Health Consultation

AN INVESTIGATION OF CANCER INCIDENCE IN CENSUS TRACTS – 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, AND 1259.06, 1978-2001

LAYTON, DAVIS COUNTY, UTAH

JUNE 15, 2007

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

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LAYTON, DAVIS COUNTY, UTAH

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Office of Epidemiology
Environmental Epidemiology Program
Under Cooperative Agreement with the
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

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SUMMARY

Since Wasatch Energy Systems (WES) began operations in 1988, a group of residents have been concerned over a perceived increase in the occurrence of brain cancer in the neighboring community. WES operates a waste to energy municipal combustor. The facility burns solid waste from Davis County and the surrounding areas. In 1992, Davis County assumed control and renamed the facility the Davis County Solid Waste Management and Energy Systems Special Service District. In 1997, the Davis County again renamed the facility WES and is currently operating under that title.

The waste materials incineration process is known to emit multiple toxic emissions into the surrounding area. The primary chemical of concern is dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin or TCDD). The Utah Division of Air Quality, a division within the Utah Department of Environmental Quality, issued Notice of Violations to WES in July of 1999, March and October of 2000, and March of 2001 for TCDD levels that exceeded the state designated levels based on stack emission testing.

WES installed a new Air Pollution Control System that was commissioned and put into service in September 2001. The testing for the initial stack (unit A and B) was conducted in October 2001 to evaluate system performance. The compliance stack testing was conducted for the Utah Division of Air Quality in November 2001. The results of the testing demonstrated that particulates, metals, acid gas, and dioxin/furan emissions from the retrofit facility are substantially lower than the required standards and currently (through 2006) continue to be in compliance.

In response to the residents' concerns regarding the level of brain cancer in the area, the Utah Department of Health, Environmental Epidemiology Program (EEP) was requested to evaluate the incidence of cancer within the area surrounding the WES. The area includes census tracts: 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06.

Cancers that were evaluated included brain cancer and cancers that may be associated with long term exposures to TCDD (lung, soft tissue, lymphocytic leukemia, and Non-Hodgkin's lymphoma). Although all cancers with one or more occurrence during the study period were analyzed, brain and cancers associated with TCDD were evaluated more closely. Potential exposure pathways to TCDD include inhalation, ingestion, and dermal contact. Inhalation and consumption of foods contaminated with TCDD are the primary sources of exposure. No ambient air measurements of TCDD or concentrations of TCDD in foods (gardens, goats milk etc) in the area were available for this investigation only stack emissions data. Therefore, the actual exposure levels of residents in the study area could not be evaluated or correlated to the cancer cases in the study area.

Data from the Utah Cancer Registry were used for this investigation. The data received from the Registry covered the years from 1973 – 2003. As mentioned, all cancer types with one or more cases during the study period were analyzed. The state of Utah was selected as the comparison

population. Population estimates for the census tracts were obtained from the 2000 U.S. Census Data. The year 2003 was the most recent year for which complete data were available and 1973 was the earliest year when census tract information was available from the Utah Cancer Registry.

This investigation applied three statistical analytical methods to evaluate the cancer incidence in the census tracts in Layton. Statistical analyses were performed by (1) Standardized Incidence Ratios (SIRs) for single years and by five five-year periods and one six-year period; (2) by SatScan; and (3) by Rapid Inquiry Facility (RIF).

SIRs for each cancer type were calculated by single years and by periods starting with 1973 to evaluate whether the area of concern had a greater or lower risk of developing cancer as compared to the comparison population. Ninety-five percent confidence intervals were applied 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; however, due to extremely small numbers of cancers in persons less than 18 years old, it was not possible to analyze those cases separately.

SatScan (software) was also used to determine the most likely time period and area where a significant cancer cluster occurred based on a spatio-temporal evaluation. This software uses a direct standardization method. The scan statistic compares incidence of cancers within growing space-time windows centered incrementally on each census block group's area centroid and each year in the study period. All possible combinations of centorid location and time are considered.

The RIF is a program that rapidly assesses apparent disease clusters by computing SIRs for disease in the vicinity of the point-sources with the potential emissions of harmful substances adjusted by social deprivation.

There was one completed exposure pathway that did exist in the past for residents living near the WES in Layton. Respirable dust inhalation from facility emission was a primary concern.. However, as mentioned earlier, remediation at the WES was completed in 2001 significantly reducing the levels of TCDD well below the required standards and since has continued to be in compliance through 2006. Therefore, this pathway no longer poses a public health concern for exposure to TCDD.

This analysis found only two cancers (brain, and lung/bronchus) that were statistically significantly elevated during the study period. The three statistical methods used by this study found some common periods in time where brain cancer rates were significantly elevated in Layton. These periods include: by SIR – single year (1997 and 1999) and period (1998-2003), by SatScan (1997-1999), and by the RIF (1998-2003). These periods of time range from 1997 through 2003. However, the period of time (from 1997-2003) in which brain cancer cases occurred more frequently in Layton, and is primarily responsible for the statistically significant elevations in the incidence of brain cancer is 1997-1999. The other (most likely) period where significant clustering of brain cancer occurred appears to have been during 1988-1992.

There is no evidence in the scientific literature linking human exposure to TCDD and the development of brain cancer. This investigation could not determine why the rates for brain

cancer during 1997-1999 were statistically significantly elevated in the area surrounding the WES.

The most likely period and year where significant clustering of lung and bronchus cancer occurred appears to have been during 1993-1997, and more specifically during year 1997 in Layton. However, the latency period for the development of lung and bronchus cancer from initial exposures to TCDD from the WES is inconsistent with the scientific literature. Therefore, the rates observed may be attributed to normal variation in lung and bronchus cancer rates over time.

A follow-up statistical review of cancers should be conducted in the census tracts that surround WES when three additional years of cancer data has been compiled by the Utah Cancer Registry. The EEP, in coordination with the Davis County Health Department, will conduct this follow-up health statistics review when the data is available.

The communities living in census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06 should be provided or have access to a copy of this health consultation via the Utah Department of Health Website or through the Davis County Health Department Public Information Office.

The EEP will provide the communities with this information.

BACKGROUND AND STATEMENT OF ISSUES

Background

The city of Layton is in Davis County and is located geographically just north of Salt Lake City in the state of Utah. The land area in Layton is about 20.7 square miles and its elevation is 4,356 feet. According to the 2000 U.S. Census, the population is 58,474 (Census, 2000).

Since 1988, a group of residents have been concerned over a perceived increase in the occurrence of brain cancer in the community surrounding Wasatch Energy Systems (WES). The facility has been operating since 1988, burning solid waste from Davis County and the surrounding areas. According to the Utah Division of Air Quality (UDAQ), a division within the Utah Department of Environmental Quality, Notice of Violations were forwarded to WES for dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin or TCDD) stack emission levels that exceeded state designated levels in 1999, 2000, and 2001 (UDEQ 2000a, UDEQ 2000b, UDEQ 2001). The dioxin/TCDD compliance history and TCDD levels for the WES are located in Appendix A.

The stack emission levels (as cited by the Notice of Violations) exceeded the 360 nanograms (ng = 1/1,000,000,000 of a gram) per dry standard cubic meter (ng/dscm) adjusted to 7% oxygen as set by Condition 7 of the Approval Order (#DEQE-850-96) set by UDAQ dated September 10, 1996 in accordance with Utah Administrative Code R307-1-3.1 (UDEQ 1996).

Health effects related to short-term human exposure of high levels of TCDD may result in skin lesions, such as chloracne[1] and patchy darkening of the skin, and altered liver function. Long-term exposure is linked to impairment of the immune system, the developing nervous system, the endocrine system, reproductive functions, and cancer (WHO, 1999).

In 2002, the University of Utah conducted an epidemiological investigation in the area surrounding the WES. The investigation, "Was There an Epidemic of Environmentally Caused Brain Cancer in Davis County? Answers from an Epidemiological Investigation," found no evidence of an excess brain cancer burden in Layton nor found any scientific association with the development of brain cancer and exposure to TCDD (Biggs et al 2002). This study evaluated the incidence of cancer from 1990 through 2000.

In response to the residents' concerns regarding the perceived high level of brain cancer in the area, the Environmental Epidemiology Program (EEP) was requested to evaluate the incidence of cancer within the surrounding area of the incinerator (WES) that include census tracts: 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06, respectively (See Map A - Appendix B). This study evaluated the incidence of cancer from 1973 through 2003 and includes a smaller area than the University of Utah study.

[1] Chloracne is a skin disease, often accompanied by severe disfiguration, severe joint pain, headaches, fatigue, irritability and chronic weakness; and it can persist in the body for at least 30 years after exposure (Kimbrough & Grandjean, 1989).

Community Health Concerns

Residents who live in the surrounding area of the WES have expressed great concern about a perceived excess in brain cancers in their neighborhood. The residents are concerned with the levels of TCDD that are emitted from WES. The UDAQ was also concerned with carbon monoxide (CO) emissions. Because the WES facility was going under new emission regulations that required a CO emission limit of 100 ppm based on a four-hour block average several projects to improve the ability to control emission that included CO were implemented. These include control upgrades, control logic modifications, feed grate replacement, grate tile changes, refractory material changes, and combustion air modifications to meet the CO emission limit of 100 ppm based on a four-hour block average (UDEQ 2003). However, this was not the primary concern of the residents.

The residents are also concerned and looking for assurances that site-related contamination from TCDD is not affecting them or is the cause of the perceived excess in brain cancer.

Facility and Site Description

The WES is located at 650 East Highway 193 in Layton, just east of Hill Air Force Base. The WES operates a waste to energy municipal combustor. The facility has been operating since 1988, burning solid waste from Davis County and the surrounding areas.

The area of concern is described as follows: To the northeast and northwest of WES are two golf courses (Hubbard and Sun Hills). Directly east are two residential estates (Eastridge and North Hills), and south of WES are numerous residential areas. The area is primarily residential with some commercial and retail businesses located on 3000 North (south of WES). (See Map B - Appendix B).

