found to be associated with cancer (EPA, 2006).

The carcinogenicity of molybdenum has not been classified by either the EPA or IARC.

6) Nickel
Respiratory tract cancers have been associated with inhalation of high levels of nickel; however, these studies were done in occupational settings where persons were exposed to many other known carcinogens in addition to nickel (ATSDR, 2005c). No adverse effects are 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.

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.

7) Selenium
Selenium has not been found to be associated with cancer (ATSDR, 2003). 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.

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.

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

Vanadium is not assigned a carcinogenicity classification by the EPA. It is considered a possible human carcinogen by the IARC.

END-STAGE RENAL DISEASE

End-Stage Renal Disease (ESRD) is usually due to prolonged damage to the kidney resulting in progressively less function. When persons are no longer able to completely clear toxins from their blood with the kidneys, they are considered to have ESRD. By far, the most common causes of ESRD in the United States are diabetes and hypertension. Diabetes alone accounts for approximately 40% of all ESRD cases in the United States (USRDS, 2005).

End-stage renal disease (ESRD) has been associated with exposure to uranium (ATSDR, 1999a) It appears to be related to the chemical properties of uranium rather than its radioactive properties. An ATSDR Health Consultation evaluated deaths due to renal failure and the diagnosis of ESRD in San Juan County (ATSDR, 1999b). Elevated deaths in women due to renal failure were found for San Juan County, however due to the small sample sizes, deaths in Monticello residents were not evaluated separately from San Juan County. ESRD was elevated in minority residents of San Juan County compared to the state of Utah. No elevation was found in white residents. No analysis was performed of Monticello residents specifically.

EXPOSURE PATHWAYS

In order to determine whether humans have been exposed to contaminants from the mill site, the existence of exposure pathways must be evaluated. A pathway consists of five components:

1) A source of contamination, such as mill tailings
2) An environmental medium in which contaminants might enter, such as ground water
3) Point(s) of human exposure, such as drinking water
4) Route(s) of exposure, such as ingestion
5) A receptor population, such as Monticello residents.

An exposure pathway is considered completed if all five components existed in the past, currently exist or will exist in the future. A pathway is considered potential if one or more of the five elements are missing. In the situation of potential pathways, sufficient information may not exist to establish that all the components exist, did exist or will exist, or there is the possibility that the missing component(s) may develop in the future. An exposure pathway can be eliminated if at least one of the five elements is missing and will never again be present.

Remediation work in the MMTS and MVP has made substantial progress and many exposure pathways in the Monticello area have been eliminated. However, the exposure pathways identified by ATSDR were complete pathways at some point in the past (ATSDR, 1997). Additionally, the ATSDR Public Health Assessment (PHA) identified several potential exposure pathways.

COMPLETED 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 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, 1997

On-Site Surface Soils

This exposure pathway refers 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). The permanent storage site has a soil cap of appropriate depth and additional vegetation that will stabilize the soil cap and prevent erosion. Sediment ponds are in place at the permanent site to prevent contaminated solid materials from being washed off site by rainwater.

Control and ownership of the original mill site and selected surrounding properties have been transferred to the city of Monticello (DOE, 2003). According to the terms of the transfer of ownership, the property can be used for recreational activities, but no structures can be built on the property and it may not be used for overnight camping. Restoration of the site to recreate wetlands and manage erosion was performed by the city of Monticello using DOE guidelines. Ongoing maintenance of the site is the responsibility of the city of Monticello. Since the majority of contaminated materials were removed from the site during remediation and there was new ground cover installed on the site, current exposures to onsite surface soils are no longer a concern.

While the past exposures from the former mill site have been addressed by remediation, historically, individuals who worked, lived or played at the mill site were also exposed to this completed pathway. Available data on the level and types of exposures are limited (ATSDR, 1997).

Off-Site Surface Soils

Tailings from the mill were used as fill for open lands, backfill around water, sewer and electrical lines, sub-base for driveways, sidewalks and concrete slabs and in backfill, plaster and

mortar for construction in the city of Monticello. Approximately 135,000 tons of tailings are believed to have been removed from the mill site for construction purposes (ATSDR, 1997).

Four hundred twenty-four properties in the town of Monticello that were identified as being contaminated from tailings or other sources of contamination were remediated by the DOE. Remediation was completed in 2001 (DOE, 2003; DOE, 2005).

Additionally, contaminated soils were windblown from the site to adjacent areas, which are currently used for cattle pasture and crop production (for cattle, not human consumption) (DOE, 2003). These contaminants could lead to exposures via several routes: a) entering the food chain when they are ingested with food products or animal products, b) inhalation of contaminated dust or soil, and c) dermal absorption from contact with contaminated soils. These contaminated peripheral properties were remediated as part of Operable Unit II in 2001 and are no longer a public health hazard.

Off-Site Air

Radium-226 was a by-product of uranium and vanadium processing at the mill and is found in mill tailings. Radium-226 undergoes radioactive decay to radon-222 gas. Radon gas emerges from soil or other materials into the atmosphere and can then be inhaled. When radon is released from outdoor soils and contaminated materials, the gas disperses quickly and poses little health risk to humans. When radon is released in confined spaces, such as homes or basements, the gas accumulates and has been associated with elevated lung cancer risk.

Indoor air concentrations of radon from mill tailings were addressed by remediation of Monticello Vicinity Properties by DOE; therefore, this exposure pathway is extinct. Radon from naturally occurring radioactive material may also be an issue in the area; however, property owners are responsible for removal of naturally occurring materials.

POTENTIAL EXPOSURE PATHWAYS

Groundwater and Surface water

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 watering and institutional controls are in place to prevent it from being used for these purposes in the future (DOE, 2004). The shallow alluvial aquifer and Montezuma Creek are in the process of remediation as part of OU III. These activities address 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

As discussed above, there is 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 in the Monticello deer and cattle were similar to levels in the reference animals (Everett, 1998).

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

METHODS

CANCER DATA

Data for this investigation were obtained from the Utah Cancer Registry, 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 2003.The year 2003 was the most recent year for which complete data were available and 1973 was the earliest year census tract information was available. Cases from the registry were examined in five-year intervals except for the last time period, which had six years (1973-1977, 1978-1982, 1983-1987, 1988-1992, 1993-1997, and 1998-2003). A single five or six-year interval will be referred to as a period. Separate analyses were also performed on combined data for the full study period (1973-2003).

There have been several contaminants of concern in Monticello and those contaminants have been associated with various cancer types. All cancer types with one or more cases during the study period were analyzed, however the analysis particularly focused on cancer that have been associated with exposure to the contaminants of concern in the Monticello area.

All cancers that were evaluated for elevated rates during the study period are listed below. Cancer types with an asterix (*) have been associated with the contaminants of concern found in Monticello. See Appendix E for a list of the International Classification of Diseases for Oncology (3rd edition) codes that were used to select the cancers included in this study.

