Health Consultation

AN INVESTIGATION OF CANCER INCIDENCE IN CENSUS TRACTS 126901, 127002, 127003, AND 127004

BOUNTIFUL, WEST BOUNTIFUL, AND WOODS CROSS, DAVIS COUNTY, UTAH

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Summary

The Bountiful/Woods Cross 5th South PCE Plume is located approximately 11 miles north of Salt Lake City, in the Bountiful, West Bountiful, and Woods Cross areas of Davis County, Utah. The contaminated groundwater plume is approximately 245 acres in size. The U.S. Environmental Protection Agency (EPA) and the Utah Department of Environmental Quality (UDEQ) continue to study the extent of the contamination. The primary contaminants in the groundwater are tetrachloroethene (PCE) and associated volatile organic compounds (VOCs) such as trichloroethene (TCE), dichloroethene (DCE), vinyl chloride, methyl-tert-butyl-ether (MTBE) and benzene. The Agency for Toxic Substances and Disease Registry (ATSDR) has requested that the Environmental Epidemiology Program (EEP) of the Utah Department of Health conduct this public health consultation to identify health hazards posed by this plume. The site is classified as an indeterminate health hazard until more information is collected.

In 1996, EPA and UDEQ discovered PCE, TCE, DCE, vinyl chloride, and related chemicals in the groundwater in the Bountiful/Woods Cross area. PCE is a synthetic chemical used for dry cleaning fabrics and metal-degreasing, as well as other industrial uses. TCE, DCE, and vinyl chloride are breakdown products of PCE. In the Bountiful/Woods Cross area, exposure to PCE, TCE, 1,2-DCE, MTBE, vinyl chloride, and benzene is possible from drinking water from contaminated wells (monitoring and residential). Forty-five municipal wells are located within a four-mile radius of the site. The wells are public supply wells for the south Davis County area and are part of blended drinking water systems. Three of the wells are within 1/4 to one mile of the site. Only the Woods Cross Well #1, located at 300 West 1500 South in Bountiful, has been contaminated with PCE above the maximum contaminant level.

EPA and UDEQ continue to study the site to identify other possible sources of contamination. Additional sampling was conducted in June 2003. Results indicate that contaminant levels, with the exception of PCE, are below levels harmful to human health. Cleanup plans will remain uncertain until the nature of the contamination and related sources are better understood. A plan of action has been designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment from the Bountiful/Woods Cross 5th South PCE Plume.

Concerned local residents requested the Environmental Epidemiology Program to evaluate the incidence of cancer in the Bountiful/Woods Cross 5th South PCE Plume area.

Cancer data for this investigation were obtained from the Utah Cancer Registry for the state of Utah (comparison population) and for census tracts 126901, 127002, 127003, and 127004, respectively. These tracts surround the Bountiful/Woods Cross 5th South PCE plume and include Bountiful, West Bountiful, and the Woods Cross area. The data were broken down into the following periods: 1978–1981 (4 years), 1982–1986 (5 years), 1987–1991 (5 years), 1992–1996 (5 years), 1997–2001 (5 years), and 1978–2001 (24 years). The year 2001 was the most recent year for which complete data were available.

Population demographics for the selected census tracts and the state of Utah were obtained from 1970, 1980, 1990, and 2000 U.S. Census data. The state of Utah was selected as the comparison population *minus* the population and the observed number of cases found in the census tracts.

Standardized incidence ratios were calculated for each period and used to determine if a greater or lower risk of developing cancer exists as compared with the comparison population. Confidence intervals (95%) were applied to determine if a statistically significant difference had occurred in the number of observed cases versus the number of expected cases. Incidence rates were also age-adjusted to the 2000 U.S. Standard population (per 100,000 person years) (a unit of incidence measurement)

The results of the investigation did not find any cancer type that was statistically significantly increasing at a greater frequency in the Bountiful, West Bountiful, and the Woods Cross area as compared to the state of Utah from 1978–2001. However, several cancers that were not significantly increased demonstrated incidence rates consistently higher than the state of Utah in at least five of the periods evaluated (includes the cumulative period of 1978–2001). Testicular cancer demonstrated high incidence rates in five of the periods, and cancer of the soft tissue demonstrated consistently higher rates in all the periods evaluated.

IEEP is recommending that the communities living near the Bountiful/Woods Cross 5th South PCE Plume be provided with cancer and site remediation information and a copy of this health consultation.

Background

Site Description

The Bountiful/Woods Cross 5th South PCE Plume (henceforth referred to as the Bountiful Plume) is located between 400 North to 750 South, and 200 West to 1100 West in the Bountiful/Woods Cross area of Davis County. This site is in north-central Utah, approximately 11 miles north of Salt Lake City and is sandwiched between the Wasatch Mountains to the west and the Great Salt Lake to the east (Appendix A). More specifically, "the site is bounded by private residences and agricultural lands on the west, commercial properties and residences to the south, industrial sites and residential properties to the north, and interstate highway 15, railroad tracks, and commercial properties progressively farther east" (1). (ATSDR needs to be consistent with reference citing protocols)

The extent of the contaminated groundwater is approximately 245 acres (1). The vertical depth of contamination is unknown but may be over 100 feet deep (1). The plume has not yet been completely defined, and the investigation is still underway. Multiple sources are likely in this area (2). The former W. S. Hatchco/J. B. Kelley Trucking facility located at 643 South 800 West has been identified as a responsible party for a portion of the site.

In the Bountiful Plume area, exposure to PCE, TCE, 1,2-DCE, MTBE, vinyl chloride, and benzene is possible from drinking water from contaminated wells. Water from municipal wells is considered safe to drink.

Possible sources for these types of contaminants include businesses that routinely use solvents, generally as cleaning agents. Dry cleaners, automotive and machinery shops, and facilities with waste oil tanks (often inappropriately used to containerize solvents) are among the most likely sources for this type of contamination. Migration routes for contaminants include spills, leaks from containers, and leaks from sewer lines.

EPA and UDEQ continue to study the site to identify other sources of contamination. Additional sampling was conducted in June 2003. Results indicate that contaminant levels, with the exception of PCE, are below levels harmful to human health. Cleanup plans will remain uncertain until the nature of the contamination and related sources are better understood.

In response to the concerns of local residents regarding the level of cancer in the area of interest, the Environmental Epidemiology Program was requested to evaluate the incidence of cancer within the surrounding area of the Bountiful Plume that include census tracts 126901, 127002, 127003, and 127004.

Study Methods

Cancer Data

Data for this investigation were obtained directly from the Utah Cancer Registry. The Utah Cancer Registry receives reports on each newly diagnosed case of cancer in Utah from hospitals, radiation therapy facilities, pathology laboratories, nursing homes, and physicians. Each newly diagnosed case is assigned to the census tract of residence at the time of diagnosis.

The data from the Utah Cancer Registry was separated by cancer site/type, sex, age group, and year of diagnosis for the residents of the study area (2000 census tracts 126901, 127002, 127003, and 127004) and the state of Utah. Cases were grouped by year into periods. The following periods were used: 1978–1981 (4 years), 1982–1986 (5 years), 1987–1991 (5 years), 1992–1996 (5 years), 1997–2001 (5 years), and 1978–2001 (24 years). The year 2001 was the most recent year for which complete data was available, and 1978 was the first year in which census tract data was available.

Census Data

The population demographics for the study area (2000 census tracts 126901, 127002, 127003, and 127004) and for the state of Utah were obtained from the 1970, 1980, 1990, and 2000 U.S. Census data, provided electronically by Geolytics CensusCD products. The intercensal populations were estimated linearly from the 1970, 1980, 1990, and 2000 populations. The population estimates were based on the assumption of a constant rate of growth (Appendix B).

Geographic Data

The 2000 census tracts 126901, 127002, 127003, and 127004 were selected for this study because the tract boundaries closely correspond to the area of concern within Bountiful, West Bountiful, and Woods Cross, and for other data consistency considerations. Census tract 126901 has remained relatively constant throughout the study period. The 1980 census tract 127000 split into two tracts and became 127001 and 127002 in 1990. In the 2000 census, 127001 split again into two tracts and became 127003 and 127004 (Appendix C).

Comparison Population

A comparison population to the study population was selected to evaluate whether the observed cases in the study population are statistically different from that which would be expected if the population had not been at any special risk. The state of Utah, minus the population of the study area, was used as the comparison population for this investigation. From this point after, census tract 126901, 127002, 127003, and 127004 will be referred to as the *Bountiful/Woods Cross area* and the state of Utah will be referred to as *Utah*, unless otherwise specified.