Operating History and Process

The WES has been operating since 1988. It was originally owned by DESCO, a subsidiary of Day-Seghers, Inc. In 1992, Davis County assumed control and renamed the facility the Davis County Solid Waste Management and Energy Systems Special Service District. In 1997, Davis County again renamed the facility WES and is currently operating under that title (UDEQ 2000a).

The incineration process of waste materials is known to emit multiple toxic emissions into the surrounding area that include dioxin/TCDD (UDEQ 2001a). TCDD is considered the most toxic of the dioxin congeners (ATSDR 1998). Other toxic emissions Notice of Violations that the WES was served with include hydrogen chloride, CO, hydrogen fluoride, cadmium, and sulfur dioxide. These Notices of Violations were resolved by the WES with UDAQ (UDEQ 2003). None of these contaminants mentioned have been linked to the development of brain cancer.

In July of 1999, March and October of 2000, and March of 2001, UDAQ served WES with Notice of Violations for TCDD stack emission levels that exceeded the 360 ng/dscm (adjusted to 7% oxygen) as set by Condition 7 of the Approval Order. Dates and results of TCDD emissions from the WES are listed in Appendix A, under WES Dioxin Compliance History.

The WES consists of two mass burn refractory lined furnaces. Each produces approximately 52,000 pounds per hour (pph) of steam at 550 pounds per square inch (psi) at 515°F (degrees Fahrenheit). Each incinerator was originally equipped with an Environmental Element Corporation three field Electrostatic Precipitator (ESP) for control of particulate emissions. Acid gases were controlled by dry sorbent injection at the economizer inlet. Performance of the dry sorbent injection system was marginal due to short retention times and poorly controlled temperatures. The dry sorbent injection also aggravated fouling of the economizer section and increased particulate loading to the ESP (UDEQ 2003).

Remediation Activity

WES installed a new Air Pollution Control System that was commissioned and put into service in September 2001. The testing for the initial stack (unit A and B) was conducted in October 2001 to evaluate system performance. The compliance stack testing was conducted for UDAQ in November 2001. The results of the testing demonstrated that particulates, metals, acid gas, and TCDD/furan emissions from the retrofit facility were substantially lower than the required state standards per Condition 7 of the Approval Order, and the 60 ng/dscm (adjusted to 7% oxygen) of the Environmental Protection Agency's (EPA), Part 60, Subpart BBBB Emission Guidelines for Existing Small Municipal Waste Combustors (WES 2003, Federal Register 1999) that became effective in 2005. This standard remains the current EPA compliance standard.

In addition, the WES has completed several projects that have improved their ability to control multiple emissions. These include control upgrades, control logic modifications, feed grate replacement, grate tile changes, refractory material changes, and combustion air modifications (UDEQ 2003).

Demographics

The city of Layton is located in Davis County, Utah approximately 21 miles north of Salt Lake City. The population is 58,474 and its elevation is 4356 feet. The population is 50.4% male and 49.6% female with 87% being white non-Hispanic and 7% Hispanic. The remaining 6% are comprised of other races. The median age is 26.8 years (Census 2000)

In census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06 the total population is 46,758. The population is 51% male and 49% female with 91% of the population being white non-Hispanic. The median age is 26.1 years (Census 2000).

EXPOSURES TO CONTAMINANT

Potential Pathways

To determine whether nearby residents are exposed to contaminants at the site, the U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR) and EEP evaluate the environmental and human components that make up a human exposure pathway. An exposure pathway consists of the following five elements (ATSDR 2004):

(1) A source of contamination,
(2) Transport through an environmental medium,
(3) A point of exposure,
(4) A route of human exposure, and
(5) A receptor population.

ATSDR categorizes an exposure pathway as either *completed, potential, or eliminated*. In a *completed* exposure pathway, all five elements exist and indicate that exposure to a contaminant has occurred in the past, is occurring, or will occur in the future. In a *potential* exposure pathway, at least one of the five elements has not been confirmed, but it may exist. Exposure to a contaminant may have occurred in the past, may be occurring, or may occur in the future. An exposure pathway can be *eliminated* if at least one of the five elements is missing and will never be present (ATSDR 2004).

When an exposure pathway is identified, ATSDR comparison values (CVs) for air, soil, or drinking water are used as guidelines for selecting contaminants that require further evaluation [ATSDR 2004). To protect the more susceptible population, CVs for children are used when available. Since the WES installed a new Air Pollution Control System potential human exposure pathways to TCDD have been significantly reduced to levels less than 3.0 ng/dscm emissions from WES (EPA 2006).

Completed Exposure Pathways

There is one completed exposure pathway for residents living near the WES in Layton: respirable dust inhalation from facility emissions. Elements of the completed exposure pathway are described here.

Completed Exposure: Dust

Exposure element	WES
1) A source of contamination	Air emissions from WES
2) Transport through environmental medium	Airborne contaminants/dust
3) A point of exposure	Residential area
4) A route of human exposure	Inhalation
5) A receptor population	Residents and visitors near the site

A completed pathway of exposure to airborne respirable dust (from facility emissions) is found

due to the proximity of residential homes to the WES in Layton. Examples of this exposure pathway include children playing outside in the area and breathing in small dust particles, residents working in their yards, or visitors running in contaminated air and dust. The inhalation pathway existed in the past and continues to exist because the site exposures are residential with unrestricted access.

Air Quality Monitoring

TCDD/Furan[2] are formed in incinerators through reactions of hydrocarbons and chlorine and in waste heat boilers and controls through reactions of hydrocarbons, chlorine, and particulate in the temperature range between 392 and 932°F (degrees Fahrenheit) (UDEQ 2000).

On September 10, 1996, the Executive Secretary (Utah Air Quality Board) issued an Approval Order (AO) to WES in accordance with Utah Administrative Code (UAC) R307-1-3.1. Condition 7 of that AO limits TCDD/furan emissions from each combustor unit (A and B) to 360 ng/dscm at 7% oxygen (UDAQ 1996). This limit was based on results of TCDD/furan tests conducted in 1993 while the WES was operating under a good combustion practice.

In 1999, the EPA proposed regulations (Federal Register No. 167, Part 40, Subpart BBBB) that required facilities such as WES that were constructed before August 30, 1999 to meet a new TCDD/furan limit of 60 ng/dscm by 2005 (Federal Register 1999). The TCDD/furan limit of 60 ng/dscm remains the current EPA compliance standard to control emissions from existing municipal waste combustion units.

In 1995, the EPA established a level of 1,000 ng/dscm at 7% oxygen as a typical TCDD/furan value (midpoint) for municipal waste incinerators (such as the WES) that did not have add-on controls such as carbon injections (EPA 1995).

From 1996 through 2000, the WES exceeded the TCDD/furan limit in five out of 12 tests conducted by UDAQ. Most of the failure-to-comply violations were resolved with UDAQ. However, Notice of Violations were issued to WES by UDAQ for exceeding the TCDD/furan emission limits as set by Condition 7 of the 1996 AO for the following time periods:

July 1999 -	Unit A - 624 ng/dscm	Unit B - 685 ng/dscm
March 2000 -	Unit A - 1101.1 ng/dscm	Unit B – In compliance
October 2000 -	Unit A - 555.9 ng/dscm	Unit B – In compliance
March of 2001 -	Unit A - 365.4 ng/dscm	Unit B – In compliance

(See Dioxin Compliance History, Appendix A.)

(UDEQ 2000a, UDEQ 2000b, and UDEQ 2001)

[2] Furan, also known as furane and furfuran, is a heterocyclic organic compound, produced when wood, especially pine-wood, is distilled. Furan is a clear, colorless, very volatile and highly flammable liquid with a boiling point close to room temperature. It is toxic and may be carcinogenic (Windholz 1976 and Sax and Lewis 1989).

The WES also exceeded the EPA's midpoint value (1,000 ng/dscm at 7% oxygen) in one out of 12 tests during the same period. However, the average of all 12 tests was below the EPA's midpoint value (UDEQ 2000c).

In September 2001, the WES installed a new Air Pollution Control System that has since substantially lowered TCDD/furan emissions well below the required standards under the now final, 2005 Part 40, Subpart BBBB limit of 60 ng/dscm (WES 2003). According to the EPA, Office of Air Quality and Panning Standards, Energy Strategies Group, the most recent results from the WES (for total TCDD mass production) for 2006 by unit A and unit B is 1.1 ng/dscm and 2.2 ng/dscm, respectively.

Carcinogenicity

TCDD is known to be a human carcinogen on the basis of sufficient evidence of carcinogenicity from studies in humans, involving a combination of epidemiological and mechanistic information that indicates a causal relationship between exposure to TCDD and human cancer (NPT 2001).

The evidence that TCDD is a human carcinogen is also supported by experimental studies in rats, mice, and hamsters that have shown that TCDD induces benign and malignant neoplasms in multiple species. Tumors have been produced in rats, mice, and hamsters, in both sexes, in various strains, in multiple organs and tissues, and from multiple routes of dosing. It was also found that TCDD exposure lead to an increased frequency of cancers in a dose-dependent fashion (NIEHS 2001).

Some studies have shown that chemical workers exposed to higher levels of TCDD have an increased rate of cancer (NIEHS 2001). As mentioned earlier, there are some data that suggest a possible association between non-Hodgkin's lymphoma, soft-tissue sarcoma, respiratory cancer, and lymphocytic leukemia with TCDD exposures. However, the evidence for site-specific cancers in humans is weak (NTP 2001).

Other Health Effects

In addition to cancer, exposure to TCDD can also cause other adverse health effects. According to the EPA there are no "safe" levels of exposure to dioxin. Over 90% of human background exposure is estimated to occur through the diet, with food from animal origin being the predominant source (WHO 1998). Therefore, short and long term exposures to high levels of dioxin may result in different health effects or adverse outcomes.

Short-term exposure to humans of high levels of TCDD may result in skin lesions, such as chloracne and patchy darkening of the skin. It can also cause altered liver function (WHO 1999 and ATSDR 1998).

Long-term exposure is linked to impairment of the immune system, the developing nervous system, the endocrine system, and severe reproductive functions (WHO 1999 and ATSDR 1998).

Some delays in nervous system development as well as changes of behavior have been seen in children of mothers who had been highly exposed to dioxins. The effects were likely due to exposure through the placenta rather than through breast milk (WHO 1998).