Gastrointestinal Tract
- Oral Cavity & Pharynx
- *****Stomach
- Colorectal
- Liver & Intrahepatic Bile Duct
- *****Gallbladder & Biliary Ducts
- Pancreas

Urinary Tract
- *Bladder
- *****Kidney & Renal Pelvis

Blood and Lymph
- Hodgkin's Lymphoma
- Non-Hodgkin's Lymphoma
- *****Multiple Myeloma
- *****Acute Lymphocytic Leukemia
- *****Chronic Lymphocytic Leukemia

Head and Neck
- Brain
- *****Thyroid
- Other Endocrine

Other Urinary

Skin, Bone, Soft Tissue
- Bones & Joints
- Soft Tissues (including heart)
- Cutaneous Melanoma

Respiratory Tract
- *****Lung & Bronchus

Female-specific cancers
- Breast
- Uterus
- Ovary

Male-specific cancers
- Prostate

Other site-not specified

The following cancer types had no cases reported in Monticello during the time period of the study: esophagus, small intestine, anus, other digestive system cancers (excluding the sites already mentioned), larynx, other respiratory tract cancers (excluding larynx and lung/bronchus), cervix, other female genital cancers (excluding the sites already mentioned), testes, other male genital cancers (excluding prostate and testes), eye/orbit, non-brain central nervous system cancers, myeloid leukemia, and monocytic leukemia. The category "other site-not specified" was included in the analysis of all cancers combined, but was not analyzed separately since it does not represent a single cancer type.

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.

POPULATION OF INTEREST

The population chosen for analysis included residents of the city of Monticello and surrounding area. Residence in the zip code 84535 was used as proxy for residence in the Monticello area since many addresses in the Utah Cancer Registry were PO Boxes in this zip code and did not have a specific physical address. Therefore, it was not possible to limit the study to residents who lived within the city limits of Monticello. The 84535 zip code includes the city of Monticello and the surrounding area; no other population centers are located within the 84535 zip code.

Information on where persons may have lived prior to a diagnosis was not available from the Utah Cancer Registry; therefore it was not possible to include former residents of Monticello in this analysis. In addition, persons continue to be exposed to environmental contaminants of other types after moving away from Monticello. The type of analysis performed in this study was unable to account for this. Therefore, persons who may have been residents of Monticello prior to their diagnosis of cancer were excluded from the analysis. A cancer case was only included if the person was a resident of the 84535 zip code at the time of diagnosis. In five

cases, the street or city address listed in the cancer registry was not within the 84535 zip code, despite the fact that this was the listed zip code for the cases; these five cases were excluded since it was not possible to confirm the actual zip code of residence of these individuals.

Population denominator data for the state of Utah and San Juan County was obtained from the Utah's Governor's Office of Planning and Budget. Monticello zipcode data were obtained from the U.S. Census and were only available from 1990 through 2003. Earlier population estimates for the zip code 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 for 1973 through 1989 were estimated based the estimated total population, using the age distribution of the San Juan County population.

The state of Utah was selected as the comparison population for this investigation. A cancer case was included in the comparison population if the person was a resident of the state of Utah at the time of diagnosis. For the purpose of analysis, zip code 84535 will be referred to as *Monticello* and the state of Utah will be referred to as *Utah*, unless otherwise specified.

STATISTICAL ANALYSIS

Observed and expected numbers of cancer cases were compared using Standardized Incidence Ratios (SIR) for each period (Kelsey, et al 1986; 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 were used for the direct standardization. A single SIR was calculated for each cancer in a 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). Confidence intervals were not calculated for periods in which there were zero observed cases.

Chi-square tests for linear trend were performed for cancer from all sites and for cancer types when there were cases in more than two time periods. Fisher's exact test was used for all trend analyses to account for the small number of cases.

Interpreting SIRs and Confidence Intervals

An SIR is used to evaluate whether one population has a higher number of cancers than we would expect if that population had the same age-specific cancer rates as the state 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. 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 determines 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 result. A 95%

confidence interval gives a range of values for the result; there is a 95% chance that the true value of the result exists somewhere in that range. If the confidence interval of an SIR includes 1.0, then the result is not statistically significant, because there is a greater than 5% chance that the difference found is due to chance alone. If a confidence interval does not include 1.0, then the result is statistically significant; however, statistical significance alone does not prove that cancer risk is truly higher or lower than expected. Confidence intervals are generally wide when the sample size (or the number of people in the study) is low. A wide confidence interval indicates that the SIR is not very reliable or precise. See Appendix B for further discussion of the statistical methods used in this study.

RESULTS

Results are presented below for all cancer types that have been associated with uranium milling and its products. These include lung cancer, gallbladder and biliary tract cancer, stomach cancer, liver cancer, bladder cancer, kidney cancer, leukemias, multiple myeloma, and thyroid cancer. Data are also presented on all cancer sites combined and on other urinary cancers.

CANCER FROM ALL SITES

Cancer from all sites in Monticello residents was examined by 5- or 6-year time periods. Two time periods had statistically significantly decreased SIRs, 1988-1992 and 1998-2003 (See Table 2). The cumulative SIR for all time periods was 0.74 and was statistically significantly decreased (95% CI = 0.63, 0.87). Trend analysis did not demonstrate any statistically significant trends in either direction over the time period of the study.

Table 2. Annual age-adjusted incidence rates for all cancers by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

Time Period	Monticello Rate per 100,000	Utah Rate per 100,000	Monticello Observed number cases	Monticello Expected number cases	SIR[1]	95% CI[2]
1973-1977	276.8	310.9	15	19.6	0.76	0.43 , 1.20
1978-1982	293.5	316.1	20	22.3	0.90	0.55 , 1.33
1983-1987	313.0	321.5	24	24.6	0.97	0.62 , 1.40
1988-1992	207.1	353.8	19	34.2	0.56*	0.33 , 0.83
1993-1997	271.4	346.0	32	42.4	0.76	0.52 , 1.04
1998-2003	191.3	349.2	31	57.6	0.54*	0.37 , 0.74
1973-2003	264.8	336.3	141	189.6	0.74*	0.63 , 0.87
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
* Statistically significant decrease (p<0.05) from the expected number of cases						
Data Source: Utah Cancer Registry, 1973-2003.						

LUNG AND BRONCHIAL CANCER

Lung and bronchial cancer in Monticello residents was statistically significantly elevated compared to Utah's overall rate during only one period, 1993-1997 (Table 3). The SIR for this period was 2.39 (95% CI = 1.02, 4.34). SIR's were below or near to one (i.e., decreased risk or same risk) in four other periods and elevated by an amount that was not statistically significant in one other period. It is important to note that statistically elevated rates were not found in any

other time period and no pattern of increasing rates over time was observed. This finding is most compatible with random variation over time. The cumulative SIR for all time periods was elevated (SIR=1.42), but that increase was not statistically significant.

Table 3. Annual age-adjusted incidence rates for lung and bronchial cancer by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	59.9	25.5	4	1.6	2.54	0.66 , 5.64
1978-1982	33.0	26.9	≤3	1.9	1.07	0.10 , 3.05
1983-1987	34.0	27.8	≤3	2.1	0.96	0.09 , 2.76
1988-1992	11.9	26.5	≤3	2.6	0.39	0.00 , 1.53
1993-1997	75.1	27.2	8	3.3	2.39*	1.02 , 4.34
1998-2003	25.0	25.2	4	4.2	0.95	0.25 ,2.10
1973-2003	44.0	26.5	21	14.8	1.42	0.88 , 2.10

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are less than or equal to 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, 1973-2003.