Statistical Analysis

Standardized incidence ratios (SIR) were used for the quantitative analysis of cancer incidence in the evaluation areas and different time periods (3). A SIR was calculated for each period and used to determine if a greater or lower risk of acquiring a disease or condition exists among comparison populations. The SIR is calculated by dividing the crude observed count by the expected count (4). The ratio of observed to expected is then used to determine if a greater or lower risk exists between populations of acquiring a disease or condition. The expected count was calculated by multiplying the age-specific comparison rate (Utah) by the age-specific population of the study population (Bountiful/Woods Cross area) and summing the results. A SIR of 1.0 indicates rates are equal no increased risk exists. A SIR greater than 1.0 indicates an increased risk for the study group, while a SIR less than 1.0 indicates a decreased risk for the study group. Random fluctuations may account for some SIR deviations from 1.0 (Appendix D).

The statistical significance of deviations from SIR=1.0 was evaluated using a 95% confidence interval. The confidence interval for the SIR is the range within which the true SIR value has a specified probability of being included. The specified probability is called the confidence level, and the endpoints of the confidence interval are called the confidence limits. We calculated the confidence limits using the method of Frumkin and Kantrowitz (1987). By assessing the confidence interval, we obtained information about the variability of the data and the statistical significance of the SIR. The differences between the observed versus the expected (or SIRs >1.0) were considered significant (not a random occurrence or due to chance alone) if the confidence interval does not include 1.0. Statistical significance here does *not* mean causally associated. It does mean that the recognized association has stability and may need further evaluation.

The SIRs and associated confidence intervals were calculated using a Microsoft Excel 2002 spreadsheet. The statistical formula for the SIR confidence interval (95%) is presented in Appendix D.

Age-Adjusted Rates

Age-adjusted rates of morbidity (per 100,000 person-years) were calculated through direct standardization and adjusted to the 2000 U.S. Standard Population. This adjustment provides a basis for comparison across populations by reducing the effects of differences in the age distributions of the population being compared. It is computed by using the weighted age-specific rates in the population of interest and the proportions of the persons in the corresponding age groups within a standard population. From this point after, the age-adjusted rates will be referred to as *incidence rates* or *rates*, unless otherwise specified.

Literature Search

A literature search was conducted for associations between the cancers found to be elevated and the contaminants of concern in this investigation. This investigation used the National Library of Medicine's Medical Literature Analysis and Retrieval System (MEDLINE®). The computer files of the National Library of Medicine consist of more than 30 biomedical databases. MEDLINE® contains more than 20 years of bibliographic data from more than 3,600 major medical journals. Our search analysis included bibliographic data for the years 1970 through 2003.

Cancers of Concern

This investigation evaluated all the cancers reported to the cancer registry from 1978–2001 that occurred in the Bountiful/Woods Cross area. The following are the cancers that have a potential association to one or more of the contaminants of concern. The International Classifications of Diseases for Oncology codes are listed next to each of the cancers.

Brain (C71.9)	*Testis (C62.9)*	*Lung (C34.0)*
Liver (C22.0)	*Kidney (C64.9)*	*Myeloid leukemia (M-9860/3)*
Soft tissue (C49.9)	*Esophagus (C15.9)*	

Non-Hodgkin's lymphoma and chronic lymphocytic leukemia (M-9591/3)

Note: Chronic lymphocytic leukemia is now considered a non-Hodgkin's lymphoma (5). Therefore, despite being classified separately by the International Classifications of Diseases for Oncology, the cases for non-Hodgkin's lymphoma and chronic lymphocytic leukemia (NHL/CLL) were combined.

Results

The results of the investigation did not find any of the cancers evaluated to be statistically significantly increasing at a greater frequency in the Bountiful/Woods Cross area as compared to Utah from 1978–2001. This investigation did find two cancers (testis and soft tissue) for which most of the SIRs (and rates) were not statistically significant but were elevated in each of the periods evaluated. These cancers, along with cancer of the esophagus, brain, lung, liver, kidney, NHL/CLL, and myeloid leukemia, are presented below.

Interpretation of these results should be approached cautiously because of the small number of cases diagnosed in any of the periods evaluated.

Tables that present the incidence rates (per 100,000 person years) and the SIRs (with confidence intervals) for the cancers mentioned above are presented in Appendix E.

Cancer of the Testis

The incidence rates of cancer of the testis exceeded the rates of Utah in every period except for 1992–1996. The SIRs were also greater than 1.0 (highest SIR = 1.93) in every period but one (1987–1991). The cumulative SIR (1978–2001) was 1.47, and the cumulative rate also exceeded the rate of Utah (rates = 7.75 vs. 5.42). (See Table 1).

Soft Tissue Cancer

The incidence rates of cancer of the soft tissue exceeded the rates of Utah in every period evaluated, including the cumulative period from 1978–2001. However, these rates are based on

periods with less than three cases from 1978–1996. The SIRs were also greater than one in all the periods evaluated. The highest SIR was observed in 1987–1991 (SIR = 1.56), and the highest incidence rate was observed during 1997–2001 (rate = 4.56). (See Table 2)

Brain Cancer

The incidence rate of brain cancer exceeded the rate of Utah in two periods: slightly in 1987–1991 (rates = 7.20 vs. 7.12) and more than doubled in 1997–2001 (rates = 15.70 vs. 6.73). Except for the period of 1997–2001 (SIR = 2.06), all SIRs were less than 1.0. During the period 1997 – 2001, the observed number of cases (n = 10) exceeded the expected number (n = 4.9) by two times the expected rate. Cumulatively, the rates were slightly higher than the rate of Utah, with a SIR just slightly over 1.0 (SIR = 1.08). (See Table 3)

Lung Cancer

The incidence rates and SIRs of lung cancer were higher in 1978–1981 (rate = 35.70, SIR = 1.13) and in 1997–2001 (rate = 41.69, SIR = 1.34) as compared to Utah. The rates (and SIRs) have been increasing since 1992–2001. (See Table 4)

Liver Cancer

Incidence rates of liver cancer exceeded the rates of Utah for two periods. During 1978–1981 the incidence rate for liver cancer was slightly higher than the rate of Utah (rates = 3.22 vs. 1.92). However, this rate is based on a period with less than three cases. During 1992–1996 the incidence rate was almost three times higher as compared to Utah (rates = 7.84 vs. 2.74). The SIR during this period was 2.53. (See Table 5)

Cancer of the Kidney

Only one period (1997–2001) was observed where the SIR (1.17) exceeded 1.0, where the observed number of cases exceeded the expected, and where the incidence rate exceeded the rate of Utah (rate = 11.27 vs. 9.28). The SIRs and the incidence rates of cancer of the kidney have increased steadily from 1978–2001. (See Table 6)

NHL/CLL

The observed number of cases of NHL/CLL have increased from 1987–2001. However, the observed number of cases did not exceed the expected number of cases in any period evaluated. During one period 1978–1981,the incidence rate exceeded the rate of Utah. The highest SIR was observed in period 1992–1996 (SIR = 1.07). The cumulative SIR was 0.89. (See Table 7)

Myeloid Leukemia

The incidence rates of myeloid leukemia have fluctuated from 1978–2001. The highest incidence rate was observed during 1982–1986 (rate = 8.17). The cumulative SIR (0.80) was less than 1.0, and the incidence rate (3.85) was below the rate of Utah (rate = 4.87). (See Table 8)

Esophagus

Cancer of the esophagus did not have enough cases to evaluate from 1978–2001.

Discussion

The Bountiful Plume is an indeterminate public health hazard. The site characterization is currently incomplete and still under evaluation by UDEQ and EPA. The site contains residential, commercial, and agricultural areas. A crude oil refinery, formerly owned by Phillips 66, is in the center of the site. (The Woods Cross refinery was acquired by Holly Corporation in June 2003.) This area has a contaminated groundwater plume that is approximately 245 acres in size. The contaminants of concern include PCE and associated chemicals, such as TCE and vinyl chloride. With the exception of Woods Cross Well #1, located at 300 West 1500 South in Bountiful, these chemicals have not affected the wells used for the municipal/city water system. The Woods Cross Well #1 has been contaminated with PCE above the maximum contaminant level[1]. Forty-five municipal wells are located within a four-mile radius of the site.

Possible sources for these types of contaminants include businesses that routinely use solvents, generally as cleaning agents. Dry cleaners, automotive and machinery shops, and facilities with waste oil tanks (often inappropriately used to containerize solvents) are among the most likely sources for this type of contamination. Migration routes for contaminants include spills, leaks from containers, and leaks from sewer lines.