In a study conducted in the Netherlands showed that breast fed infants had a better neurobehavioural development compared to formula fed infants. However, within the group of breast fed infants, those receiving milk with higher dioxin content had poorer neurobehavioural test results (WHO 1998).

CONTAMINANT OF CONCERN

Dioxin / TCDD

TCDD is odorless and occurs as a colorless to white crystalline solid. It is insoluble in water, slightly soluble in *n*-octanol, methanol, and lard oil, and soluble in organic solvents (dichlorobenzene, chlorobenzene, benzene, chloroform, acetone). TCDD is stable in water, 95% ethanol, or acetone. It can undergo a slow photochemical and bacterial degradation, but it is normally extremely stable. TCDD is formed during combustion processes and released in emissions from municipal waste and industrial incinerators, forest fires and backyard trash burning, and during manufacturing processes such as herbicide manufacture and paper manufacture. TCDD is one of the most toxic forms of dioxin (ATSDR 1998, NTP 2001).

TCDD is a by-product formed during the manufacture of 2,4,5-trichlorophenol (2,4,5-TCP). 2,4,5-TCP was used to produce hexachlorophene (used to kill bacteria) and the herbicide, 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Various formulations of 2,4,5-T have been used extensively for weed control on crops and rangelands, and along roadways throughout the world. 2,4,5-T was a component of Agent Orange, which was used extensively by the U.S. military in the Vietnam War. In most industrialized countries the use of products contaminated with TCDD has been greatly reduced. Use of hexachlorophene and the herbicide 2,4,5-T is currently restricted in the U.S. (ATSDR 1998, NTP 2001).

The general population may be exposed to TCDD by inhalation, ingestion, and dermal contact. Foods are an important source of exposure (Schecter et al 1997). Meat, fish, and dairy products are the major source (>90%) of human exposure to TCDD. The average daily intake of TCDD for an adult in the U.S. from meat alone was 23 picograms[3] per day, or approximately 50% of the total daily intake from food sources. The average daily intakes of TCDD from milk, produce, and fish were 13, 5, and 5 picograms per day, respectively; however, for certain subpopulations (recreational and subsistence fishers), fish consumption may be the most important source of

[3] Picogram is one-trillionth (10^{-12}) of a gram.

exposure. A developing fetus may also be exposed to TCDD transferred across the placenta and breast-fed babies may be exposed to TCDD in their mother's milk (NTP 2001, ATSDR 1998).

TCDD has been found in plastic packaging, clothes dryer lint, vacuum cleaner dust, room and car air filters, furnace filter dust, and bleached paper products (ATSDR 1998).

UNIVERSITY OF UTAH STUDY

In 2001 the University of Utah conducted an epidemiological investigation entitled "Was There an Epidemic of Environmentally Caused Brain Cancer in Davis County? Answers from an Epidemiological Investigation," in Layton, Davis County, Utah. The investigation evaluated the incidence of brain cancer from 1990-2000 in Layton and whether a putative link between dioxin exposure from the incinerator emissions and the development of brain cancer exists.

A total of 16 census tracts (1990 US Census) were evaluated (1251.01, 1251.02, 1252, 1253.01, 1254.02, 1256, 1257, 1258.01, 1258.02, 1258.04, 1259.03, 1259.04, 1260, 1261.01, 1261.02, and 1261.03, respectively) in North Davis County from 1990 – 2000. Each year was evaluated separately.

The University investigation found elevated risk ratios (RRs) in the incidence of brain cancer in Davis County (specifically in the city of Layton) during years 1997 (RR=2.06), 1998 (RR=1.87), 1999 (RR=2.06) and 2000 (RR=1.06). However, the increases were not considered to be statistically significant. In addition, this investigation did not find a relationship between dioxin exposure and the development of brain cancer.

This investigation differs from the EEP investigation by the areas (census tracts) evaluated. The EEP evaluated census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06 (2000 US Census - tracts) from 1973-2003 in Layton. The University evaluated a larger area in Davis County that included the 16 census tracts mentioned above, of which 10 were specific to Layton.

The University's study evaluated the incidence of brain cancer by single years, which may have limited the statistical power in the analysis. The EEP also evaluated the incidence of brain cancer by single years. The EEP found significant elevated SIRs in 1997 (SIR = 2.14) and 1999 (SIR =1.97). Year 1998 was not considered significant but demonstrated a SIR of 2.15. Most of the years evaluated by the EEP, from 1990-2003, had three or less reported cases of brain cancer. However, the years 1997 through 1999, although still a small sample, all had more than three cases (Table 1). As mentioned earlier, the small sample sizes in years with three or less cases make it difficult to interpret and detect true increases or decreases in the incidence of brain cancer.

The results from the EEP found significant increases by single year during 1997 and 1999. These years fall into the years (1997-2000) that the University of Utah found non-significant increases in the rates of brain cancer in Layton.

Lung and bronchus cancer rates were also examined by the EEP by single years and found that year 1997 (SIR = 2.23, CI 1.15, 3.90) was significantly elevated (Table 7). The University of Utah study did not find any significant or non-significant increases in lung and bronchus cancer.

The statistical methodology used by the University of Utah's investigation was not available for review by the EEP.

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. Cases are assigned to the census tract of residence at the time of diagnosis. 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 broken down by single years from 1973 through 2003 and by five five-year intervals and one six-year interval (1973-1977, 1978-1982, 1983-1987, 1988-1992, 1993-1997, and 1998-2003). Intervals are used to evaluate the temporal distribution and incidence (decreases and increases) of the cancer types (particularly the cancers of concern) during the 31-year period under evaluation. From this point after a single five or six-year interval will be referred to as a *period* unless otherwise specified.

The data for the study area (2000 census tracts - 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) and the state of Utah was categorized by cancer site/type, sex, age group, and year of diagnosis from 1973 through 2003.

Census Data

The population demographics for the study area (2000 census tracts - 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) and 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 on the basis of the 1970, 1980, 1990, and 2000 populations. The populations were estimated on the basis of a constant rate of growth.

Population of Interest

The population under analysis or study area was the residents of Layton who reside in the 2000 census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06. This area was selected for this study by the correspondence of the tract boundaries to the area of concern surrounding WES, and other data consistency considerations. Census tract history is as

follows: From the 1980 census the tracts that were used were 1251.01, 1258.02, 1258.03, 1259.01, and 1259.02, respectively. In 1990, tracts 1258.03, 1259.01, and 1259.02 were reorganized and became 1258.04, 1259.03, and 1259.04. The 2000 census split 1251.01 into 1251.03 and 1251.04, and 1258.02 into 1258.05 and 1258.06, as well as 1259.03 into 1259.05 and 1259.06. Census tract 1259.03 was included for continuity between census year boundary changes.

Population denominator data for specific census tracts and the state of Utah were obtained from the 1970, 1980, 1990, and 2000 U.S. Census Data. As mentioned earlier, the intercensal populations were estimated assuming a linear constant rate of growth between census years.

Comparison Population

A comparison population was selected in order to evaluate whether the observed cases in the study population is statistically different from that which would be expected if the population had not been at any special risk. The state of Utah was used as the comparison population for this investigation. For the purpose of analysis, from this point after census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06 will be referred to as *Layton* and the state of Utah will be referred to as *Utah*, unless otherwise specified.

Statistical Analysis

This investigation employed three statistical methods to evaluate the cancer incidence in Layton. Analyses were performed by single years and periods (based on Standardized Incidence Ratios), by SatScan, and by the Rapid Inquiry Facility.

Standardized Incidence Ratios (SIRs) - Single Year and Periods

The observed and expected numbers of cancer cases were compared and evaluated using SIRs (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 Layton. Five-year age groups were used for the direct standardization. A single SIR was calculated for each cancer in a single year or period. No sub-analysis by age-group (e.g. for persons under 18 years old) was calculated due to small sample sizes. The statistical significance of the SIR was evaluated using 95% confidence intervals. (See Appendix C for a discussion of SIRs, their interpretation and statistical significance.) The confidence intervals were calculated using a non-parametric method to account for the non-normal distribution and small sample sizes using Byar's method (Berslow and Day 1987, Regidor et. al. 1993). Confidence intervals were not calculated for periods in which there was zero (0) observed cases.

Chi-square tests for linear trend were performed for all cancers under evaluation. Fisher's exact test was used for all trend analyses due to the small number of cases.

SatScan

This investigation applied SatScan to evaluate spatio-temporal clusters. SatScan (version 5.1) was developed by Dr. Martin Kulldorff of the Harvard Medical School and Information Management Systems, Inc. to perform spatio-temporal cluster analysis of diseases invents, using the scan test (Kulldorff 2004). This software uses a direct standardization method. The scan statistic compares incidence of cancers within growing space-time windows centered incrementally on each census block group's area centroid (Kulldorff 1997 and Kulldorff et. al. 2004) and each year in the study period. All possible combinations of centorid location and time are considered. The space-time windows are then incrementally enlarged to include adjacent census block groups and consecutive years up to a maximum of 50% of the study area and 50% of the study period. The Poisson probability model was used. Each clustered combination of census block groups and study period years are evaluated for significantly increased incidence of cancer events. Significance was determined by evaluating the distribution of 9,999 Monte Carlo permutations of the data. This test can find none to many clusters of adjacent census block groups and study period years with increased risk. The test is constrained to not allow overlapping clusters. The standardized incidence of cancer cases identified in space and time by the Scan statistic is statistically significant when:

- The number of cases after adjustment were 5 or greater;
- The SIR is greater than 1.0;
- The log-likelihood p-value was less than 0.01.

The cluster of cancer cases in space and time are considered biologically meaningful (Caldwell 1990, Elliott and Wartenberg 2004) when:

- The cluster of cases was statistically significant;
- The SIR was equal to or greater than 2.0;
- The type of cancer has been associated to one or more of the chemicals of concern in a peer-reviewed publication.
- Fifty-one percent (the majority) of the population included within the spatial boundary of the cluster are from the potentially exposed population.