GALLBLADDER

The SIRs for gallbladder cancer in Monticello residents were not statistically significantly elevated in any time period (See Table 4). The Monticello rate was elevated above the Utah rate in two time periods, but these elevated rates were based on very few cases and the elevations were not statistically significant. There was no statistically significant trend in gallbladder cancer rates over time. The number of cases of gallbladder cancer was very small, with four periods reporting no cases.

Table 4. Annual age-adjusted incidence rates for gallbladder cancer by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	29.1	2.8	≤3	0.1	6.81	0.00 , 26.69
1978-1982	0.0	2.4	0	0.1	0.00	-
1983-1987	0.0	2.2	0	0.1	0.00	-
1988-1992	0.0	1.8	0	0.2	0.00	-
1993-1997	15.7	2.0	≤3	0.2	8.41	0.79 , 24.12
1998-2003	0.0	1.9	0	0.3	0.00	-
1973-2003	5.9	2.1	≤3	1.1	2.82	0.53 , 6.91

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are less than or equal to three in order to protect the confidentiality of the cases

Data Source: Utah Cancer Registry, 1973-2003.

STOMACH CANCER

No statistically significant increases or decreases were found for stomach cancer (Table 5). Stomach cancer cases only occurred in one time period, 1998-2003. This was the only time period in which cases of stomach cancer occurred. The cumulative SIR for all time periods was elevated, but was not statistically significant. No trend analysis was performed since cases only occurred in one time period.

Table 5. Annual age-adjusted incidence rates for stomach cancer by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	9.1	0	0.5	0.00	-
1978-1982	0.0	7.5	0	0.5	0.00	-
1983-1987	0.0	6.4	0	0.5	0.00	-
1988-1992	0.0	5.9	0	0.6	0.00	-
1993-1997	0.0	4.5	0	0.5	0.00	-
1998-2003	20.4	4.8	≤3	0.8	3.80	0.72 , 9.31
1973-2003	7.3	5.9	≤3	3.1	0.97	0.18 , 2.38

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are less than or equal to three in order to protect the confidentiality of the cases.

Data Source: Utah Cancer Registry, 1973-2003.

BLADDER CANCER

No single time period had statistically significantly elevated or decreased SIRs for bladder cancer (Table 6). Rates were elevated in Monticello compared to Utah in two time periods; lower

rates in Monticello were found in four time periods. Neither the elevations nor decreased rates were statistically significant. The number of cases was very low; one period had no cases and no periods had more than three cases. The cumulative SIR was not statistically significantly elevated. Bladder cancer rates did not have a statistically significant upward or downward trend over the time period of the study.

Table 6. Annual age-adjusted incidence rates for bladder cancer by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	50.2	14.4	≤3	0.8	2.46	0.23 , 7.05
1978-1982	11.4	14.3	≤3	0.9	1.08	0.00 , 4.23
1983-1987	11.9	12.5	≤3	0.9	1.10	0.00 , 4.33
1988-1992	8.1	12.8	≤3	1.2	0.84	0.00 , 3.29
1993-1997	0.0	13.5	0	1.7	0.00	-
1998-2003	14.8	13.3	≤3	2.2	0.89	0.08 , 2.55
1973-2003	13.3	13.4	7.0	7.1	0.98	0.39 , 1.84
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

KIDNEY CANCER

No single time period had statistically significantly elevated or decreased SIRs for kidney cancer (Table 7). Monticello rates were elevated in two time periods when compared to Utah rates, but these elevations were not statistically significant. The number of cases was very low; three time periods had no cases of kidney cancer. Kidney cancer rates did not have a statistically significant upward or downward trend over the time period of the study.

Table 7. Annual age-adjusted incidence rates for kidney cancer by five- and six -year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	12.7	5.6	≤3	0.4	2.48	0.00 , 9.74
1978-1982	0.0	5.9	0	0.5	0.00	-
1983-1987	0.0	6.0	0	0.5	0.00	-
1988-1992	11.9	6.7	≤3	0.7	1.50	0.00 , 5.90
1993-1997	0.0	6.1	0	0.7	0.00	-
1998-2003	6.9	8.0	≤3	1.3	0.77	0.00 , 3.00
1973-2003	6.9	6.6	≤3	3.9	0.78	0.15 , 1.90
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

OTHER URINARY

Other urinary cancers include cancers of the ureter and urinary tract cancers that do not originate in either kidney or bladder. Other urinary cancers did not have any statistically significantly elevated SIRs when examined by single five-year periods (Table 8). Rates in Monticello were elevated compared to Utah in two time periods, and lower in four time periods; these findings were not statistically significant. The number of cases was very small, with four time periods reporting no cases. The cumulative SIR for the total time period of the study was also not statistically significantly elevated. Since only two time periods had cases of other urinary cancer, trend analysis was not performed.

Table 8. Annual age-adjusted incidence rates for other urinary cancers by five- and six -year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	29.1	0.5	≤3	<0.1	32.27	0.01, 126.53
1978-1982	0.0	0.6	0	<0.1	0.00	-
1983-1987	18.0	0.4	≤3	<0.1	31.60	0.01, 123.88
1988-1992	0.0	0.3	0	<0.1	0.00	-
1993-1997	0.0	0.2	0	<0.1	0.00	-
1998-2003	0.0	0.3	0	<0.1	0.00	-
1973-2003	5.5	0.4	≤3	0.2	10.07	0.95, 28.85
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

CHRONIC LYMPHOCYTIC LEUKEMIA

No single time period had statistically significantly elevated or decreased SIRs for Chronic Lymphocytic Leukemia (CLL) (Table 9). Monticello rates were higher than Utah rates in two time periods; however these were not statistically significant. The number of cases was very low; four periods had no cases and no period had more than three cases. The cumulative SIR was elevated, but was not statistically significant. Since only two time periods had cases of CLL, trend analysis was not performed.

Table 9. Annual age-adjusted incidence rates for chronic lymphocytic leukemia by five- and six - year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	3.0	0	0.2	0.00	-
1978-1982	0.0	4.0	0	0.2	0.00	-
1983-1987	0.0	3.3	0	0.2	0.00	-
1988-1992	0.0	3.0	0	0.3	0.00	-
1993-1997	9.5	3.3	≤3	0.4	2.49	0.00 , 9.78
1998-2003	6.9	3.1	≤3	0.5	1.95	0.00 , 7.63
1973-2003	5.5	3.2	≤3	1.7	1.17	0.11 , 3.34

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.

Data Source: Utah Cancer Registry, 1973-2003.

ACUTE LYMPHOCYTIC LEUKEMIA

No single time period had statistically significantly elevated or decreased SIRs for Acute Lymphocytic Leukemia (ALL) (Table 10). Monticello rates were higher than Utah rates in two time periods; however these were not statistically significant. The number of cases was very low; four periods had no cases and no period had more than three cases. The cumulative SIR was elevated, but was not statistically significant. Since only one time period had cases of ALL, trend analysis was not performed.