In response to the concerns of local residents, the Environmental Epidemiology Program examined the issue of whether an excess of cancer is present in the Bountiful/Woods Cross area. The cancers identified by this investigation that have risk factors associated with chronic exposures to PCE, TCE, and vinyl chloride (primarily through occupational exposures), MTBE, and benzene include non-Hodgkin's lymphoma, renal cell carcinoma, esophageal adenocarcinoma (6), liver cancer (7), brain cancer, cancer of the soft tissue (8), and some cancers of the blood (9). This investigation did not find a statistically significant increase in non-Hodgkin’s lymphoma, kidney (or kidney related), esophageal, liver, brain, soft tissue, lung, testicular, blood-related cancers, or any other cancer type in any of the periods evaluated. Of the cancers mentioned above, only cancer of the soft tissue exceeded the incidence rates of Utah in every period evaluated.

[1] A Maximum Contaminant Level (MCL) is calculated by the United States Environmental Protection Agency. The MCL is the highest level of a contaminant that is allowed in drinking water. MCLs are enforceable standards.

Some variation in cancer rates simply occurs by chance within a family, neighborhood, or community. During the period of 1997 – 2001 the number of brain cancers in the Bountiful/Woods Cross area exceeded the expected number by twice of what would be expected; however, this SIR was not significant. Scientifically, it is difficult to prove that an environmental pollutant caused a cancer increase in a community. Cancer increases identified in a community may not be the result of any single, external cause or hazard. Increases that have scientifically been attributed to a specific cause have been those with chronic occupational exposures. Workers (such as in a factory) are more likely to develop a particular type of cancer because of exposures to a chemical(s) they handle every day. Scientific evidence that links an environmental contaminant(s) to an increased occurrence of cancer is sparse.

Some evidence indicates that chemicals (such as arsenic and chlorination by-products) dissolved in drinking water may elevate the risk of gastrointestinal and urinary tract cancers (10, 11). No evidence has found that gastrointestinal cancers or urinary tract cancers were significantly elevated in the Bountiful/Woods Cross area.

Other cancers that were elevated, but were not statistically significant, were cancer of the testis, and prostate. No literature was found that associated human cancer of the testis and prostate with chronic occupational or environmental exposures to PCE, TCE, or vinyl chloride. One animal study did cite sperm and testicular damage (noncancerous) to animals with long-term exposure to vinyl chloride (9).

This investigation tried to determine if the residents were being exposed to the contaminants and from what source. At this time, no exposure pathway has been identified at this site. The EPA and UDEQ will continue to study the site and try to identify sources of contamination and any potential exposure pathways to the residents. Cleanup plans will remain uncertain until the nature of the contamination and related sources are better understood.

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 (12). Statistically, it is normal for cancer rates to fluctuate in smaller communities. Some years the rates are higher, other years lower; the rates tend to balance out over time.

When a subset of the population is found to have an increased rate of cancer, no definitive tests exists to determine which risk factors caused the cancer. Individual cases may result from unique risk factors present in that population or from background risk factors or genetic factors present in the general population. For example, the expected rate of a particular cancer in the general population may be 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 causal relationships to environmental exposures without exposure measurements or data is difficult. 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. A breakdown of the proportion of cancer deaths, attributed to various behavioral and environmental factors, is listed in the following table (13).

Behavioral and environmental factors	*Percentage attributed to cancer mortality*
Diet	*35%*
Tobacco	*30%*
Infections	*10%*
Reproductive and sexual behavior	*7%*
Occupation	*4%*
Alcohol	*3%*
Geophysical	*3%*
Pollution	*2%*
Medicine and medical procedures	*1%*
Industrial products	*<1%*
Food additives	*<1%*
Unknown	*?%*

From the percentages noted above, we can conclude that of the total cancer mortality attributed to environmental factors, pollution and geophysical factors account for only 5% of the cancer mortality, whereas personal behavior/lifestyle accounts for approximately 75% of the cancer mortality.

The following are risk factors associated with the etiology of the following cancers: testis, prostate, soft tissue, brain, lung, liver, kidney, NHL/CLL, chronic and acute lymphocytic leukemia, and acute and chronic myeloid leukemia.

Testicular

Testicular cancer is relatively uncommon in the United States. It is more commonly diagnosed in men ages 20–44 years. Testicular cancer accounts for only 1% of all cancers in men, and is more commonly diagnosed in whites. (11). Risk factors include cryptorchidism (undescended testicles), family history, occupational exposures, and HIV infection, and being white. About 14% of cases of testicle cancer occur in men with a history of cryptorchidism, but up to 25% of cases occur in the normally descended testicle. Men with Klinefelter's syndrome (a sex chromosome disorder that may be characterized by low levels of male hormones, sterility, breast enlargement, and small testes) are at greater risk of developing testicular cancer (8). Occupational risks include workers exposed to metals, metal dust, and cutting oils, miners, oil and gas workers, leather workers, food and beverage processing workers, janitors, and utility workers (11). A study in which male rats were given high doses of methyl-*tertiary*-butyl ether reported a significant increase in testicular cancer (14).

Soft Tissue

Soft tissue cancer is a general category that includes cancer occurring in muscle, heart, subcutaneous, and other related tissues. Because this category includes a number of different types of cancer, it is difficult to define the etiology associated with all cancers of the soft tissue. In addition, we do not yet know all of the risk factors that may lead to the development of soft tissue cancer. Soft tissue (and bone) malignant tumors are common tumors in children. It is also referred to as musculoskeletal sarcoma, which means a cancer of mesenchymal tissues, such as the bone, soft tissues, and connective tissue. This type of cancer is highly malignant and harmful to children (15).

Workers who were exposed to phenoxyacetic acid in herbicides and chlorophenols in wood preservatives, as well as workers exposed to vinyl chloride, may have an increased risk. High doses of radiation have caused soft tissue sarcomas in some patients. Patients with AIDS (acquired immune deficiency syndrome) often develop Kaposi's sarcoma, which has different characteristics and is treated differently than typical soft tissue cancer. Certain inherited diseases, such as Li-Fraumeni syndrome and von Recklinghausens's disease, are associated with an increased risk for soft tissue cancer (8).

Brain

In the United States, 17,000 new primary cancers of the nervous system are diagnosed each year. These are among the most (rapidly) fatal of all cancers, and only about half (52%) of patients are still alive 1 year after diagnosis. Brain cancer is the 10th most common type of death from cancer. The etiology of the majority of nervous system tumors remains unknown. Environmental agents, such as ionizing radiation, have been clearly implicated in the etiology of brain tumors. Other physical, chemical, and infectious agents suspected of being risk factors have not yet been established as etiologically relevant. Factors associated/suspected in the etiology of childhood and adult brain cancer include N-nitroso 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 (11). Brain cancer may also be connected with breathing vinyl chloride over long periods (16).

Lung and Bronchus

Smoking is by far the leading risk factor of lung cancer. Passive smoking is also a risk factor. Exposure to radon and asbestos are additional factors leading to lung cancer. Smoking plus these exposures greatly increases the cancer causing effects of asbestos and radon. Cancers of the lung increase after radiotherapy for Hodgkin's disease. 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 from insecticides and pesticides and during smelting or tin mining. The risk of lung cancer, mesothelioma, and asbestosis is increased in various asbestos industries. Those include mining, milling, and shipbuilding; textile, gas mask, friction products, and insulation manufacturing; and among cement workers. A high risk of lung cancer was reported in workers exposed to bis(chloromethyl)ether (BCME). Risk appears to

decrease after exposure stops, suggesting that the chemical may affect late as well as early stages of carcinogenesis. (11). An excess of lung cancer has been reported among persons with high dietary intake of foods rich in fat and cholesterol. Other risk factors implicated in lung and bronchus cancer are exposure to asbestos, coal gas, nickel, polycyclic hydrocarbons, chromium, arsenic (11), chlormethyl ethers (17), radon (18), and arsenic, asbestos and coal (19, 20, 21). Tuberculosis has also been identified as a risk factor for lung and bronchus cancer (22). More than 2% of the population in Utah will be affected with lung and bronchus cancer in their lifetime (23). Lung cancer may also be connected with breathing vinyl chloride over long periods of time (16). In a study of workers exposed to dry cleaning solvents (carbon tetrachloride, TCE, and PCE) an excess of lung cancer was observed (24).

Liver

The greatest risk factor for cancer of the liver is persistent infection with the hepatitis B or C virus. This accounts for more than three quarters of the world's cases. The remaining cases are caused by exposures that damage the liver, such as excessive alcohol consumption, and exposures that may be directly genotoxic, such as dietary 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. Liver cancer is also associated with diabetes mellitus (5). In a study in which laboratory mice were exposed to 386 milligrams per kilograms per day (mg/kg/day) of PCE for at least 1 year, the mice developed liver cancer and kidney damage (7). Workers who have breathed vinyl chloride over many years indicated an increased risk of liver cancer (16). In a study of workers exposed to dry cleaning solvents (carbon tetrachloride, TCE, and PCE), a slight excess of liver cancer was observed (24).