SIRs were calculated by SatScan to evaluate the temporal distribution of the cancer types (cancers of concern) each through the 31 years from 1973-2003 and determine the most likely cancer clusters in specific areas or census tract(s).

Rapid Inquiry Facility (RIF)

This investigation also applied the RIF to evaluate the incidence of cancer. The RIF is a functional extension of the ArcView(r) geographic information system (GIS) software developed by the Small Area Health Statistic Unit, Imperial College of London. The RIF links cancer, population and spatial reference data to identify potentially exposed populations by proximity to geographically defined environmental hazards and compute the disease rate and relative risk statistics for that potentially exposed population. An advantage of the RIF over the traditional SIR used by the UDOH is the implementation of Bayesian smoothing algorithms in the calculations of disease rates for small areas (Aylin et al 1999 and Jarup 2004). Utah cancer data

from 1973 through 2003 obtained from the UCR were geocoded by the EEP and implemented into the RIF database. Ninety percent of the cases statewide from 1988 through 2003 have been geocoded. Prior to that period, the statewide geocoding success rate is significantly reduced. The RIF programming is sensitive to the success of geocoding, therefore, only data from 1988 through 2003 were used for the RIF analysis. The relative risk (RR) of cancer incidence in the study population, compared to the cancer incidence for Utah in five-year periods and one six-year period from 1988-1992 through 1998-2003 were computed using the RIF GIS extension. The RR, as applied by the Imperial College of London, is representative of the SIR previously mentioned.

The RIF provides age x sex and age x sex x socio-economic depravation index (SDI) standardized rates. Six categories, as ranked by the U.S. 2000 census, of median household were used for the SDI. Variation in the observed and comparison case counts used to compute risk for the RIF and the standard SIR are explained by the use of cancer categorization schema. The RIF uses the ICD-10 disease coding schema, where as the SIR were computed using a proprietary UCR site-code schema that closely resembles the SEER site-code schema.

From this point forward the SIR will be used in place of Relative Risk (RR) for the RIF.

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 and review was conducted relative to the cancers of concern and the contaminant of concern for this investigation. This investigation employed the National Library of Medicine's Medical Literature Analysis and Retrieval System. The computer files of the National Library of Medicine consist of more than 30 biomedical databases. Medline contains more than 30 years of bibliographic data from more than 3,600 major medical journals. Search analysis was conducted covering the years 1970 through 2005.

CANCERS OF CONCERN

This investigation focused primarily on brain cancer and cancers that have risk factors associated with TCDD exposures that occurred in Layton from 1973 - 2003. The following (with the exception of brain cancer) are the cancers that may be associated or linked to TCDD exposures.

There was no evidence found in the scientific literature between human exposure to TCDD and the development of brain cancer.

Brain *Lung and Bronchus* *Soft tissue*

Lymphocytic leukemia *Non-Hodgkin's lymphoma (NHL)*

(ATSDR 1998, NTP 2001, and Burmeister et al 1982)

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.

RESULTS

Results by SIR - Year and Periods

By Year

This investigation found significant increase of brain cancer during multiple years and lung cancer during one year from 1973 – 2003. Cancer of the soft tissue, NHL, and lymphocytic leukemia did not demonstrate significant increases in any of the years evaluated.

Tables (1 through 5) that present single year incidence rates (per 100,000 person years), and SIRs (with confidence intervals - CI) for the cancer of concern are presented in Appendix D.

Cancer of the Brain

Statistically significant increases were observed in the occurrence of brain cancer during 1985 (SIR = 3.74, 95% CI 1.01, 9.57), 1988 (SIR = 3.75, 95% CI 1.21, 8.75), 1997 (SIR = 2.23, 95% CI 1.61, 7.36), and 1999 (SIR = 3.04, 95% CI 1.11, 6.61). Due to the small sample sizes it is difficult to assess the yearly rates of brain cancer and perform a meaningful linear trend analyses. From 1973 – 1996 the majority of the single years evaluated contained 3 or less[4] cases of brain cancer (Table 1, Appendix D).

Lung and Bronchus Cancer

A statistically significant increase was observed in the occurrence of lung cancer during 1997 (SIR = 2.23, 95% CI 1.15, 3.90). The rates during 1997 exceeded the rates for Utah by over two-fold. From 1991 through 2003 (period where cases are greater than 3) the rates for lung cancer, for the most part, exceeded the rates of Utah (Table 2, Appendix D). Due to the small sample sizes it is difficult to perform a meaningful linear trend analyses.

[4] Observed cases are presented as 3 or less (≤ 3) in order to protect the confidentiality of the cases.

By Periods

The results of this investigation found the frequency of brain cancer statistically significantly elevated during periods 1988 - 1992 and 1998 – 2003. Lung and bronchus cancer was significantly elevated during period 1993 – 1997. Cancer of the soft tissue, NHL, and lymphocytic leukemia did not demonstrate significant increases or decreases in any of the periods evaluated.

Tables (6 through 10) that present the five-year periods and six-year period with incidence rates (per 100,000 person years), and SIRs (with confidence intervals - CI) for the cancers of concern are presented in Appendix D.

Cancer of the Brain

The incidence rates for brain cancer have consistently increased from period 1988–1992 through 1993-1997 and exceeded the rates of Utah from period 1983-1987 through 2003. Brain cancer was significantly elevated during periods 1988-1992 (SIR = 1.89, 95% CI 1.03, 3.17) and 1998-2003 (SIR = 1.70, 95% CI 1.07, 2.58). Period 1993-1997 demonstrated a non-significant SIR of 1.54. All SIRs exceeded one in all periods except 1978–1982 (Table 6, Appendix D). Therefore, with the exception of period 1993-1997, brain has been significantly elevated by periods from 1988 through 2003. Brain cancer rates did not demonstrate a statistically significant linear trend up or down over the full study period.

Lung and Bronchus Cancer

Lung and bronchus cancer rates exceeded the rates of Utah from period 1983-1987 through 2003. However, only one period was statistically significantly elevated, 1993-1997 (SIR = 1.43, 95% CI 1.01, 1.98). With the exception of period 1978-1982 all SIRs exceeded one (Table 7, Appendix D). Lung cancer rates did not demonstrate a statistically significant linear trend up or down over the full study period.

Results by SatScan

The results (or most likely clusters) of the Scan test found SIRs that were statistically significantly elevated at the 95% confidence probability for cancer of the brain, lung and bronchus, and lymphocytic leukemia. However, only brain cancer was found within the study area, while lung and bronchus cancer, and lymphocytic leukemia were found well beyond the study area.

The census tracts involved within the study area include 1251.03, 1258.04, 1258.05, and 1259.04. The 2000 U.S. Census Population for these census tracts is 18,393, which accounts for only 39.3% of the population of the study area. Furthermore, based on the scientific literature search and review, no evidence was found between human exposure to TCDD and the development of brain cancer. Therefore, although brain cancer was statistically significantly elevated, this outcome is not biologically meaningful.

Cancer of the Brain

A statistically significant SIR (4.70) with a p-value of 0.009 was found for brain cancer during the years of 1997-1999 (Table 11, Appendix D). The number of cases ranged from 5-8 cases from 1997-1999.

Results by RIF

The results of the RIF found brain and lung and bronchus cancer significantly evaluated during multiple periods.

Tables (12 and 13) that present the five-year periods and one six-year period with incidence rates (per 100,000 person years), and SIRs (with confidence intervals - CI) for brain and lung and bronchus cancer are presented in Appendix D.

Cancer of the Brain

The results of the RIF indicate that cancer of the brain was significantly elevated during two periods, 1988-1992 (SIR = 1.95, 95% CI 1.03, 3.17) and 1998-2003 (SIR = 1.71, 95% CI 1.07, 2.58). Period 1993-1997 demonstrated a non-significant SIR of 1.53. (Table 12, Appendix D). Although the SIRs have fluctuated, the rates for brain cancer have increased by period from 1988-1992 through 1998-2003.

Lung and Bronchus Cancer

Lung and bronchus cancer rates were found to be significantly elevated during period 1988-1992 and 1993-1997. Period 1988-1992 demonstrated a SIR of 1.59 (95% CI 1.03, 2.35) and period 1993-1997 demonstrated a SIR of 1.56 (95% CI 1.09, 2.16). During period 1998-2003 (SIR = 1.38) the SIR was elevated but was not significant (Table 13, Appendix D).

DISCUSSION

Since WES began operating in 1988, a group of citizens have been concerned over a perceived increase in the occurrence of brain cancer in the community surrounding WES. The group is concerned with the levels of TCDD that are emitted from WES and suspect that the perceived increase in brain cancer is due to TCDD stack emissions. According to UDAQ, Notice of Violations were forwarded to WES for TCDD levels that exceeded state designated levels during July 1999, March 2000, October 2000, and March 2001(UDEQ 2000a, UDEQ 2000b, and UDEQ 2001). In addition, residents were also concerned with unregulated emissions from the burning of miscellaneous residential garbage in the area (i.e., paints, solvents, pesticides, etc.).

Dioxin is a general term that describes a family of 75 different compounds that are highly persistent in the environment. The most toxic compound is 2,3,7,8-tetrachlorodibenzo-p-dioxin or TCDD. TCDD can be released into the environment through emissions from municipal waste and industrial incinerators. It can enter the body through breathing contaminated air, eating contaminated food, or by having skin contact with contaminated soil or other materials (ATSDR

1998). Most people are constantly exposed to TCDD through ingestion of dioxins that are present at low levels as environmental contaminants in food (NTP 2001). Inhalation and consumption of foods contaminated with TCDD are the primary sources of exposure.

No ambient air measurements of TCDD or concentrations of TCDD in foods (gardens, goats milk etc) were available for this investigation for the study area only stack emission data. Therefore, the actual exposure levels of residents in Layton could not be evaluated or correlated to the cancer cases in the study area.

TCDD is known to be a human carcinogen on the basis of sufficient evidence of carcinogenicity from studies in humans, involving a combination of epidemiological and mechanistic information that indicates a causal relationship between exposure to TCDD and human cancer (NTP 2001).