Table 10. Annual age-adjusted incidence rates for acute lymphocytic leukemia by five- and six - year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	1.1	0	0.2	0.00	-
1978-1982	16.5	1.3	≤3	0.2	4.02	0.00 , 15.76
1983-1987	0.0	1.5	0	0.3	0.00	-
1988-1992	0.0	1.4	0	0.2	0.00	-
1993-1997	0.0	1.2	0	0.2	0.00	-
1998-2003	0.0	1.2	0	0.2	0.00	-
1973-2003	1.6	1.3	≤3	1.4	0.73	0.00 , 2.86

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.

Data Source: Utah Cancer Registry, 1973-2003.

MULTIPLE MYELOMA

No single time period had statistically significantly elevated or decreased SIRs for Multiple Myeloma (Table 11). Monticello rates were higher than Utah rates in two time periods; however these were not statistically significant. The number of cases was very low; three time periods had

no cases reported. The cumulative SIR was not statistically significantly elevated. There was no statistically significant trend upward or downward for multiple myeloma rates over the time period of the study.

Table 11. Annual age-adjusted incidence rates for multiple myeloma by five- and six -year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	27.5	3.8	≤3	0.2	9.41	0.89 , 26.98
1978-1982	0.0	4.4	0	0.3	0.00	
1983-1987	11.5	4.3	≤3	0.3	3.31	0.00 , 12.98
1988-1992	0.0	4.7	0	0.4	0.00	
1993-1997	0.0	4.4	0	0.5	0.00	
1998-2003	4.3	4.6	≤3	0.8	1.29	0.00 , 5.06
1973-2003	6.6	4.3	4.0	2.3	1.75	0.45 , 3.87
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

LIVER CANCER

No single time period had statistically significantly elevated or decreased SIRs for liver cancer (Table 12). Monticello rates were elevated in one time period when compared to Utah rates, but this elevation was not statistically significant. Only one time period had liver cancer cases reported. It was not possible to perform trend analysis since only one time period had liver cancer cases.

Table 12. Annual age-adjusted incidence rates for liver cancer by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	1.3	0	0.1	0.00	-
1978-1982	0.0	1.8	0	0.1	0.00	-
1983-1987	0.0	1.8	0	0.2	0.00	-
1988-1992	0.0	2.3	0	0.2	0.00	-
1993-1997	7.2	2.7	≤3	0.3	3.07	0.00 , 12.05
1998-2003	0.0	2.8	0	0.5	0.00	-
1973-2003	1.6	2.3	≤3	1.3	0.79	0.00 , 3.11
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

THYROID

No single time period had statistically significantly elevated or decreased SIRs for thyroid cancer (Table 13). Monticello rates for thyroid exceeded Utah rates in only one time period and it was not statistically significant. The number of cases was very low; four periods had no cases. The cumulative SIR was non- statistically significantly decreased. It was not possible to perform trend analysis since only two time periods had thyroid cancer cases.

Table 13. Annual age-adjusted incidence rates for thyroid cancer by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	4.8	0	0.4	0.00	-
1978-1982	0.0	4.8	0	0.5	0.00	-
1983-1987	18.0	5.2	≤3	0.5	1.95	0.00 , 7.66
1988-1992	0.0	6.4	0	0.6	0.00	-
1993-1997	0.0	6.7	0	0.8	0.00	-
1998-2003	6.9	8.7	≤3	1.4	0.74	0.00 , 2.89
1973-2003	5.5	6.5	≤3	4.2	0.47	0.04 , 1.35
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

CANCERS NOT ASSOCIATED WITH URANIUM MILLING

The following cancer types have not been associated with uranium milling or its products: oral cancers, colorectal cancer, pancreatic cancer, soft tissue cancer, melanoma, breast cancer, uterine cancer, ovarian cancer, prostate cancer, brain cancer, Hodgkin's disease, and non-Hodgkin's lymphoma. Results for these cancers are provided in Appendix C.

Colorectal cancer had statistically significantly fewer cases in Monticello compared to Utah during the cumulative time period, 1973-2003 (SIR=0.57; 95%CI 0.28, 0.95) (Appendix C, Table 15). Prostate cancer also had statistically significantly fewer cases in Monticello compared to Utah during the following time periods: 1993-1997 (SIR=0.28; 95%CI 0.05, 0.69), 1998-2003 (SIR=0.27; 95%CI 0.07, 0.61) and the cumulative time period 1973-2003 (SIR=0.57; 95%CI 0.36, 0.82) (Appendix C, Table 19). Neither of these cancers have been associated with uranium milling.

DISCUSSION

Cancer is a single term that refers to the uncontrolled growth and spread of abnormal cells anywhere in the body. However, cancer is not 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, can be related to many different causes, including genetic predisposition, personal habits such as smoking, and environmental exposures.

Residents of Monticello have expressed concern that they are experiencing higher rates of cancer than would be expected in a population of their size. Completed exposure pathways for potentially cancer-causing contaminants have existed in Monticello in the past. However, that does not indicate whether Monticello residents were actually experiencing excess cancer. The purpose of this investigation was to specifically evaluate cancer incidence in Monticello, Utah from 1973-2003 as compared to Utah.

This investigation did not find evidence of an increase in risk of cancer for residents of Monticello. When cancers of all types were analyzed together, cancer actually occurred at rates lower than expected based on comparison with the remainder of Utah (Table 2). In examining site-specific cancer rates, we similarly found little evidence of increased risk. A statistically significant increase in site-specific cancer was found only for lung cancer during only one time period (1993-1997) (Table 3). For other cancer types, elevated cancer rates were observed during some time periods but these were based on small numbers of cancers and analysis indicated these elevations were not statistically significant, that is they could have easily occurred by chance alone.

LIMITATIONS

In areas with small populations (such as Monticello) the numbers of expected cases of a given cancer are sometimes too small to be appropriately analyzed. These types of cancer cluster investigations lack the statistical power to detect small or medium elevations in cancer rates. For example, during the 1978-1982 period, the power of the study to statistically detect a 50% difference in lung cancer rates in Monticello compared to Utah was 0.20. In other words, there was only a 20% chance that this study would have been able to detect an elevation or decrease in rates if one actually existed. Therefore, the failure of this study to detect an increased risk of cancer does not prove that an increased risk does not exist. Unfortunately, there are few statistical methods available to improve detection of elevated cancer rates in this type of situation.

It is also possible that the Utah Cancer Registry has not fully captured all cancer cases associated with Monticello. Due to the location of the town and the lack of a full-service medical facility in the immediate area, residents may have pursued medical care outside of the state (e.g. in Colorado, Arizona or New Mexico). If a cancer diagnosis is made in a Utah resident in another state, that diagnosis should be reported to that state's cancer registry. Cooperative agreements with those states' cancer registries to share those reports with the Utah Cancer Registry were not established until 1991 for Colorado, 1998 for Arizona, and 2005 for New Mexico. Cancer diagnosed in Utah residents receiving care in those states prior to those agreements would not have been reported to the Utah Cancer Registry. If cases were not ascertained for those reasons, this could have led to an underestimate of Monticello cancer rates.