Kidney and Renal Pelvis

In the United States, 2% of new cancers are from malignant tumors of the kidney, more in men (60%) than in women (40%). Since the 1970s, 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, even more so with cancer of the renal pelvis. Smoking accounts for a large percentage of these cancers in both men and women. The best way to prevent most of these cancers is to avoid tobacco use. Abuse of prescription analgesics is another risk factor and has been causally linked to this type of cancer. Regular use of prescription diuretics may increase risk. Consistently, obesity has 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 kidney cancer mortality (25). Workers exposed to TCE also have a high risk of developing renal cell carcinoma (6).

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 also some evidence that a major proportion of the 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 subsequent to 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 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 (11). Workers exposed to TCE also have a high risk of developing NHL (6).

Chronic Lymphocytic Leukemia

Chronic lymphocytic leukemia is a disease of later life, predominantly present in the elderly. It is more common in males than females, for unknown reasons. The etiology of chronic lymphocytic leukemia is almost entirely unknown (23). This disorder has not been convincingly linked to any myelotoxic agent, and sufficient data rule out an association with ionizing radiation. This condition does have a reported association with butadiene, ethylene oxide, nonionizing radiation, herbicides, and solvents (26). 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 (23). Some cancers of the blood may also be connected with breathing vinyl chloride over long periods (16) and long-term exposures to TCE and PCE (24).

Acute Lymphocytic Leukemia

Acute lymphocytic leukemia accounts for about 5% of the cancer in the 40 years and older age group. However, it is the most common type of childhood cancer in the nation. Environmental risk factors include occupational exposure to benzene, radiation, farming chemicals, paints, butadiene, styrene, and ethylene oxide. Such exposures have been implicated in the etiology of acute lymphocytic leukemia (11). Childhood leukemia has been associated with pregnancy-related diagnostic X-ray exposure. Children who have inherited certain genetic problems such as Down syndrome are at increased risk of developing acute lymphocytic leukemia, as are children who receive medical drugs to suppress their immune systems after organ transplants (28). Some cancers of the blood may also be connected with breathing vinyl chloride over long periods of time (16) and long-term exposures to TCE and PCE (24).

Acute Myeloid Leukemia

Acute myeloid leukemia accounts for 15%–25% of all childhood leukemia and 20%–40% in children 4 years of age and younger. The incidence of acute myeloid leukemia has increased among men 50 years of age and older. Environmental factors associated in the etiology of acute myeloid leukemia include nonionizing electric magnetic fields, benzene, and ethylene oxide and related chemicals. Occupations associated with acute myeloid leukemia include farmers, embalmers, anatomists, and pathologists (11). There are some cancers of the blood that may also be connected with breathing vinyl chloride over long periods (16) and long-term exposures to TCE and PCE (24).

Chronic Myeloid Leukemia

Chronic myeloid leukemia accounts for approximately 1% to 3% of all childhood leukemias. The incidence of chronic myeloid leukemia is higher among males than females. Unlike other

leukemias, the incidence of chronic myeloid leukemia in the United States is higher among blacks than whites. This leukemia first becomes apparent in the early mid-teens, followed by an increased rise in early adulthood. The rates continue to rise throughout middle age and among the elderly. Environmental risk factors include exposure to benzene, radiation (nonionizing and ionizing), and butadiene. Occupations associated with chronic myeloid leukemia include farmers, welders, metal mill workers, male barbers and hairdressers, and dry cleaners (11). Some cancers of the blood may also be connected with breathing vinyl chloride over long periods of time (16) and long-term exposures to TCE and PCE (24).

Esophagus

Cancer of the esophagus is relatively uncommon and, most often, rapidly fatal, even where medical care meets the highest standards available (11). It is most often associated with tobacco use and alcohol abuse, which may explain the fact that the rates in Utah are only half the national rates (23). It is more prevalent among males than females. Other risk factors associated in the etiology of cancer of the esophagus include genetics, diet, ionizing radiation, silica, and lower socioeconomic status. Occupations at higher risk include plumbers, brass and bronze workers, chimney sweepers, vulcanization workers (11), and workers exposed to TCE (6).

Contaminants

1,2-Dichloroethene

1,2-Dichloroethene (also called 1,2-dichloroethylene) is a highly flammable, colorless liquid with a sharp odor that is noticeable in very small amounts, beginning at a level of about 17 parts per million (ppm). The chemical exists in two forms or as a mixture of both; one form is called cis-1,2-dichloroethene, and the other form is called trans-1,2-dichloroethene. The chemical is commonly released into the environment from industries involved in solvent production, pharmaceutical manufacturing, and rubber extraction. When 1,2-dichloroethene is released into air, it takes 5–12 days for half of any amount to break down. When it is released into groundwater, it takes 13–48 weeks for half of a given amount to break down because it has less opportunity to evaporate. Small amounts of 1,2-dichloroethene may break down into vinyl chloride, a more toxic chemical. Also, 1,2-dichloroethene is a breakdown product of other volatile compounds, such as TCE (29).

People can be exposed to 1,2-dichloroethene by breathing contaminated air, by drinking contaminated water, or bathing in contaminated water. Animal studies have shown that once 1,2-dichloroethene is in the body, it is absorbed by the blood and other tissues and is eventually broken down by the liver (29).

EPA has determined that the maximum contaminant level (MCL) for the cis- form is 70 ppb and for the trans- form is 100 ppb (30). The trans- form is approximately twice as potent as the cis- form in its ability to depress the central nervous system (30). On the basis of animal studies,

ATSDR established an oral minimal risk level[2] (MRL) for intermediate exposures of 0.3 mg/kg/day and 0.2 mg/kg/day for the cis- and trans- forms, respectively.

Exposure doses of cis-1,2-DCE were estimated for children and adults because levels detected at the site exceeded ATSDR comparison values for drinking water. If children were to drink groundwater contaminated with the maximum level of cis-1,2-DCE detected on site, exposure would be estimated at 0.372 mg/kg/day, which slightly exceeds the MRL. Dose exposure for adults is much less (0.106 mg/kg/day). Using the most recent analytical data for cis-1,2-DCE (353 ppb), the estimated exposure dose for children would be 0.0353 mg/kg/day, which is below the MRL by a factor of ten; for adults, 0.01 mg/kg/day would be the estimated exposure.

Cis-1,2-dichloroethene does not cause cancer in humans; no studies have been conducted to assess whether trans-1,2-dichloroethene can cause cancer in humans (30).

Tetrachloroethylene (PCE)

PCE has many names. Among these are tetrachloroethylene, perchloroethylene, perc, perclene, and perchlor. PCE is a synthetic chemical that is widely used for dry cleaning of fabrics and metal-degreasing, as well as other industrial uses (9). Exposure to PCE can occur by using certain consumer products. Examples include spot removers, adhesives, wood cleaners, and water repellents.

Exposure to PCE occurred in the past when residents were drinking water from private wells with levels as high as 30 ppb. Exposure doses were calculated for both children and adults and compared to ATSDR's MRLs. The estimated drinking water exposure doses to PCE for children (0.003mg/kg/day) and adults (0.00086 mg/kg/day) are well below the MRL for this chemical (0.05mg/kg/day). Exposure dose estimates were also calculated for children and adults exposed to the maximum concentration of PCE detected in the groundwater in 2003. Again, these results were below the MRL, with adult exposure estimated at 0.0075mg/kg/day, and children at 0.0264 mg/kg/day.

The MRL for PCE, 0.05 mg/kg/day, is considered an estimate of the daily human oral exposure to PCE that is likely to be without appreciable risk of adverse non-cancer health effects. This number is derived from studies in which changes were observed in the behavior of laboratory mice given 5 mg/kg/day of PCE for 60 days (31).

Despite the identification of this MRL, the human health effects of drinking water or breathing in air with low levels of PCE are not definitively known. The effects on infants of consuming PCE in breast milk also are unknown. PCE has been used as a general anesthetic agent and at high concentrations can cause dizziness, amnesia, and loss of consciousness. PCE has also been used to treat hookworm and other intestinal worms (9). Laboratory mice exposed to 386 mg/kg/day for at least one year developed liver cancer and kidney damage (7). Laboratory rats exposed to

[2] Minimal risk level (MRL) is an estimate of daily exposure of a human being to a chemical that is likely to be without an appreciable risk of adverse non-cancer effects over a specified duration of exposure.

900 mg/kg/day for 14 days or less showed neurological, reproductive, and developmental abnormalities (32).

Exposure to PCE can occur through using certain consumer products. Examples include spot removers, adhesives, wood cleaners, and water repellents. Clothes that have been dry-cleaned may release small amounts of PCE into the air (9). In a study of workers exposed to dry cleaning solvents (carbon tetrachloride, TCE, and PCE), an increased risk of malignant neoplasms resulted primarily from an excess of lung cancer and a slight excesses of leukemia and liver cancer (24).