The problems with epidemiological studies linking exposure to TCDD with human cancer are complicated because TCDD may cause several kinds of cancer and not all exposed people develop cancer. Furthermore, agents other than TCDD may cause the cancer, and it takes time before exposure to TCDD shows its effects. It should also be emphasized that some of the human studies do not provide adequate exposure data and were confounded by concomitant exposure to other chemicals (ATSDR 1998). Some studies have shown that TCDD exposure at high levels in exposed chemical workers have led to an increase in cancer (NIEHS 2001). However, the evidence for site-specific cancers is weak, with some data suggesting a possible relationship between NHL, soft-tissue sarcoma, respiratory cancer (ATSDR 1998, NTP 2001), and lymphocytic leukemia (Burmeister et al 1982) with exposures to TCDD. In 2001, the National Toxicology Program classified TCDD as a human carcinogen (NTP 2001) but does not implicate any site-specific cancers.

There were eight census tracts (2000 Census) evaluated to determine whether an excess of cancers that may have a possible association with TCDD or perceived to be caused by exposures to TCDD by the community were present in the areas surrounding the WES as compared to Utah. In examining the results of cancer incidence in Layton, this investigation did not find a statistically significant increase in NHL, lymphocytic leukemia, or cancer of the soft tissue with any of the methods applied. However, brain cancer and cancer of the lung and bronchus were found to be statistically significantly elevated in multiple periods of time.

The breakdown of the statistical methods used and years and periods where brain cancer was statistically significantly elevated is as follows:

SIR:	Single year: 1985, 1988, 1997, and 1999; Periods: 1988-1992 and 1998-2003;
RIF:	1988-1992 and 1998-2003; and
SatScan:	1997-1999

The three statistical methods used by this investigation found some common periods of time where brain cancer rates were significantly elevated in Layton. These periods include: by SIR -

single-year (1997 and 1999) and period (1998-2003), and by the RIF (1998-2003). Although the results from SatScan are based on 39% of the population in the study area, the incidence of brain cancer was significantly elevated during years1997-1999. Therefore, for the most part, this period of time ranged from 1997 to 2003. However, in examining this period of time more closely, the years were cancer cases occurred more frequently (range = 5 to 8 number of cases) were 1997, 1998, and 1999, respectively (Table 1). These three years are primarily responsible for driving the statistically significant outcomes (elevations) observed from 1997 to 2003.

Although the rates of brain cancer in Layton were significantly elevated, primarily during the years from 1997-1999, the rates for brain cancer have decreased annually from 1998 to 2003 (Table 1).

The other (most likely) period where significant clustering of brain cancer occurred appears to be during period 1988-1992.

This investigation also conducted a comprehensive literature search and review, and determined that there was no epidemiological human evidence or animal studies (rats, mice, hamsters) that demonstrated an association between the development of brain cancer and exposure to TCDD. At this point, this investigation could not determine why brain cancer was significantly elevated during multiple years and periods in Layton.

Although brain tumors account for over 90% of all cancer of the central nervous system, the etiology of brain cancer remains unclear. The environmental agent that has clearly been implicated in the etiology of brain cancer is ionizing radiation (Shottenfeld and Fraumeni 1996).

In evaluating lung cancer rates, based on the three statistical methods, this investigation found rates significantly elevated during the following year or periods of time:

SIR:	Single year: 1997;
	Period: 1993-1997;
RIF:	1988-1992 and 1993-1997; and
SatScan:	No significant clusters found

The most likely period where significant clustering of lung cancer occurred (based on two statistical methods) appears to have been during 1993-1997. However, the year responsible for driving the significant outcome for this period was 1997. One-third of the lung cancer cases occurred during this year (Table 2). The RIF was the only statistical method that found period 1988-1992 as significantly elevated. This period may be considered a statistical artifact.

In reviewing the scientific literature the latency period for developing lung cancer is quite long, ranging from 15 to 20, or even 30 or more years from first exposure. If this latency period is applied to initial exposures to TCDD from the WES, which began operations in 1988, the length of time from initial exposures to TCDD to the development of lung cancers (significant increases) in Layton is significantly short, which is not consistent with what is known about the latency period for lung cancer. However, it must also be mentioned that while lung cancer has

been well documented as attributed to cigarette smoking (which has a similar latency period), less attention has been focused on TCDD-related lung cancers and latency period.

Lung cancer rates in Utah are among the lowest in the country. It is estimated that over 85% of all cancer deaths are attributed to cigarette smoking. Although cigarette smoking accounts for the majority of lung cancer cases, other factors have also been implicated in the etiology of lung cancer, such as occupational exposure to asbestos (UCR 2000 and Shottenfeld and Fraumeni 1996).

In a respiratory cancer study of 5,172 workers exposed to TCDD when working for 12 companies in the U.S. Fingerhut et al (l991), from the National Institute for Occupational Safety and Health, found an excess (non-significant) of cancer mortality among the cohort of workers exposed to TCDD. However, the study design could not exclude the possible contribution of factors such as smoking and occupational exposure to other chemicals. Therefore, the study was inconclusive.

The EPA estimates that the proportion of lung cancers due to air contaminants is about 1% of all lung cancer deaths per year in the U.S. population with a small proportion due to occupational exposures (EPA 1990).

This investigation could not determine the cause for the significant increase in lung cancer in the selected census tracts during period 1993-1997 and more specifically during year 1997. The rates observed may be due to normal variation in lung cancer rates over time.

In 2002, the Utah Cancer Registry published the "Incidence Rates for the Top Ten Cancers 1991-2000: by County." In the publication Davis County is listed as the only county or district in which brain cancer is ranked 10^{th} for both females and males and exceeds the rates of Utah and the U.S. for both sexes during the same time period. The only other county or district that includes brain cancer in the top 10 cancers is Tooele, but only for females. Lung cancer was in the top ten cancers for both females and males in all the counties and districts (UCR, 2002).

Interpretation of Results

Interpretation of all results should be approached cautiously due to the small number of cases diagnosed in any of the years or periods evaluated. A small number of cases can be deceptive or statistically problematic for drawing certain conclusions or inferences due to the large fluctuations that occur in the cancer rates during a lengthy time period.

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 (and with some cancers the latency period), then it may provide further evidence that the elevated rates are not due to random variation.

Cancer Incidence

There has been a steady rise in the overall death rate from cancer in the U.S. in the past 50 years. One of the major causes of this rise is the increase in lung cancer, which is strongly associated with increases in smoking, particularly among females (NCI 2003). Another major factor for the rise in cancer rates is that individuals live longer. In 1900 people on average lived to about 50 years of age. Medical science has extended that human life span by over 50%, to an average age of about 77. Scientists believe that mutations (or abnormal changes) to our body's cells are a primary cause of cancer. The increase in our life span has allowed significantly more chances of these mutations to occur (MDCH 2000). In addition, more people are overweight and obese, and physical activity is increasing only slightly (NCI 2003).

Although the incidence has increased, death rates for many types of cancer, other than lung cancer, have decreased by 15% between 1950 and 1991. This decrease is due to the improvements in the early detection and treatment of specific types of cancers such as breast, colon, and cervical (NCI 2003, MDCH 2000).

The average annual age-adjusted incidence rates for non-Hodgkin's lymphoma increased among both males and females in Utah from 1981 to 2000. These increments are consistent with trends in the U.S., where incidence rates more than doubled between 1950 and 1985. Nationwide, increases in the overall incidence rates were greatest in those over 65 years of age. Rates among those between the ages of 35-64 years increased to a lesser extent, while rates among those under 35 years of age remained relatively constant over the past 30 years (UCR 2000). The incidence of non-Hodgkin lymphoma as well as cancers of the breast and lung in women, liver in men and women, and melanoma are rising (NCI 2003).

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).

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 what causes the cancer remains 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 (Shottenfeld and Fraumeni, 1996)

In 1996, the National Cancer Institute reported that the frequency (incidence rate) for cancer of all types in children increased 10% between 1973 and 1991. This means that 10% more new cases of cancers (per million children) were found in 1991 than in 1973. During this period, brain cancer and soft tissue sarcoma each increased more than 25% (NCI 1996).

Cancer Epidemiology

Cancer is a name applied to many diseases with many different causes. Cancer is 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). Statistically, 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 and which cases are due to the background risk factors or genetic factors present in the general population. 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. Only a relatively small percentage of cancers can be attributed to environmental factors (Klaassen 1996).

The following are risk factors associated with the etiology of the cancers of concern: brain, lung, NHL, lymphocytic leukemia (acute and chronic), and soft tissue.

Brain

In the U.S., 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 one year after diagnosis. Brain cancer is listed as the 10th most common type of death from cancer (Shottenfeld and Fraumeni 1996). Brain tumors account for over 90% of all cancers in the central nervous system (UCR 2000). 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 (Shottenfeld and Fraumeni 1996).

Other physical, chemical, and infectious agents suspected of being risk factors have not yet been established as etiologically relevant. Factors and chemical exposures associated or 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 (Shottenfeld and Fraumeni 1996). Brain cancer may also be connected with breathing vinyl chloride over long periods (ATSDR 1997).

Lung & Bronchus

Smoking is by far the leading risk factor for developing lung and bronchus cancer. Passive smoking is also a risk factor. Exposure to radon and asbestos are also factors that lead to the development of lung cancer, however, smoking in addition to these exposures greatly increases the cancer causing effects of asbestos and radon (Archer et al 1973 and Shottenfeld and Fraumeni, 1996). Other risk factors implicated in lung and bronchus cancer are exposure to gas, nickel, polycyclic hydrocarbons, chromium (Shottenfeld and Fraumeni 1996), and chlormethyl ethers (Gowers et al 1993). And as mentioned, risk increases when exposure to these contaminants occurs in conjunction with cigarette smoking.

The risk of lung cancer (mesothelioma and asbestosis) has increased in various asbestos industries and occupations that include mining (arsenic, asbestos and coal) (Ames et al 1983, McDonald and McDonald 1987, and Taylor et al 1989) and uranium milling (UCR 2002), textile production, friction products, and insulation products Schottenfeld & Fraumeni, 1996). A high risk of lung cancer was also reported in workers exposed to bis(chloromethyl)ether (BCME). Occupational lung cancer may also result from exposure to inorganic arsenic compounds (insecticides, pesticides, and smelters). However, 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). 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 an excess of lung cancer was observed (Blair et al 1979). Some studies have suggested a possible association between respiratory cancer and exposures to TCDD (ATSDR 1998, NTP 2001).