According to local residents, Monticello has had substantial migration over the past 30 years. This is a common issue in rural areas that have seen stable or declining populations over the past century (Census, 2006). 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 the area where they were exposed. Additionally, persons who have left an area 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. Unfortunately, the methodology of this type of investigation is not able to analyze such migration. To the extent that this migration occurred, it may have decreased the ability of this study to detect an increased risk of cancer in Monticello residents.

It is also important to note that the analysis was conducted by zip code of residence and not specific address. Many persons in the cancer registry provided a Post Office (PO) Box rather than a street address. A PO Box is insufficient to determine whether someone lived within the city limits of Monticello or outside of the city in a more rural area. Physical proximity to the mill site may have a substantial impact on the exposures experienced by residents. Therefore, including residents who may have lived several miles from the mill-site in the analysis could have reduced the chance of detecting an increased risk in those who lived closest to the site. This study was unable to address this issue since physical address was not available for analysis. Additionally, it is possible that people moved within the Monticello area 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.

An additional limitation of this study is the lack of data prior to 1973. 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 done in adult uranium workers and do not address latency time in children who are exposed to uranium milling (ATSDR, 1999a). Even assuming a latency period of 20 years, cancers possibly associated mill-related exposures may have developed as early as 1963. Unfortunately, there are no reliable sources of data on cancer incidence for the time period before 1973.

As with most cancer cluster investigations, this study was unable to address individual-level factors that are related to the development of cancer, such as personal habits, diet, occupational exposures, and familial history. Humans live and work in many environments and are exposed to complex mixtures of toxic pollutants at home and at work. The lack of adequate information on other exposures, such as tobacco, limits our ability to conclude that a statistically significantly elevated level of cancer in a study area is due to a specific exposure, or to conclude that the absence of elevated cancer rates is because exposure resulted in no adverse health effect.

The lack of information on the timing of exposure relative to the development of cancer is another limitation in this kind of study. If cancer is diagnosed before or immediately after an individual experiences an exposure, it is unlikely that the exposure caused the cancer. In the case of Monticello, the prolonged period of possible exposures make it unlikely for this to be an issue. In order to more completely describe a relationship between an exposure and cancer, additional information is needed on the timing of individual exposures, the dose received the duration of exposure and the development of cancer.

CHILD'S HEALTH CONSIDERATIONS

ATSDR and EEP recognize the unique vulnerabilities of infants and children. 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 investigation attempted to evaluate the incidence of pediatric cancers in Monticello. However, between 1973 and 2003, there were three or fewer cases of any cancer in persons between 0 and 18 years old. 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.

INTERPRETATION

As discussed in the above section on limitations, the small size of the Monticello population and the resulting small number of cancer cases make it difficult to interpret these findings. It is possible and we believe likely that the elevated risk of lung cancer observed in a single time period was due to normal variation in cancer rates over time. However, the limitations described above also mean that an elevated risk of one or more types of cancer could exist in residents of Monticello and yet not have been detected by this study.

It is important to examine the consistency of the results within this study. The one cancer type that was found to significantly elevated, was only elevated in a single isolated time period. This is inconsistent with the prolonged exposure to mill-associated contaminants experienced by Monticello residents. Since the exposures occurred over a prolonged period of time (from 1943 until the completion of remediation in 2004), we would expect to see elevations of cancer throughout the time period of the study.

Consistency of findings between studies is also an important aspect in interpreting results. ATSDR (ATSDR, 1997) reviewed cancer-related mortality data from San Juan County between 1950 and 1980. They found increasing mortality over time due to lung and breast cancers; the statistical significance of these findings was not reported. These data were not available for Monticello specifically. An analysis of lung cancer mortality between 1967 and 1992 in Monticello residents demonstrated statistically significantly elevated odds of dying of lung cancer in Monticello residents compared to other San Juan County residents (Odds Ratio= 2.5, 95% CI 1.03-5.8). ATSDR also found evidence of a cluster of four childhood leukemia cases between 1956 and 1965, substantially more than would be expected in a single decade. All of these findings involve data from time periods outside of the period investigated by this study and are not directly comparable to this study. Unlike the ATSDR findings, the results from this study did not find any evidence of elevated breast cancer or leukemia.

Another consideration in interpreting statistical associations is whether the association is biologically plausible. If the cancer types with elevated rates are consistent with the known contaminants and exposures that provides further evidence that the elevated rates are not due to random variation. As discussed previously, uranium, radon, arsenic, beryllium, nickel and

chromium have all been associated with lung cancer. These are all known contaminants of concern for the Monticello community. Therefore, elevated lung cancer is consistent with the known exposures. It is difficult to interpret the significantly lower numbers of cancer cases seen when all cancers were combined. Cancer is not a single disease entity, and each of the cancer types addressed in this study have different risk factors and different physiological processes.

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 United States. This study did not specifically examine smoking rates in Monticello; however, if Monticello has higher rates of smoking than Utah as a whole, this may be a reason for elevated lung 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 D for further discussion of cancer risk factors.

CONCLUSION

Residents of Monticello were exposed to numerous chemical and radioactive contaminants due to activities of the uranium-processing mill. Completed exposure pathways did exist in the past. However, 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. Potential exposure pathways do 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 not find elevated risk for cancer in residents of Monticello and the surrounding area. 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, limiting the ability to correctly count all cancers that might have resulted from exposure in Monticello. Therefore, this study was not able to conclusively determine whether or not cancer rates in the Monticello area are or were truly elevated.

RECOMMENDATIONS

The Environmental Epidemiology Program (EEP) recommends that further analysis of cancer rates in Monticello be performed. Specifically, an attempt should be made to validate cancer cases that were identified through surveys done by the Monticello Victims of Mill Tailings Exposure committee. These surveys may have identified cancer cases that might be missing from the Utah Cancer Registry due to being diagnosed in another state.

The EEP recommends that the feasibility of further epidemiologic studies, be explored. Such studies would need to collect information on individual behaviors such as smoking, migration and exposure to non-mill associated environmental contaminants. Additionally, the specific focus of such studies should be carefully considered. Statistical power and sample size will remain an issue that may not be surmountable by using different study designs. More in depth epidemiological studies are very time and resource intensive and may not provide clear answers to the concerns of the community. The availability of resources to perform such studies should also be considered.

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

PUBLIC HEALTH ACTION PLAN

The EEP will provide the Monticello community with a copy of this health consultation and will meet with the community and other interested parties to discuss the results of this health consultation.

The EEP is working on validating the cancer cases identified through surveys performed by the Monticello Victims of Mill Tailings Exposure committee. This is a lengthy and involved process that may take considerable time to complete. Upon completion of this process, the EEP will repeat the analyses presented in this report including the additional, validated cases where the inclusion is appropriate based on the study methods.