High concentrations of tetrachloroethylene (particularly in closed, poorly ventilated areas) can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness, and death.

The EPA is currently reviewing the carcinogenicity of PCE. The International Agency for Research on Cancer has determined that, based on limited human evidence and sufficient evidence in animals, PCE probably causes cancer in humans. The National Toxicology Program identifies PCE as "reasonably anticipated to be a carcinogen" (33).

Trichloroethene (TCE)

TCE (trichloroethylene) is a non-flammable, colorless liquid with a sweet taste. It has a sweet odor that becomes noticeable at a level of about 100 ppm. The largest source of TCE in the environment is evaporation from factories that use TCE as a solvent to remove grease from metals. TCE can also be found in typewriter correction fluid, paint removers, and adhesives. When TCE is released into air, it takes 7 days for half of any amount to break down. When TCE is released into groundwater, it takes much longer to break down because it has less opportunity to evaporate (34).

People can be exposed to TCE by breathing contaminated air, by drinking contaminated water, or by bathing in contaminated water. When a person breathes air that contains TCE, the blood and other organs absorb about half the amount inhaled; the rest will be exhaled. If a person drinks water that contains TCE, most of the contaminant will be absorbed directly into the bloodstream. If TCE comes in contact with human skin, some of it will enter into the body, although not as much as from inhalation or ingestion. Once TCE is in the body, the liver changes it to other chemicals that are excreted in the urine within a day. If exposure continues, TCE and its breakdown products can build up in body fat (34).

Exposure doses for ingesting groundwater contaminated with TCE at the highest concentration detected (1,380 ppb) were estimated for children and adults. These doses were below the MRL of 0.2 mg/kg/day.

EPA established the MCL of TCE that is permissible in community water systems at 5 parts per billion (ppb). Some studies in humans exposed to TCE in drinking water reported impaired fetal development in pregnant women (34). A New Jersey survey suggested an association between TCE exposure at levels averaging about 55 ppb in water (level >10 ppb) to oral clefts, central

nervous system defects, neural tube defects, and major cardiac defects (34). The small case numbers and exposure classification limited interpretation of the findings of that study.

People who breathe 38–172 ppm of TCE may experience headaches or dizziness. Those levels are about 100 to 1,000 times the amount found in monitoring well water at the Bountiful site. TCE is a mild irritant to the lungs and respiratory tract. However, phosgene and hydrogen chloride, TCE's breakdown products in air, are severe lung irritants (34).

Skin contact with TCE may lead to the development of rashes and skin irritations. However, dermal effects are usually due to direct skin contact with concentrated solutions of TCE. Because the concentration of TCE found in the on-site monitoring well is considered dilute at 650–855 ppb, this concentration is unlikely to cause dermal irritation (34).

The International Agency for Research on Cancer has determined that, on the basis of extensive animal research and limited human data, TCE likely causes cancer in humans (34). A subcohort study of highly exposed workers to TCE found elevated risks for NHL, renal cell carcinoma, and esophageal adenocarcinoma (6). A study of workers exposed to dry cleaning solvents (carbon tetrachloride, TCE, and PCE), found an increased risk of malignant neoplasms. Those resulted primarily from an excess of lung cancer and a slight excess of leukemia and liver cancer (24). Associations between TCE exposure and other cancers are less consistent. More studies are needed to establish the relationship between TCE exposure and cancer.

Vinyl Chloride

Vinyl chloride is a colorless gas at normal temperature. It is also known as chloroethene, chloroethylene, ethylene monochloride, or monochloroethylene. All vinyl chloride is manufactured or results from the breakdown of other manufactured substances, such as TCE and PCE. Most of the vinyl chloride produced in the United States is used to make polyvinyl chloride (PVC). PVC is used in the manufacturing of a variety of plastic products including pipes, wire and cable coatings, and packaging materials. Other uses include furniture and automobile upholstery, wall coverings, housewares, and automotive parts (16).

Liquid vinyl chloride evaporates easily into the air. Vinyl chloride in water evaporates rapidly if it is near the surface. Vinyl chloride released into the air will break down within a few days. The breakdown of vinyl chloride in air often results in the formation of other harmful chemicals. A limited amount of vinyl chloride can dissolve in water. It can enter groundwater and can also be found in groundwater with other chemicals (16).

Breathing high levels (10,000 ppm) of vinyl chloride can cause a person to become dizzy or sleepy. Studies in animals show that extremely high levels of vinyl chloride can damage the liver, lungs, kidneys, and heart, and prevent blood clotting. It is unlikely that vinyl chloride will build up in plants or animals (16).

People who have breathed vinyl chloride for several years, especially at high levels, may experience changes in liver structure. People who have worked with vinyl chloride may suffer from nerve damage or may develop an immune reaction. The lowest levels of exposure that may result in liver damage, nerve damage, or an immune reaction in humans are not known. Certain

occupations related to PVC production expose workers to very high levels of vinyl chloride. These workers may experience problems with blood flow, specifically in the hands. The fingers turn white and hurt when exposed to lower temperatures. In some of these people, the appearance of the skin of the hands and forearms has changed. Also, bones at the tips of the fingers have broken down. Studies suggest that some people may be more sensitive to these effects than others (16).

Some men who work with vinyl chloride have complained of lack of sex drive. Studies in animals showed that long-term exposure might damage the sperm and testes. Some women who work with vinyl chloride have reported irregular menstrual periods and/or high blood pressure during pregnancy. Studies of women who live near vinyl chloride manufacturing plants did not show that vinyl chloride causes birth defects. Studies using pregnant animals showed that breathing high levels of vinyl chloride might harm unborn offspring. Animal studies also show that vinyl chloride may cause increased numbers of miscarriages early in pregnancy. It may also cause decreased weight and delayed skeletal development in fetuses. The same very high levels of vinyl chloride that caused these fetal effects also caused adverse effects in the pregnant animals (16).

Results from several studies have suggested that breathing air or drinking water containing low levels of vinyl chloride may increase the risk of cancer. However, the levels used in these studies were much higher than those found in the ambient air and/or most drinking water supplies at the Bountiful site. Examination of workers who have breathed vinyl chloride over many years indicated an increased risk of liver cancer. Brain cancer, lung cancer, and some cancers of the blood also may be connected with breathing vinyl chloride over long periods. Studies of long-term exposure in animals showed that increases in cancer of the liver and mammary gland may occur at very low levels of vinyl chloride in the air (no range/levels provided). Analysis has shown that animals consuming low levels of vinyl chloride each day during their lifetime also had an increased risk of liver cancer (16).

Child and adult exposure doses for drinking water with the maximum level of vinyl chloride detected at the Bountiful Plume exceed ATSDR's MRL of 0.00002 mg/kg/day. These doses were estimated at 0.0467 mg/kg/day for children and 0.0133 mg/kg/day for adults.

The U.S. Department of Health and Human Services has determined that vinyl chloride is a known carcinogen. The International Agency for Research on Cancer has determined that vinyl chloride is carcinogenic to humans, and the EPA has determined that vinyl chloride is a human carcinogen (16).

Vinyl chloride is regulated in drinking water, food, and air. Because it is a hazardous substance, regulations on its disposal, packaging, and other forms of handling also exist. EPA requires that the amount of vinyl chloride in drinking water not exceed 0.002 milligrams per liter (mg/L) of water (0.002 ppm). Under the EPA's Ambient Water Quality Criteria for the protection of human health, a concentration of zero has been recommended for vinyl chloride in ambient water (16).

Methyl Tert-Butyl Ether (MTBE)

MTBE is the common name for a synthetic chemical called methyl tert-butyl ether. It is a flammable liquid made from combinations of chemicals like isobutylene and methanol. It has a distinctive odor that most people find disagreeable. It was first introduced as an additive for unleaded gasoline in the 1980s to enhance octane ratings. MTBE is an oxygenating agent that enables fuel to burn more efficiently during the winter months. When MTBE is mixed with gasoline, people can come in contact with it if exposed to automobile fuel vapors or exhausts. MTBE has other special uses as a laboratory chemical and in medicine to dissolve gallstones (14).

MTBE will evaporate quickly from open containers. In the open air, it will quickly break down into other chemical compounds, with half of it disappearing in about four hours. Like most ethers and alcohols, MTBE dissolves readily in water. If MTBE is spilled on the ground, rainwater can dissolve it and carry it through the soil into the groundwater. Spills or leaks from storage containers can seep into deeper soil layers and pollute groundwater, especially near manufacturing sites, pipelines, and shipping facilities. Leakage from underground storage tanks, such as tanks at gasoline filing stations, can also add MTBE to groundwater. MTBE is not expected to concentrate in fish or plants found in lakes, ponds, and rivers (14).