An excess of lung cancer has also been reported among persons with high dietary intake of foods rich in fat and cholesterol. Tuberculosis has also been identified as a risk factor for lung and bronchus cancer (Zheng et al 1987).

Currently more than 2% of the population in Utah will be affected with lung and bronchus cancer in their lifetime (UCR 1996).

Non-Hodgkin's Lymphoma (NHL)

The cause of most of the cases of NHL remains unknown. What is known is that 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 (Schottenfeld and Fraumeni 1996).

Individuals at increased risk for NHL include those with primary immunodeficiency diseases, acquired immunodeficiency diseases, and patients who are immuno-suppressed subsequent to transplantation. Also, increased risk for NHL has been observed in patients with testicular cancer and Hodgkin's disease (Schottenfeld and Fraumeni 1996).

In general, occupations of somewhat higher social class are associated with a higher risk of the 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 wood workers, meat workers, and metal workers (Schottenfeld and Fraumeni 1996). Some studies have suggested a possible association between NHL and exposures to TCDD (ATSDR 1998, NTP 2001).

Lymphocytic Leukemia

Lymphocytic Leukemia is classified by two factors, how quickly the disease develops and progresses and what cells are affected. The disease is either classified as acute or chronic. Acute lymphocytic leukemia is the most common type seen in children, but can also seen in adults over 65. Chronic lymphocytic leukemia is most often seen in people over age 55, but can affect younger adults and is rarely seen in children (Schottenfeld and Fraumeni 1996). Some studies have suggested a possible association between lymphocytic leukemia and exposures to TCDD, primarily from herbicides (Burmeister et al 1982).

The following are risk factors associated with the etiology of acute and chronic lymphocytic leukemia:

Acute Lymphocytic Leukemia

*A*cute lymphocytic leukemia accounts for about 5% of the cancer in the 40 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 (herbicides and pesticides), paints, and butadiene. Exposures to styrene and ethylene oxide have also been implicated in the etiology of acute lymphocytic leukemia (Shottenfeld and Fraumeni, 1996). Childhood leukemia has been associated with pregnancy-related diagnostic X-ray exposure. Children with Down's Syndrome or other abnormal chromosome condition are at increased risk of developing acute lymphocytic leukemia (NCI, 1996).

Chronic Lymphocytic Leukemia

Chronic lymphocytic leukemia is a disease of later life, predominantly in the elderly. It is more common in males than females, for unknown reasons. The etiology of chronic lymphocytic leukemia is almost entirely unknown (UCR, 1996). This disorder has not been convincingly linked to any myelotoxic agent and sufficient data rules out an association with ionizing radiation. This condition does have a reported association with butadiene, ethylene oxide, nonionizing radiation, herbicides, pesticides, asbestos, and solvents (Kipen, 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).

Soft Tissue

Soft tissue cancer is a general category that includes cancer occurring in muscle, heart, subcutaneous tissue, and other related tissues. Because this category includes a number of

different types of cancer, it is difficult to define the etiology that is associated with all cancers of the soft tissue. It is important to mention that not all risk factors have been established. What has been established is that they occur more frequently in children and young adults.

Occupations were workers have been exposed to phenoxyacetic acid in herbicides and chlorophenols in wood preservatives as well as workers exposed to vinyl chloride may have an increased risk of developing soft tissue cancer (Schottenfeld and Fraumeni 1996). Some studies have suggested a possible association between cancer of the soft-tissue and exposures to TCDD (ATSDR 1998, NTP 2001).

High doses of radiation have also 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. Also, certain inherited diseases such as Li-Fraumeni syndrome and von Recklinghausens's disease are associated with an increased risk in developing soft tissue sarcomas (NCI 2002).

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 Layton. However, between 1973 and 2003, the sample size for any cancer in persons between 0 and 18 years old was extremely small. Due to these 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.

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 of concern presents a problematic issue relative to exposure and latency. In addition, 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 by this investigation.

Factors such as latency or induction period, population migration, personal habits, race, diet, occupational exposures, and familial history make drawing a conclusion problematic. The lack of

adequate exposure information also limits one's ability to infer that a positive association between study area and health outcome was due to exposure, or to infer that the absence of an association was because exposure resulted in no adverse health effect. Therefore, it is important to note that in most cancer incidence investigations no exposure or potential cause is ever apparent or established (MMWR 1990).

The primary objective of a cancer incidence 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 and to determine if there is a plausible carcinogenic association to the contaminants of concern. This investigation should not be viewed as a tool to definitively identify a source (cause and effect) to the cancers that are associated or have a possible link to the chemical of concern. In addition, cancer incidence investigations that fail to explain why increases in specific cancers are occurring in a community should not be interpreted as supporting environmental pollution.

CONCLUSIONS

There was one completed exposure pathway (respirable dust inhalation) for TCDD that has existed since 1988 for residents living near the WES in Layton. Remediation was completed at the WES in 2001 that significantly reduced the levels of TCDD well below the required standards. Therefore, this pathway no longer poses a public health concern for exposure to TCDD.

The three statistical methods used by this study found some common periods in time where brain cancer rates were significantly elevated in Layton. These periods include: by SIR – single year (1997 and 1999) and period (1998-2003), by SatScan (1997-1999), and by the RIF (1998-2003). These periods of time range from 1997 through 2003. However, the period of time (from 1997-2003) in which brain cancer cases occurred more frequently in Layton, and is primarily responsible for the statistically significant elevations in the incidence of brain cancer is 1997-1999. The other (most likely) period where significant clustering of brain cancer occurred appears to have been during 1988-1992.

This investigation found no evidence in the scientific literature between human exposure to TCDD and the development of brain cancer. In addition, this investigation could not determine why the rates for brain cancer during 1997-1999 were statistically significantly elevated in the area surrounding the WES.

The most likely period and year where significant clustering of lung and bronchus cancer occurred appears to have been during 1993-1997, and more specifically during year 1997 in Layton. However, the latency period for the development of lung and bronchus cancer from initial exposures to TCDD from the WES is inconsistent with the scientific literature. Therefore, the rates observed may be attributed to normal variation in lung and bronchus cancer rates over time.

RECOMMENDATIONS

The EEP recommends that a follow-up statistical review of cancers be conducted in the census tracts that surround WES when three additional years of cancer data has been compiled by the Utah Cancer Registry.

The EEP also recommends that the communities living in census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06 be provided or have access to a copy of this health consultation via the Utah Department of Health Website and through the Davis County Health Department Public Information Office.

PUBLIC HEALTH ACTION PLAN

The EEP, in coordination with the Davis County Health Department will conduct a follow-up statistical review of cancers in the census tracts that surround WES when three additional years of cancer data has been compiled by the Utah Cancer Registry.

The EEP will provide the communities living in census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06 with access information to this health consultation via the Utah Department of Health Website and the Davis County Health Department Public Information Office.

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CERTIFICATION

This Health Consultation, An Investigation of Cancer Incidence in Census Tracts - 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06, in Layton, 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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APPENDIX A – WES DIOXIN COMPLIANCE HISTORY

Dioxin Compliance History of Wasatch Energy Systems (WES), 1983 - 2001
Monitored by the Utah Department of Environmental Quality, Division of Air Quality (DAQ).

September 10, 1996
On September 10, 1996, the Executive Secretary issued an Approval Order (AO) to WES in accordance with Utah Administrative Code (UAC) R307-401. Condition 7 of that AO limits dioxin/furan emissions from each combustor unit to 360 nanograms (ng = 1/1,000,000,000 of a gram) per dry standard cubic meter (ng/dscm) adjusted to 7% oxygen (@ 7% O_2).
(Source: DAQ Document 1996. "Approval Order - September 10, 1996", http://www.airquality.utah.gov/Permits/wes/E-850-96.pdf)

September 15-17, 1998
WES performs annual stack testing to demonstrate compliance with AO emission limits. Both Units A & B exceed dioxin/furan limit of 360 ng/dscm. (Unit A = 624 ng/dscm, Unit B = 685 ng/dscm).
(Source: DAQ Document, 2000. "Compliance History of Wasatch Energy Systems (1983 through November, 2000)", http://www.airquality.utah.gov/Permits/wes/HISTORY.pdf)

July 9, 1999
Notice of Violation (NOV) issued for failing to comply with condition 7 and 8 of the AO dated 9/10/96, for exceeding the dioxin/furan emission limit for units A and B. Results for unit A were 624 ng/dscm and unit B were 685 ng/dscm. NOV - Condition 8 for failure to conduct testing, which constitutes an annual compliance demonstration.
(Source: DAQ Document, 2000. "Compliance History of Wasatch Energy Systems (1983 through November, 2000)", http://www.airquality.utah.gov/Permits/wes/HISTORY.pdf)

March 2, 2000
NOV issued for exceeding the dioxin/furan emission limits set in condition 7 of the AO dated 9/10/96. The results for unit A were 1101.1 ng/dscm for dioxin/furan.
(Source: DAQ Document, 2000. "Compliance History of Wasatch Energy Systems (1983 through November, 2000)", http://www.airquality.utah.gov/Permits/wes/HISTORY.pdf)