The EEP will collaborate with ATSDR to determine appropriate methods for further evaluating hazardous exposures which have occurred in the Monticello community.

The EEP will explore the feasibility of performing a more in-depth epidemiologic study and whether such a study could overcome some of the limitations presented in this report.

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CERTIFICATION

This Health Consultation, An Investigation of Cancer Incidence in Monticello, Utah, was prepared by the Utah Department of Health, Environmental Epidemiology Program under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the public health consultation was begun. Editorial review was completed by Cooperative Agreement partner.

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The Division of Health Assessment and Consultation, ATSDR, has reviewed this health consultation and concurs with its findings.

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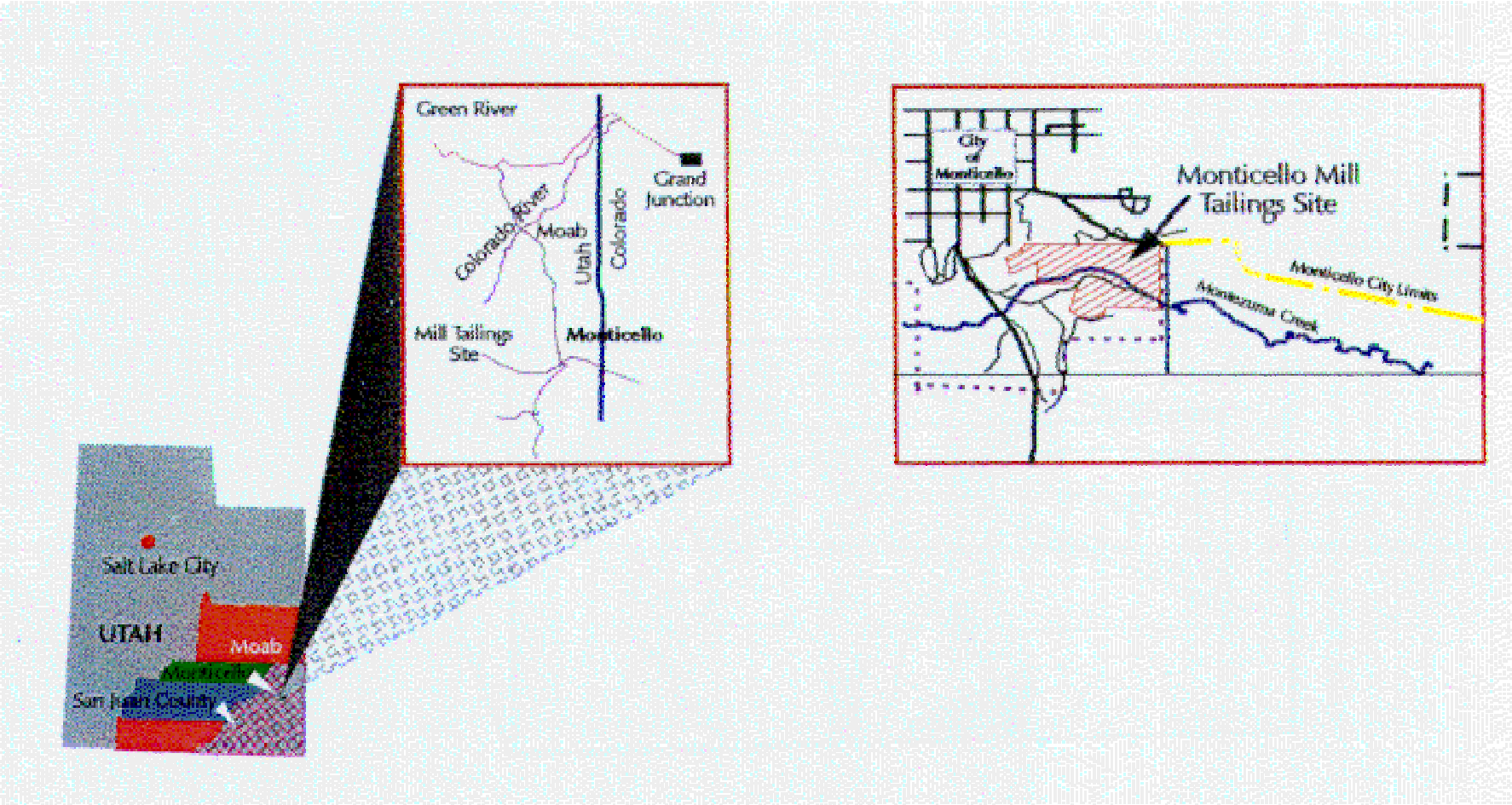
APPENDICES

APPENDIX A – MAP OF STUDY AREA

Figure 2. Map demonstration location of Monticello and the Monticello Mill Tailings Site



APPENDIX B – STATISTICAL DEFINITIONS AND CALCULATIONS

DEFINITIONS

Age Adjustment

Different populations have different numbers of people who are different ages. Cancer rates increase as people get older; therefore, it is not possible to compare two populations with different numbers 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 (persons under 18 or persons over 65). Age adjustment controls 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. Whenever a statistical test is performed, the result is only an estimate of the true result. A 95% confidence interval gives a range of values for the result; 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 greater than five percent chance that the difference found is due to chance alone. If a confidence interval does not include 1.0, then the result is statistically significant; however, this does not **prove** that the cancer rates are elevated.

Generally, as the sample size (or the number of people in your study) increases, the confidence interval becomes more narrow.

Expected number of cases

The expected number of cases is the total number of cases that would be expected if the town had the same cancer rates as the rest of Utah. This is calculated by multiplying the cancer rate in all of Utah 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). When this has been done for all age groups, the numbers are totaled.

Because the expected number of cases is based on mathematical calculations and not real-life scenarios, it is possible for the expected number of cases to be less than one. However, it is not possible to have less than one observed case. In a situation like this, it is difficult to interpret an SIR since it will be elevated if there was even one case of cancer during the time period being examined. It is important to examine the confidence interval and evaluate whether the elevation meets the criteria for significance; this information can assist with deciding whether the SIR is a reliable estimate of cancer risk.

Power

Power is the ability of a study to detect a difference if that difference really exists. 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 one

there. The best way to increase the power of a study is to increase the sample size. In the case of cancer cluster investigations in defined populations, it is not possible to do this.

Sample Size

Sample size refers to the number of people or number of observations in your study. If a town has a population of 2000 and there are 10 cases of cancer, there are a total of 2000 observations. In cancer cluster investigations, the population of the area being examined determines the sample size. It is not possible to change the size of the population or increase the sample size.

Significance

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.

Standardized Incidence Ratio (SIR)

An SIR is used to evaluate whether one population has a higher number of cancers than we would expect if that population had the same cancer rate as the state as a whole. An SIR is calculated by dividing the number of observed cancer cases by the expected number of cancer cases.