Exposure to MTBE can occur from auto exhaust when driving or from gasoline while fueling cars. People can also be exposed to MTBE if they drink polluted groundwater. Low levels of MTBE can be present in both indoor and outdoor air (mostly because MTBE is used as a gasoline additive.

More is known about how MTBE affects the health of animals than the health of humans. Evidence shows that MTBE can affect kidney function in male and female rats exposed at doses as low as 100 mg/kg/day (90 days, oral gavage). Evidence also shows that at higher doses and longer exposure duration (250 and 1000 mg/kg/day respectively, oral gavage for two years), MTBE caused lymphoma and leukemia in female rats and testicular Leydig cell tumors in male rats (35 as described in 14).

Exposure dose estimates for MTBE at the Bountiful/Woods Cross PCE Plume site are estimated at 1.3 mg/kg/day. This level exceeds the minimal risk level for MTBE, calculated at 0.3 mg/kg/day and based on the above-mentioned 100 mg/kg/day oral LOAEL (lowest-observed-adverse-effect-level) for MTBE (14)

Benzene

Benzene is a colorless liquid with a sweet odor. It evaporates into the air very quickly and dissolves slightly in water. It is highly flammable and is formed from both natural processes and human activities (36).

Benzene is widely used in the United States; it ranks in the top 20 chemicals for production volume. Some industries use benzene to make other chemicals that are used to make plastics, resins, and nylon and synthetic fibers. Benzene is also used to make some types of rubbers,

lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene include volcanoes and forest fires. Benzene is also a natural part of crude oil, gasoline, and cigarette smoke (36).

Most people are exposed to a small amount of benzene on a daily basis. Exposure can occur in the outdoor environment, in the workplace, and in the home. Exposure of the general population to benzene is mainly through breathing air that contains benzene (36).

For most people, the level of exposure to benzene through food, beverages, or drinking water is not as high as through air. Typical drinking water contains less than 0.1 ppb benzene. Leakage from underground gasoline storage tanks or from landfills and hazardous waste sites containing benzene can result in benzene contamination of well water. People with benzene-contaminated tap water can be exposed from drinking the water or eating foods prepared with the water. In addition, exposure can result from breathing in benzene while showering, bathing, or cooking with contaminated water (36).

Benzene has been detected at the Bountiful Plume site at levels that exceed ATSDR's comparison value for drinking water. The EPA has set the maximum permissible level of benzene in drinking water at 5 ppb. The levels of benzene detected in groundwater at the Bountiful Plume site are as high as 301 ppb. Exposure doses have been calculated for children and adults drinking groundwater with benzene at this level. ATSDR has not determined an oral MRL for benzene; therefore, the estimated doses were compared to EPA's acute oral reference dose (RfD) of 0.004 mg/kg/day. The estimated exposure dose for children is 0.0301 mg/kg/day, and adults, 0.0086 mg/kg/day. Both doses exceed the RfD.

Although definitive scientific data are not available on oral absorption of benzene in humans, case studies of accidental or intentional poisoning indicate that benzene is absorbed by the oral route. Eating or drinking foods containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and death (36).

The major effect of benzene from chronic (365 days or longer) exposure is on the blood. Benzene causes harmful effects on the bone marrow and can cause a decrease in red blood cells leading to anemia. It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection (36). Long-term exposure to high levels of benzene in the air can cause leukemia, cancer of the blood-forming organs. It is not known whether benzene exposure affects the developing fetus in pregnant women or fertility in men.

The EPA, International Agency for Research on Cancer, and the National Toxicology Program have determined that benzene is a known human carcinogen.

Child 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 also examined the incidence of pediatric cancers in the Bountiful/Wood Cross area and found no excess of cancer among the age group of 0 to 18 years of age.

Limitations of Investigation

Factors that must be considered in the development and etiology of most cancers, but could not be evaluated in this investigation, include latency period, population migration, personal habits, diet, occupational exposures, and familial history. The latency, or induction period, for most adult cancers ranges from 10 to 30 years after initial exposure to a carcinogen. Therefore, ascertaining the place and time of exposure to a carcinogen is difficult. Migration of people into and out of the area presents a problematic issue relative to exposure and latency. Humans live and work in many environments and are exposed to complex mixtures of toxic pollutants at home and at work. Information was not available for individual occupational exposures. Lifestyle factors such as smoking and alcohol consumption could not be examined.

Factors such as latency or induction period, population migration, personal habits, race, diet, occupational exposures, and familial history make drawing a conclusion problematic. In most cancer cluster investigations, no exposure or potential cause is ever apparent or established (37).

The primary objective of a cancer cluster investigation is to identify whether the number of cases that have occurred is significantly greater than what would be expected to occur by chance in the study area. The goal also is to determine if a plausible carcinogenic association of increased cancer rates to the contaminants of concern exists. This investigation should not be viewed as a tool to definitively identify a source to the cancers that are associated or linked to any of the chemicals of concern.

Conclusion

No conclusive evidence was found to suggest that any of the cancers evaluated by this investigation were occurring at a significantly greater frequency in the Bountiful/Woods Cross area as compared to Utah from 1978–2001. This investigation could not identify an exposure pathway to the residents. EPA and UDEQ will continue to study the site and try to identify sources of contamination and potential exposure pathways to the residents. Cleanup plans will remain uncertain until the nature of the contamination and related sources are better understood.

Recommendations

The EEP will provide the communities living near the Bountiful/Woods Cross 5th South PCE Plume with cancer and site remediation information.

Provide the community with a copy of this health consultation.

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Certification

This Health Consultation, "An Investigation of Cancer Incidence in Census Tracts 126901, 127002, 127003, and 127004, Bountiful, West Bountiful, and Woods Cross, Davis County, 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.

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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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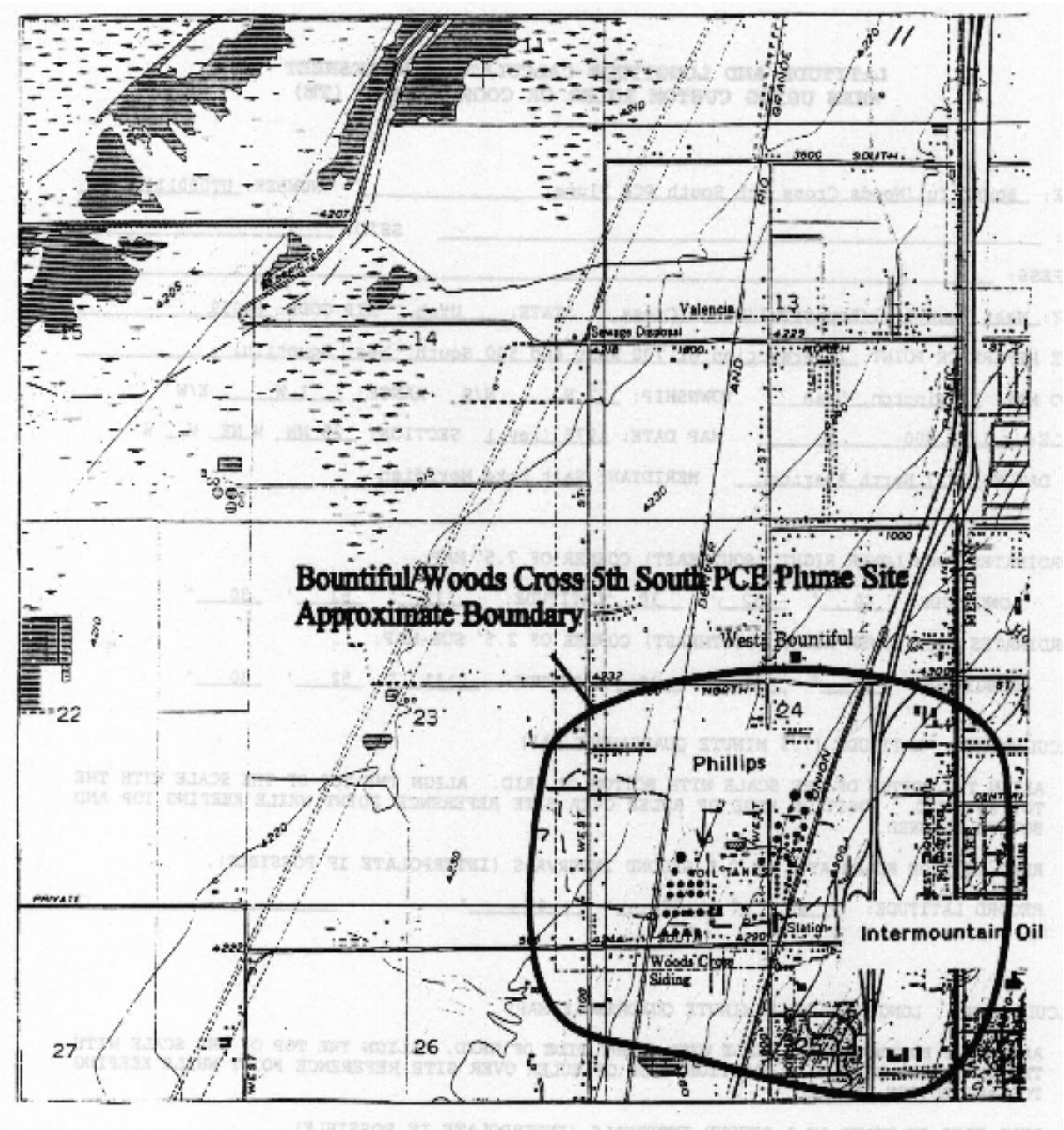
APPENDIX A – 2003 SITE BOUNDARIES of Bountiful/Woods Cross 5th PCE Plume.