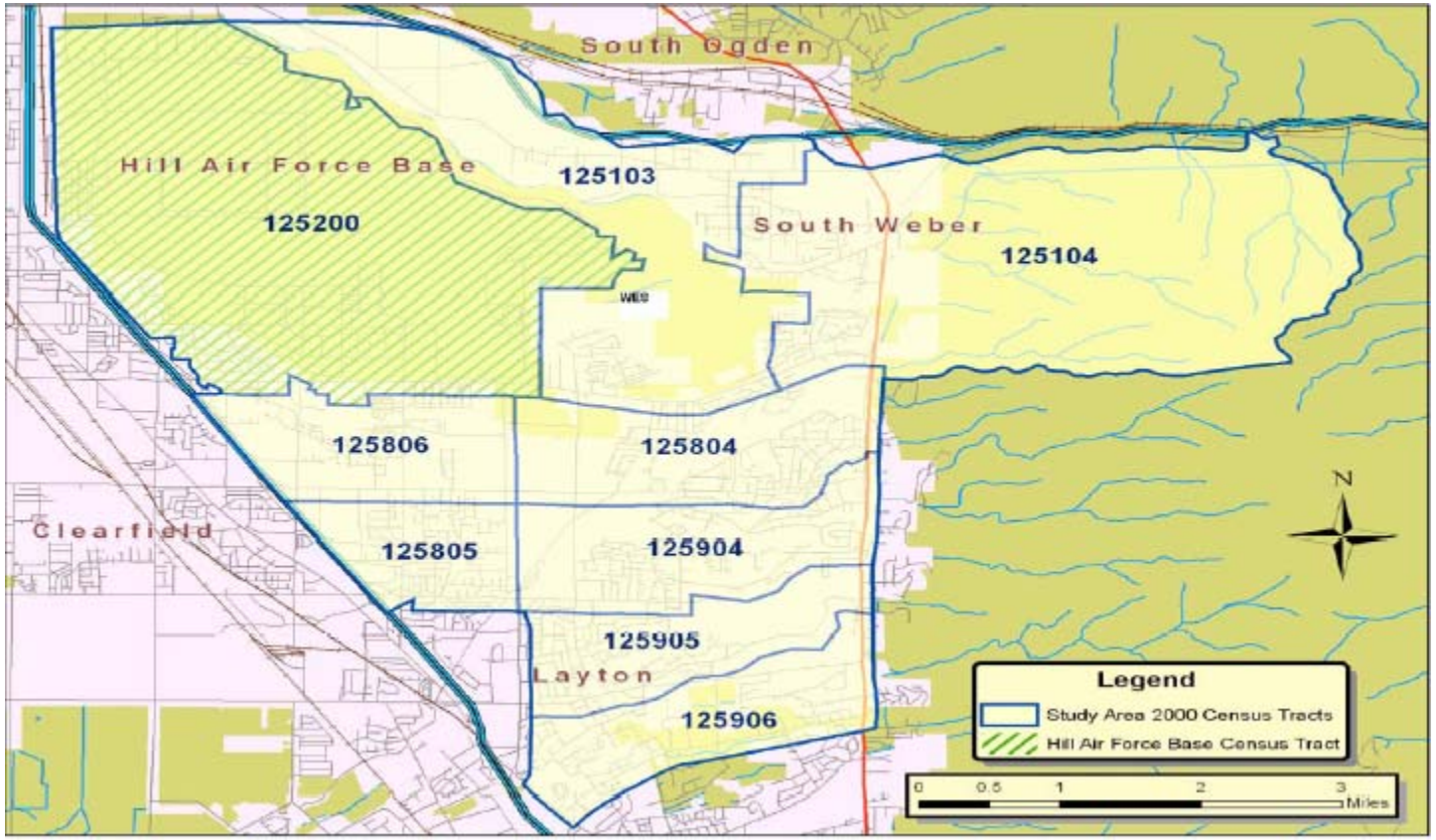
October 10-14, 2000
NOV issued for exceeding the dioxin/furan emission limits set in condition 7 of the AO dated 9/10/96. The test report indicated that dioxin/furan emissions (from Unit A) averaged 555.9 ng/dscm for the test period. Wasatch Energy Systems (WES) did not demonstrate that Unit A had been brought back into compliance with the dioxin/furan emission limit until a subsequent test was performed on January 18-22, 2001. The report for the January 18-22, 2001 test indicates that the average dioxin/furan emissions for Runs 1, 2, and 4 of the Unit A dioxin/furan test were 273.2 nanograms per dry standard cubic meter, adjusted to 7 percent oxygen (ng/dscm @ 7% O2). Using the oxygen concentrations determined from the test grab samples, DAQ calculates that the average dioxin/furan emissions for Runs 1, 2, and 4 of the January 18-22, 2001 Unit A dioxin/furan test were 298.3 ng/dscm @ 7%O2. Run 3 of the January 18-22,2001

Unit A dioxin/furan test was not included in the dioxin/furan test results because two different meter boxes were used during that run.
(Source: DAQ Document, Notice of Violation - April 9, 2001 (October 2000 Dioxin, CO and Reporting Violations) http://www.airquality.utah.gov/Permits/wes/C-016-2001.pdf)

March 6-9 2001
NOV issued for exceeding the dioxin/furan emission limits set in condition 7 of the AO dated 9/10/96. The test report indicated that dioxin/furan emissions (from Unit A) averaged 365.4 ng/dscm for the test period.
(Source: DAQ Document, Notice of Violation - June 14, 2001 (March 2001 Dioxin Violation) http://www.airquality.utah.gov/Permits/wes/C-884-2001.pdf)

APPENDIX B - STUDY AREA

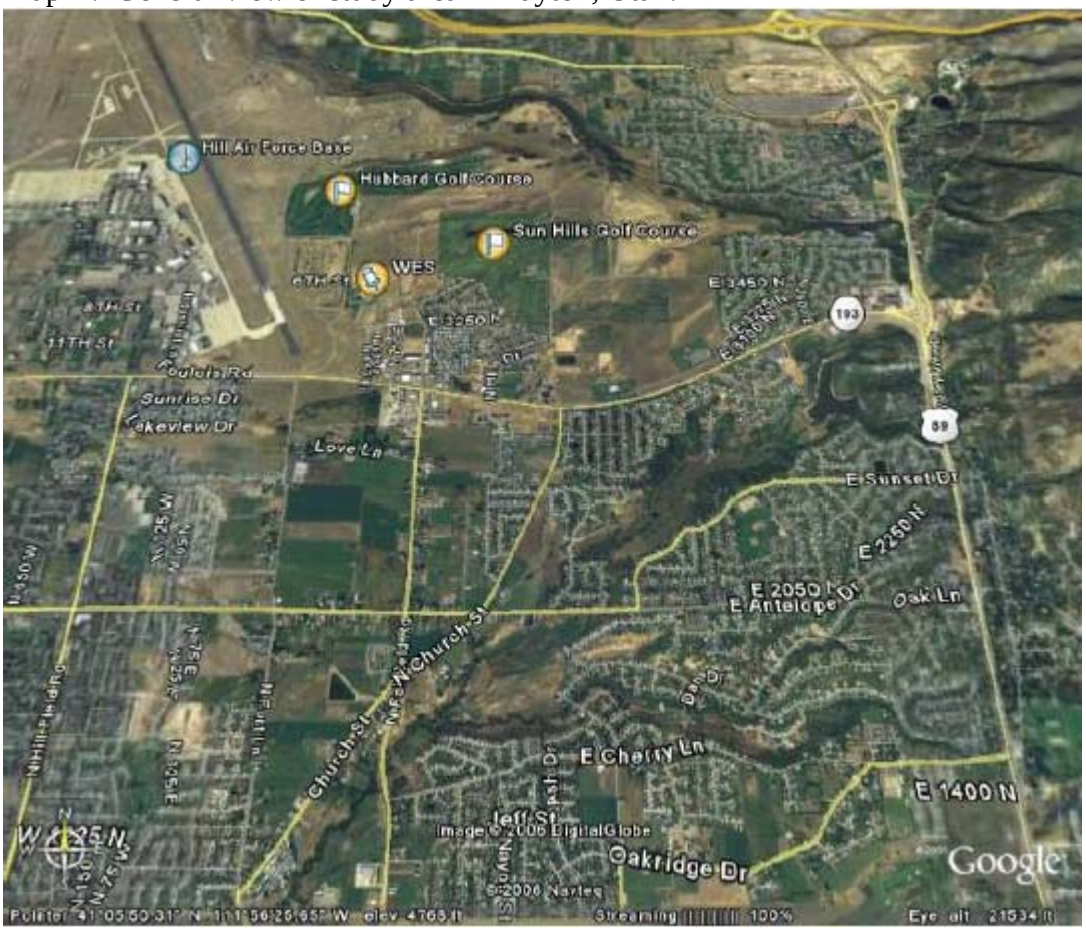
Map A. The wind blows most often in a westward direction.



Wind Source: Hill Air Force Base, Meteorological Data, National Weather Service, 2001.
Census tract 125200 identifies the Hill Air Force Base and is not part of study area.

STUDY AREA

Map B. General view of study area in Layton, Utah.



APPENDIX C – 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 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, this does not prove that the cancer rates are elevated.

Generally, as the sample size (or the number of people in a 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, Layton). 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 Rates and Ratios

Direct Standardized Cancer Incidence Rate: The incidence of cancer by type in the exposed population (the combined population of all of the exposed census tracts) were standardized to the incidence of cancer in the unexposed population using a direct standardization method.

$$X_S = \left(\sum_{a,s,r,f} \left(X_{E:a,s,r,f} \frac{P_{U:a,s,r,f}}{P_{E:a,s,r,f}} \right) \right) \left(\frac{P_E}{P_U} \right)$$

$$X_E = \left(\sum_{a,s,r,f} \left(X_{U:a,s,r,f} \right) \right) \left(\frac{P_E}{P_U} \right)$$

Where: X_S is the standardized cancer incidence count for the exposed population for the study period, standardized to the unexposed population and re-proportioned to the exposed population . This is the standardized observed count of cancer incidence.

X_E is the cancer incidence count for the unexposed population re-proportioned to the exposed population . Since this method is standardizing to the unexposed population, the cancer incidence count does not need to be adjusted. This is the standardized expected count of cancer incidence.

Standardized Incidence Ratio: Standardized incidence ratio (SIR) was calculated for each cancer type for the study period from the standardized incidence count for the exposed population and the proportional incidence count for the unexposed population (the expected incidence count for the exposed population). The lower and upper confidence limits were obtained using Byar's method (Berslow and Day 1987, Regidor et. al. 1993).

$$SIR = \frac{X_S}{X_E}$$

$$\underline{SIR} = \frac{X_S}{X_E}\left(1 - \left(\frac{1}{9X_S}\right) - \left(\frac{Z_\alpha}{3\sqrt{X_S}}\right)\right)^3$$

$$\overline{SIR} = \frac{(X_S + 1)}{X_E}\left(1 - \left(\frac{1}{9(X_S + 1)}\right) + \left(\frac{Z_\alpha}{3\sqrt{X_S + 1}}\right)\right)^3$$

Where: Z_α for the 95% confidence interval is 1.96.