A SIR of one (1.0) indicates rates are equal and there is no increased risk. A SIR greater than one (1.0) indicates an increased risk for the study group, while a 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 (SIR) were calculated using a statistical method applicable to both the direct and indirect age-adjustment or standardization methods. This method uses the age distribution of each population group and the age-specific rates for the standard population (state of Utah) to calculate the expected number of cancer cases if the rates of disease were constant as in the standard population. The observed number of incidences is then compared (divided) with the expected number of incidences in the study population (zip code 84535) 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 (zip code 84535))
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:

$$\frac{\left[\sqrt{n} \pm (1.96 \, X \, 0.5)\right]^2}{x}$$

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 C – ADDITIONAL RESULTS

ORAL CANCER

Table 14. Annual age-adjusted oral cancer incidence rates by five and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	13.2	0	0.9	0.00	-
1978-1982	12.1	12.8	≤3	0.9	1.13	0.00 , 4.42
1983-1987	13.1	10.8	≤3	0.8	1.19	0.00 , 4.68
1988-1992	0.0	8.5	0	0.8	0.00	-
1993-1997	0.0	8.3	0	1.0	0.00	-
1998-2003	0.0	6.8	0	1.1	0.00	-
1973-2003	3.3	9.4	≤3	5.4	0.37	0.04 , 1.07
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are less than or equal to three in order to protect the confidentiality of the cases						
Data Source: Utah Cancer Registry, 1973-2003.						

COLORECTAL CANCER

Table 15. Annual age-adjusted colorectal cancer incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	39.9	0	2.3	0.00	-
1978-1982	79.3	38.8	5	2.5	2.02	0.64 , 4.18
1983-1987	0.0	39.4	0	2.8	0.00	-
1988-1992	12.4	38.2	≤3	3.6	0.28	0.00 , 1.10
1993-1997	26.1	34.3	≤3	4.1	0.73	0.14 , 1.78
1998-2003	12.6	34.2	≤3	5.5	0.36	0.03 , 1.04
1973-2003	19.6	36.8	11	19.4*	0.57	0.28 , 0.95
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are less than or equal to 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, 1973-2003.						

PANCREATIC CANCER

Table 16. Annual age-adjusted pancreatic cancer incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	8.9	0	0.5	0.00	-
1978-1982	23.0	7.7	≤3	0.5	2.03	0.00 , 7.98
1983-1987	16.0	8.0	≤3	0.6	1.79	0.00 , 7.03
1988-1992	0.0	7.7	0	0.7	0.00	-
1993-1997	7.2	6.9	≤3	0.8	1.21	0.00 , 4.73
1998-2003	0.0	8.0	0	1.3	0.00	-
1973-2003	6.0	7.8	≤3	4.1	0.74	0.14, 1.81

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are less than or equal to three in order to protect the confidentiality of the cases

Data Source: Utah Cancer Registry, 1973-2003.

BREAST CANCER (FEMALE)

Table 17. Annual age-adjusted female breast cancer incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	32.0	81.9	≤3	2.5	0.40	0.00 , 1.58
1978-1982	11.8	80.5	≤3	2.8	0.35	0.00 , 1.39
1983-1987	57.3	91.8	≤3	3.5	0.86	0.16 , 2.11
1988-1992	63.1	97.8	≤3	4.7	0.63	0.12 , 1.56
1993-1997	74.9	99.9	5	6.1	0.82	0.26 , 1.71
1998-2003	63.8	100.4	5	7.9	0.63	0.20 , 1.31
1973-2003	63.4	94.3	18	26.3	0.68	0.40 , 1.04

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are less than or equal to three in order to protect the confidentiality of the cases

Data Source: Utah Cancer Registry, 1973-2003.

UTERINE CANCER

Table 18. Annual age-adjusted uterine cancer incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	27.2	0	0.9	0.00	-
1978-1982	0.0	27.1	0	1.0	0.00	-
1983-1987	26.5	22.8	≤3	0.8	1.18	0.00 , 4.62
1988-1992	22.1	23.5	≤3	1.2	0.87	0.00 , 3.39-
1993-1997	14.2	21.5	≤3	1.3	0.76	0.00 , 2.96
1998-2003	9.2	20.7	≤3	1.7	0.60	0.00 , 2.37
1973-2003	15.0	23.3	4	6.6	0.60	0.16 , 1.34
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

OVARIAN CANCER

Table 19. Annual age-adjusted ovarian cancer incidence rates by five- and six -year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	15.2	0	0.5	0.00	-
1978-1982	24.7	13.0	≤3	0.5	2.10	0.00 ., 8.22
1983-1987	0.0	14.2	0	0.6	0.00	-
1988-1992	19.7	12.5	≤3	0.6	1.62	0.00 , 6.37
1993-1997	0.0	12.7	0	0.8	0.00	-
1998-2003	0.0	11.5	0	0.9	0.00	-
1973-2003	6.9	12.9	≤3	3.6	0.55	0.05 , 1.57
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

PROSTATE CANCER

Table 20. Annual age-adjusted prostate cancer incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	104.8	99.7	≤3	2.5	1.19	0.22 , 2.92
1978-1982	125.1	121.5	≤3	3.5	0.85	0.16 , 2.07
1983-1987	141.3	126.1	6	4.1	1.47	0.53 , 2.89
1988-1992	83.0	187.9	4	8.6	0.47	0.12 , 1.03
1993-1997	58.9	173.5	≤3	10.7	0.28*	0.05 , 0.69
1998-2003	43.9	170.5	4	14.7	0.27*	0.07 , 0.61
1973-2003	89.4	152.2	23	40.6	0.57*	0.36 , 0.82
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are less than or equal to 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, 1973-2003.						

BRAIN

Table 21. Annual age-adjusted brain cancer incidence rates for other leukemia by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	5.2	0	0.5	0.00	-
1978-1982	7.8	5.6	≤3	0.6	1.75	0.00 , 6.86
1983-1987	0.0	5.9	0	0.6	0.00	-
1988-1992	12.5	6.6	≤3	0.7	1.36	0.00 , 5.32
1993-1997	9.3	5.9	≤3	0.8	1.30	0.00 , 5.10
1998-2003	4.3	5.9	≤3	1.0	1.00	0.00 , 3.90
1973-2003	5.7	5.9	4	4.1	0.97	0.25 , 2.16
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

SOFT TISSUE

Table 22. Annual age-adjusted soft tissue cancer incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	2.3	0	0.2	0.00	-
1978-1982	0.0	1.7	0	0.2	0.00	-
1983-1987	0.0	2.5	0	0.2	0.00	-
1988-1992	0.0	2.0	0	0.2	0.00	-
1993-1997	0.0	2.7	0	0.3	0.00	-
1998-2003	6.0	2.6	≤3	0.4	2.29	0.00 , 8.96
1973-2003	1.6	2.4	≤3	1.5	0.65	0.00 , 2.56
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are three or less to protect the confidentiality of the cases.						
Data Source: Utah Cancer Registry, 1973-2003.						