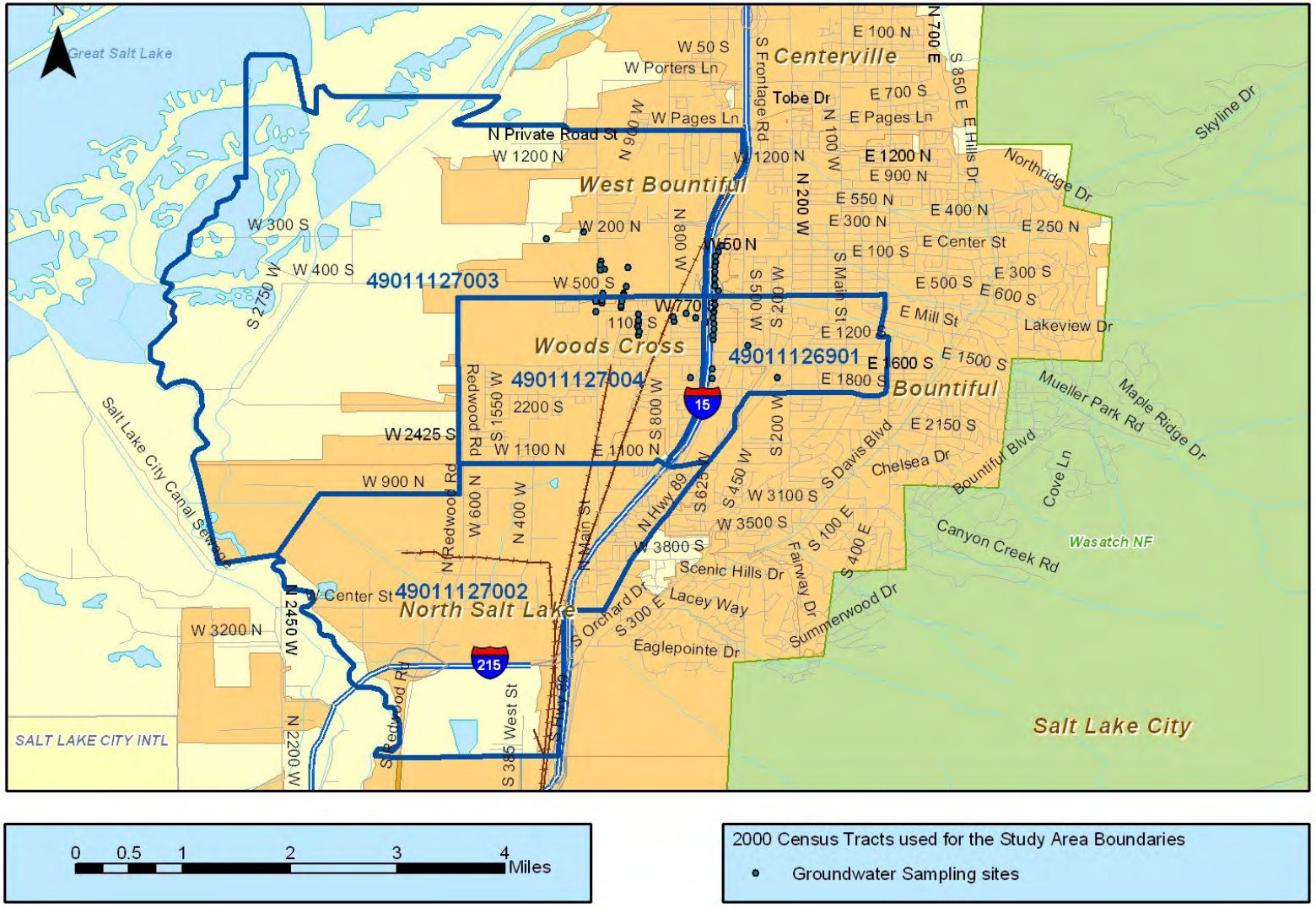


APPENDIX B - Population Estimates

The intercensal population estimates for the Bountiful/Woods Cross area for the years 1970 through 2001. Populations were estimated linearly using the 1970, 1980, 1990, and 2000 U.S. Census data provided by Geolytics CensusCD products. Study area: 2000 Census Tracts 126901, 127002, 127003, and 127004.

	Study Area Population			Utah Population		
Year	male	female	total	male	female	total
1970	5,301	5,229	10,530	523,107	536,166	1,059,273
1971	5,471	5,418	10,889	543,246	556,203	1,099,449
1972	5,642	5,606	11,248	563,386	576,240	1,139,626
1973	5,812	5,795	11,607	583,525	596,277	1,179,802
1974	5,983	5,983	11,966	603,665	616,314	1,219,979
1975	6,153	6,172	12,325	623,804	636,351	1,260,155
1976	6,323	6,360	12,683	643,943	656,388	1,300,331
1977	6,494	6,549	13,042	664,083	676,425	1,340,508
1978	6,664	6,737	13,401	684,222	696,462	1,380,684
1979	6,835	6,926	13,760	704,362	716,499	1,420,861
1980	7,005	7,114	14,119	724,501	736,536	1,461,037
1981	7,062	7,203	14,265	737,586	749,632	1,487,218
1982	7,120	7,291	14,411	750,671	762,728	1,513,400
1983	7,177	7,380	14,557	763,757	775,824	1,539,581
1984	7,235	7,469	14,703	776,842	788,920	1,565,762
1985	7,292	7,558	14,850	789,927	802,017	1,591,944
1986	7,349	7,646	14,996	803,012	815,113	1,618,125
1987	7,407	7,735	15,142	816,097	828,209	1,644,306
1988	7,464	7,824	15,288	829,183	841,305	1,670,487
1989	7,522	7,912	15,434	842,268	854,401	1,696,669
1990	7,579	8,001	15,580	855,353	867,497	1,722,850
1991	7,644	8,064	15,708	881,721	892,161	1,773,882
1992	7,709	8,127	15,836	908,089	916,825	1,824,914
1993	7,774	8,190	15,964	934,456	941,489	1,875,946
1994	7,839	8,253	16,092	960,824	966,153	1,926,978
1995	7,904	8,316	16,220	987,192	990,818	1,978,010
1996	7,969	8,379	16,348	1,013,560	1,015,482	2,029,041
1997	8,034	8,442	16,476	1,039,928	1,040,146	2,080,073
1998	8,099	8,505	16,604	1,066,295	1,064,810	2,131,105
1999	8,164	8,568	16,732	1,092,663	1,089,474	2,182,137
2000	8,229	8,631	16,860	1,119,031	1,114,138	2,233,169
2001	8,294	8,694	16,988	1,145,399	1,138,802	2,284,201

APPENDIX C – Census Tracts of the Bountiful/Woods Cross 5th PCE Plume, 2004.



APPENDIX D - Statistical Calculations

Age-Adjustment Method (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 (census tract 126901, 127002, 127003, and 127004) 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 (census tracts 126901, 127002, 127003, and 127004)
s = area chosen as a reference standard (state of Utah)
n_{ia} = number of individuals in ith class [ith ???] 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)

(Harold A. Kahn and Christopher T. Sempos, "Statistical Methods in Epidemiology", Oxford University Press, 1989, pp 85-136.)

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 observed.
x is the number expected.

(Frumkin H, Kantrowitz W. 1987. Cancer clusters in the workplace: an approach to investigation. J Occup Med 29(12):949–52.)

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 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 KJ. Greenland S, 1998. Modern Epidemiology. Lipincott-Raven Publishers. pp. 189-191)

APPENDIX E - Tables

Presented are the number of observed cases, expected number of cases, the Standardized Incidence Ratios, and 95% confidence intervals for cancer in the Bountiful/Woods Cross area, census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census) for each of the periods analyzed. The state of Utah was selected as the comparison population. Cancers presented are: *testis, soft tissue, brain, lung and bronchus, liver, kidney, NHL/CLL, and myeloid leukemia*.