MELANOMA

Table 23. Annual age-adjusted melanoma incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	9.5	0	0.7	0.00	-
1978-1982	0.0	11.3	0	1.0	0.00	-
1983-1987	12.1	16.6	≤3	1.4	0.71	0.00 , 2.79
1988-1992	21.4	15.0	≤3	1.5	1.34	0.13 , 3.85
1993-1997	22.1	15.5	≤3	1.9	1.62	0.30 , 3.96
1998-2003	3.5	17.8	≤3	2.9	0.35	0.00 , 1.37
1973-2003	10.9	14.7	7.0	8.9	0.79	0.31 , 1.48
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3 when cases are less than or equal to three in order to protect the confidentiality of the cases						
Data Source: Utah Cancer Registry, 1973-2003.						

NON-HODGKIN'S LYMPHOMA

Table 24. Annual age-adjusted Non-Hodgkin's lymphoma incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	10.2	≤3	0.7	0.00	-
1978-1982	12.1	10.6	≤3	0.8	1.29	0.00 , 5.05
1983-1987	16.0	11.8	≤3	0.9	1.09	0.00 , 4.26
1988-1992	29.4	14.5	≤3	1.4	2.15	0.41 , 5.28
1993-1997	24.8	15.1	≤3	1.8	1.64	0.31 , 4.01
1998-2003	13.9	16.2	≤3	2.7	0.75	0.07 , 2.15
1973-2003	16.4	13.7	10	7.8	1.29	0.61 , 2.21

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are less than or equal to three in order to protect the confidentiality of the cases

Data Source: Utah Cancer Registry, 1973-2003.

HODGKIN'S DISEASE

Table 25. Annual age-adjusted Hodgkin's Disease incidence rates by five- and six-year periods and cumulative from 1973-2003 comparing Monticello to Utah – 1973-2003

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	0.0	2.8	0	0.3	0.00	-
1978-1982	17.6	2.9	≤3	0.3	6.14	0.58 , 17.61
1983-1987	7.6	2.7	≤3	0.3	3.29	0.00 , 12.91
1988-1992	0.0	2.1	0	0.3	0.00	-
1993-1997	0.0	2.3	0	0.3	0.00	-
1998-2003	7.6	2.1	≤3	0.3	2.86	0.00 , 11.21
1973-2003	16.4	13.7	4	1.8	2.22	0.58 , 4.93

[1] Standardized Incidence Ratio

[2] 95% Confidence interval

[3] Observed cases are presented as ≤3 when cases are less than or equal to three in order to protect the confidentiality of the cases

Data Source: Utah Cancer Registry, 1973-2003.

APPENDIX D – CANCER EPIDEMIOLOGY

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 United States 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. In 1900, the life expectancy was 47 years compared to 77 years in 2000 (Health, United States, 2005). Because cancer is caused by accumulated changes in our cells, it becomes more likely as we get older. Thus, longer lifespans 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

In children the most common cancers 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. However, few studies have been able to show a consistent link between cancer and these factors.

CANCER RISK FACTORS

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. Some years the rates are higher, other years lower, eventually the rates tend to balance out over time.

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.

CANCERS OF CONCERN

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

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 a Sexually Transmitted Disease (STD), the Human Papilloma Virus (HPV). However environmental exposures, such as tobacco, may also play a role in cancer formation (NCI, 2006).

Chronic Lymphocytic Leukemia

Chronic lymphocytic leukemia (CLL) is predominantly seen in the elderly. It is more common in males than females for unknown reasons. Risk factors for CLL are not completely understood (UCR, 1996). This disorder has not been convincingly linked to any myelotoxic agent and sufficient data 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 United States. 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 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 United States, 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).

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 minority of cases are caused by exposures that damage the liver, such as excessive alcohol consumption, and exposures that may be directly genotoxic, such as dietary aflatoxin (primarily produced by two Aspergillus species of mold) 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 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 from military exposures to atomic and thermonuclear weapons. 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 (BCME). Risk appears to decrease following cessation of exposure, suggesting that the chemical may affect late as well as early stages of carcinogenesis (Schottenfeld & Fraumeni, 1996).

Other risk factors implicated in lung and bronchus 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 (UCR 2000). 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 PCE) an excess of lung cancer was observed (Blair 1979). Some studies have suggested a possible association between respiratory cancer with TCDD (Dioxin) exposures (NTP 2001).

Tuberculosis has also been identified as a risk factor for lung and bronchus 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).

Currently more than two percent of the population in Utah will be affected with lung and bronchial cancer in their lifetime (UCR 1996).

Stomach

Stomach cancer is the seventh leading cause of death in the United States. 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 and uranium and coal mining (Shottenfeld and Fraumeni, 1996). Tobacco/smoking is the most important risk factor(s) that has been strongly 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; it accounts for only one percent of all cancers in the United States (NCI, 1996). It 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 (nuclear weapons, nuclear power plant accidents), 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, bisacodly and senna have been associated with thyroid cancer (Shottenfeld and Fraumeni, 1996). Familial history has also been associated with thyroid cancer.

Urinary Bladder

Each year 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; NCI, 1996).

Melanoma

In the United States, 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 United States primarily due to the light-skinned population and outdoor lifestyle (UCR, 1996).

Hodgkin's Disease

Hodgkin's Disease accounts for less than two percent of all new cancers diagnosed in the United States. 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. In young and middle-aged adults, higher education, higher socioeconomic status, less crowded housing, and early birth position are common risk factors. In older adults the risk for developing Hodgkin's Disease increases with a prior history of living in multiple houses as children. Other risk factors associated with Hodgkin's Disease include exposure to wood or wood products, occupationally exposed rubber or plastic workers, genetics, immunodeficiency, and Epstein-Barr (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 (Shottenfeld and Fraumeni, 1996).

Environmental factors associated with prostate cancer include the use of tobacco, alcohol consumption and exposure to cadmium (a non-essential trace element).

Brain Cancer

In the United States, 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, barbiturates, chemotherapy (in utero), medications, 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 (DDT) (Hecht and Hoffmann, 1991; 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 (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), 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 (Shottenfeld and Fraumeni, 1996). Colon cancer is the third leading cause of cancer-death among both men and women. Currently more than three percent of Utahns will be affected with colorectal cancer in their lifetime (UCR, 1996).

APPENDIX E

International Classification of Diseases for Oncology – 3rd Edition

Cancer types that are starred (*) have been associated with contaminants of concern. Please see Appendix E for a list of the International Classification of Diseases for Oncology (3rd edition) codes that were used to select the cancers included in this study.

Cancer Type	ICD-O-3 code †
Gastrointestinal Tract	
Oral Cavity & Pharynx	C00.0-C10.9
*Stomach	C16.0-C16.9
Colorectal	C18.0-C18.9, C26, C19.9, C20.9
Liver & Intrahepatic Bile Duct	C22.0-C22.1
*Gallbladder & Biliary Ducts	C23.9-C24.9
Pancreas	C25.0-C25.9
Urinary Tract	
*Bladder	C67.0-C67.9
*Kidney & Renal Pelvis	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	
*Lung & Bronchus	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)
*Multiple Myeloma	M9731-9732, M9734
***Acute** Lymphocytic Leukemia	(All Sites) M9826, M9835-M9837
***Chronic** Lymphocytic Leukemia	(Sites C420, C421, C424) M9823
† Lymphomas were excluded from all solid-tissue cancer sites and were analyzed as a separate category	

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	