The criteria established for determining significance involved two statistical methods:

1. A Standardized Incidence Ratio greater than 1.0.
2. A 95% confidence interval with limits that do not include 1.0.

The following terms and abbreviations are used as following:

- SIR means a Standardized Incidence Ratio.
- Study means the study population.
- Comp means the comparison population.

Table 1. The number of observed and expected, Standardized Incidence Ratios (SIR) with upper and lower 95% confidence limits, and incidence rates (study and comparison) are presented for cancer of the Testis in census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census).

Testis \ Years	1978-81	1982-86	1987-91	1992-96	1997-2001	1978-2001
Observed	<3	3	<3	3	4	<16
Expected	1.0	1.7	2.0	2.4	2.4	9.5
SIR	1.93	1.76	1.00	1.27	1.70	1.47
Upper Limit	5.53	4.30	2.87	3.13	3.77	2.35
Lower Limit	0.18	0.33	0.09	0.24	0.44	0.80
Study Rate	7.97	9.73	7.30	6.03	7.77	7.75
Comp Rate	4.19	4.94	5.42	6.18	5.65	5.42

Data source: Utah Cancer Registry, 2001.

Incidence rates (study and comparison) are the number of cases per 100,000 person years and are age-adjusted to U.S. 2000 standard population.

Observed cases are presented as < 3 when cases are less than three to protect the confidentiality of the cases.

Table 2. The number of observed and expected, Standardized Incidence Ratios (SIR) with upper and lower 95% confidence limits, and incidence rates (study and comparison) are presented for cancer of the **Soft Tissue** in census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census).

Soft Tissue \ Years	1978-81	1982-86	1987-91	1992-96	1997-2001	1978-2001
Observed	<3	<3	<3	<3	3	<15
Expected	0.8	1.4	1.3	1.9	2.3	7.7
SIR	1.24	1.43	1.56	1.06	1.30	1.30
Upper Limit	4.88	4.11	4.48	3.04	3.20	2.23
Lower Limit	0.00	0.14	0.15	0.10	0.25	0.62
Study Rate	3.22	4.01	3.61	3.34	4.56	3.81
Comp Rate	1.95	2.71	2.12	2.96	3.21	2.70

Data source: Utah Cancer Registry, 2001.

Incidence rates (study and comparison) are the number of cases per 100,000 person years and are age-adjusted to U.S. 2000 standard population.

Observed cases are presented as < 3 when cases are less than three to protect the confidentiality of the cases.

Table 3. The number of observed and expected, Standardized Incidence Ratios (SIR) with upper and lower 95% confidence limits, and incidence rates (study and comparison) are presented for cancer of the **Brain** in census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census).

Brain \ Years	1978-81	1982-86	1987-91	1992-96	1997-2001	1978-2001
Observed	<3	<3	4	3	10	<23
Expected	2.4	3.4	4.4	4.4	4.9	19.5
SIR	0.82	0.58	0.91	0.69	2.06	1.08
Upper Limit	2.36	1.66	2.02	1.68	3.53	1.59
Lower Limit	0.08	0.05	0.24	0.13	0.98	0.67
Study Rate	5.08	2.08	7.20	5.02	15.70	8.03
Comp Rate	5.99	5.96	7.12	6.43	6.73	6.50

Data source: Utah Cancer Registry, 2001.
Incidence rates (study and comparison) are the number of cases per 100,000 person years and are age-adjusted to U.S. 2000 standard population.
Observed cases are presented as < 3 when cases are less than three to protect the confidentiality of the cases.

Table 4. The number of observed and expected, Standardized Incidence Ratios (SIR) with upper and lower 95% confidence limits, and incidence rates (study and comparison) are presented for cancer of the **Lung and Bronchus** in census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census).

Lung&Brn \ Years	1978-81	1982-86	1987-91	1992-96	1997-2001	1978-2001
Observed	10	11	11	15	26	73
Expected	8.9	14.1	16.1	18.5	19.4	76.6
SIR	1.13	0.78	0.68	0.81	1.34	0.95
Upper Limit	1.93	1.31	1.15	1.27	1.90	1.18
Lower Limit	0.54	0.39	0.34	0.45	0.87	0.75
Study Rate	35.70	26.38	21.44	27.32	41.69	30.48
Comp Rate	30.62	32.91	31.66	32.71	31.16	31.91

Data source: Utah Cancer Registry, 2001.
Incidence rates (study and comparison) are the number of cases per 100,000 person years and are age-adjusted to U.S. 2000 standard population.

Table 5. The number of observed and expected, Standardized Incidence Ratios (SIR) with upper and lower 95% confidence limits, and incidence rates (study and comparison) are presented for cancer of the **Liver** in census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census).

Liver \ Years	1978-81	1982-86	1987-91	1992-96	1997-2001	1978-2001
Observed	<3	0	0	4	<3	<10
Expected	0.6	1.0	1.3	1.6	2.1	6.4
SIR	1.75	0.00	0.00	2.53	0.49	0.93
Upper Limit	6.87	0.0	0.0	5.62	1.91	1.83
Lower Limit	0.00	0.0	0.0	0.66	0.00	0.34
Study Rate	3.22	0.00	0.00	7.84	1.45	2.72
Comp Rate	1.92	2.04	2.62	2.74	3.22	2.63

Data source: Utah Cancer Registry, 2001.

Incidence rates (study and comparison) are the number of cases per 100,000 person years and are age-adjusted to U.S. 2000 standard population.

Observed cases are presented as < 3 when cases are less than three to protect the confidentiality of the cases.

Table 6. The number of observed and expected, Standardized Incidence Ratios (SIR) with upper and lower 95% confidence limits, and incidence rates (study and comparison) are presented for cancer of the **Kidney** in census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census).

Kidney \ Years	1978-81	1982-86	1987-91	1992-96	1997-2001	1978-2001
Observed	<3	<3	3	4	7	<20
Expected	2.1	3.2	4.1	4.6	6.0	20.0
SIR	0.47	0.62	0.74	0.87	1.17	0.85
Upper Limit	1.85	1.78	1.81	1.93	2.20	1.30
Lower Limit	0.00	0.06	0.14	0.23	0.46	0.49
Study Rate	3.22	5.62	6.32	7.03	11.27	7.01
Comp Rate	6.73	7.16	7.74	7.80	9.28	7.92

Data source: Utah Cancer Registry, 2001.

Incidence rates (study and comparison) are the number of cases per 100,000 person years and are age-adjusted to U.S. 2000 standard population.

Observed cases are presented as < 3 when cases are less than three to protect the confidentiality of the cases.

Table 7. The number of observed and expected, Standardized Incidence Ratios (SIR) with upper and lower 95% confidence limits, and incidence rates (study and comparison) are presented for **NHL/CLL** in census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census).

NHL&CLL \ Years	1978-81	1982-86	1987-91	1992-96	1997-2001	1978-2001
Observed	5	4	7	13	14	43
Expected	4.9	7.4	10.2	12.1	13.9	48.5
SIR	1.02	0.54	0.69	1.07	1.01	0.89
Upper Limit	2.11	1.19	1.29	1.73	1.61	1.17
Lower Limit	0.32	0.14	0.27	0.57	0.55	0.64
Study Rate	21.41	11.18	13.82	20.66	21.54	17.29
Comp Rate	16.90	17.20	19.78	20.88	21.64	19.70

Data source: Utah Cancer Registry, 2001.
Incidence rates (study and comparison) are the number of cases per 100,000 person years and are age-adjusted to U.S. 2000 standard population.

Table 8. The number of observed and expected, Standardized Incidence Ratios (SIR) with upper and lower 95% confidence limits, and incidence rates (study and comparison) are presented for **Myeloid Leukemia** in census tracts 126901, 127002, 127003, and 127004, from 1978–2001 (2000 Census).

MyelLeuk \ Years	1978-81	1982-86	**1987-91**	1992-96	1997-2001	1978-2001
Observed	<3	3	0	3	<3	<12
Expected	1.5	2.2	2.4	3.0	3.4	12.5
SMR	1.33	1.35	0.00	1.00	0.59	0.80
Upper Limit	3.88	3.31	0.0	2.45	1.71	1.37
Lower Limit	0.13	0.25	0.0	0.19	0.06	0.38
Study Rate	2.59	8.17	0.00	5.37	3.11	3.85
Comp Rate	4.70	4.80	4.62	5.01	5.02	4.87

Data source: Utah Cancer Registry, 2001.
Incidence rates (study and comparison) are the number of cases per 100,000 person years and are age-adjusted to U.S. 2000 standard population.
Observed cases are presented as < 3 when cases are less than three to protect the confidentiality of the cases.