APPENDIX D - Tables

Presented are the number of observed cases, the number of expected cases, the Standardized Incidence Ratios, Rates, and 95% confidence intervals for cancer in census tracts - 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06 from 1973 – 2001 (2000 Census) for each of the periods analyzed. The state of Utah was selected as the comparison population. Tables 1 – 5 present the incidence of cancer from 1973-2003 by single years. Tables 6 - 10 present the incidence of cancer from 1973-2003 by five five-year periods and one six year period. Cancers presented are: *Brain, Lung and Bronchus, Non-Hodgkin's lymphoma (NHL), Lymphocytic Leukemia, and Soft Tissue.* Table 11 presents the SatScan results for *brain cancer.* Tables 12 and 13 present the results of the Rapid Inquiry Facility **(**RIF) for *brain cancer, and lung* and *bronchus cancer.*

The criteria established for determining a statistical significant difference in observed cases involved two statistical methods (Single years and Periods, and RIF):
1. A Standardized Incidence Ratio greater than one (1.0).
2. A 95% confidence interval with limits that do not include one (1.0).

-Variation in Standard Incidence Ratios may exist due to rounding effect.
- SIR means a Standardized Incidence Ratio.
-C.I.LL means Confidence Interval Lower Limit.
-C.I.UL means Confidence Interval Upper Limit.

SatScan criteria for statistical significance:

- The number of cases after adjustment were 5 or greater;
- The SIR is greater than 1.0;
- The log-likelihood p-value was less than 0.01.

SINGLE YEAR PERIODS 1973 – 2003

Table 1. Annual age-adjusted **brain** cancer incidence rates by single-year periods comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973	0.00	3.95	0	0.32	0.00	-
1974	5.56	6.18	≤3	0.55	1.83	0.02, 10.19
1975	0.00	4.95	0	0.48	0.00	-
1976	0.00	5.93	0	0.59	0.00	-
1977	7.62	4.81	≤3	0.56	3.55	0.40, 12.80
1978	0.00	5.11	0	0.51	0.00	-
1979	0.00	6.20	0	0.68	0.00	-
1980	3.94	6.05	≤3	0.73	1.37	0.02, 7.64
1981	2.50	6.25	≤3	0.82	1.21	0.02, 6.76
1982	0.00	5.63	0	0.81	0.00	-
1983	9.80	5.24	≤3	0.81	2.47	0.28, 8.91
1984	4.73	5.68	≤3	0.94	1.06	0.01, 5.92
1985	14.54	5.70	***4****	1.07	3.74	1.01, 9.57
1986	2.71	5.86	≤3	1.11	0.90	0.01, 5.03
1987	0.00	7.85	0	1.44	0.00	-
1988	21.48	6.39	***5****	1.33	3.75	1.21, 8.75
1989	6.52	6.56	≤3	1.30	2.32	0.47, 6.77
1990	2.29	7.03	≤3	1.58	0.63	0.01, 3.51
1991	12.55	5.61	4	1.37	2.92	0.78, 7.47
1992	3.66	6.81	≤3	1.83	0.55	0.01, 3.03
1993	3.27	6.12	≤3	1.69	0.59	0.01, 3.30
1994	8.97	4.96	≤3	1.61	1.87	0.38, 5.46
1995	2.87	7.20	≤3	2.02	0.49	0.01, 2.75
1996	1.77	5.17	≤3	1.62	0.62	0.01, 3.43
1997	41.50	6.34	***8****	2.14	3.73	1.61, 7.36,
1998	19.68	6.87	5	2.15	2.33	0.75, 5.43
1999	18.82	5.48	***6****	1.97	3.04	1.11, 6.61
2000	9.03	6.30	≤3	2.03	1.48	0.30, 4.31
2001	5.11	5.61	≤3	2.16	1.39	0.28, 4.05
2002	5.10	6.02	≤3	2.28	1.31	0.26, 3.84
2003	4.70	5.44	≤3	2.31	0.86	0.10, 3.12

[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, 2003.

Table 2. Annual age-adjusted **lung and bronchus** cancer incidence rates by single-year periods comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973	14.86	31.42	≤3	1.04	0.96	0.01, 5.36
1974	14.98	27.04	4	0.99	4.02	1.00, 10.30
1975	12.49	27.83	≤3	1.30	0.77	0.01, 4.27
1976	10.00	31.17	≤3	1.41	0.71	0.01, 3.93
1977	13.88	26.80	≤3	1.26	1.58	0.18, 5.72
1978	10.58	30.70	≤3	1.53	0.65	0.01, 3.63
1979	59.61	30.62	≤3	1.79	1.12	0.13, 4.03
1980	0.00	28.42	0	1.98	0.00	-
1981	23.62	30.93	≤3	2.12	0.94	0.11, 3.40
1982	21.70	30.01	≤3	2.09	1.44	0.29, 4.19
1983	52.99	32.74	6	2.70	2.22	0.81, 4.83
1984	6.30	30.21	≤3	2.83	0.35	0.00, 1.96
1985	136.79	29.93	≤3	2.92	0.68	0.08, 2.47
1986	62.32	29.88	5	3.21	1.56	0.50, 3.64
1987	41.60	29.41	5	3.34	1.50	0.48, 3.49
1988	6.37	27.51	≤3	3.41	0.29	0.00, 1.63
1989	46.30	23.94	5	2.88	1.74	0.56, 4.05
1990	25.56	28.14	≤3	3.95	0.76	0.15, 2.22
1991	63.92	28.48	9	4.09	2.20	1.00, 4.18
1992	31.05	29.29	7	4.46	1.57	0.63, 3.24
1993	37.56	30.15	7	4.77	1.47	0.59, 3.03
1994	17.59	28.90	4	5.04	0.79	0.21, 2.03
1995	31.19	27.28	6	4.76	1.26	0.46, 2.74
1996	33.52	26.47	7	5.23	1.34	0.54, 2.76
1997	61.19	26.77	***12****	5.37	2.23	1.15, 3.90
1998	38.89	28.28	10	5.87	1.70	0.82, 3.13
1999	15.64	28.22	4	6.34	0.63	0.17, 1.61
2000	43.46	23.90	10	5.45	1.84	0.88, 3.38
2001	26.48	26.40	7	6.30	1.11	0.45, 2.29
2002	26.10	23.26	7	5.86	1.19	0.48, 2.46
2003	25.19	25.69	8	6.88	1.16	0.50, 2.29

[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, 2003.

Table 3. Annual age-adjusted **Non-Hodgkin's lymphoma (NHL)** incidence rates by single-year periods comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973	0.00	10.11	0	0.46	0.00	-
1974	0.00	9.51	0	0.47	0.00	-
1975	10.23	8.09	≤3	0.52	1.93	0.03, 10.74
1976	20.40	9.96	≤3	0.73	2.75	0.31, 9.94
1977	0.00	11.77	0	0.64	0.00	-
1978	0.00	9.38	0	0.74	0.00	-
1979	3.64	10.53	≤3	0.67	1.49	0.02, 8.29
1980	7.28	9.53	≤3	0.76	1.32	0.02, 7.33
1981	13.90	13.32	≤3	0.99	2.01	0.23, 7.26
1982	0.00	10.08	0	0.74	0.00	-
1983	26.17	10.67	≤3	0.95	2.11	0.24, 7.64
1984	9.41	10.75	≤3	1.25	0.80	0.01, 4.47
1985	5.25	10.70	≤3	1.25	0.80	0.01, 4.45
1986	24.23	13.30	≤3	1.75	0.57	0.01, 3.19
1987	9.62	11.63	≤3	1.54	1.30	0.15, 4.69
1988	18.83	13.61	≤3	1.87	1.60	0.32, 4.68
1989	5.54	13.18	≤3	2.08	0.48	0.01, 2.68
1990	9.68	13.62	≤3	2.08	0.96	0.11, 3.47
1991	12.84	14.35	≤3	2.43	0.82	0.09, 2.98
1992	10.60	13.39	≤3	2.61	1.15	0.23, 3.36
1993	8.62	14.83	≤3	3.00	1.00	0.20, 2.92
1994	14.01	14.78	≤3	3.04	0.99	0.20, 2.89
1995	10.56	14.51	≤3	3.32	0.60	0.07, 2.18
1996	18.66	13.64	≤3	3.19	0.94	0.19, 2.75
1997	10.09	14.75	≤3	3.51	0.85	0.17, 2.50
1998	13.40	16.26	≤3	4.20	0.72	0.14, 2.09
1999	14.36	15.77	4	4.02	1.00	0.27, 2.55
2000	8.93	15.14	≤3	4.00	0.50	0.06, 1.81
2001	20.57	16.28	6	4.53	1.33	0.48, 2.88
2002	11.59	15.97	5	4.61	1.09	0.35, 2.53
2003	26.97	16.26	7	5.12	1.37	0.55, 2.82

[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

Data Source: Utah Cancer Registry, 2003.

Table 4. Annual age-adjusted **lymphocytic leukemia** incidence rates by single-year periods comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973	10.40	5.28	≤3	0.21	4.84	0.06, 26.93
1974	10.86	5.35	≤3	0.30	3.35	0.04, 18.62
1975	3.88	3.78	≤3	0.36	2.81	0.04, 15.65
1976	0.00	4.82	0	0.37	0.00	-
1977	67.65	5.20	≤3	0.38	5.22	0.59, 18.83
1978	10.58	5.47	≤3	0.46	2.16	0.03, 12.00
1979	2.97	6.43	≤3	0.49	2.03	0.03, 11.31
1980	2.57	4.88	≤3	0.41	2.47	0.03, 13.72
1981	0.00	6.99	0	0.66	0.00	-
1982	9.18	4.79	≤3	0.46	2.16	0.03, 12.01
1983	28.72	6.76	≤3	0.69	1.45	0.02, 8.09
1984	0.00	4.64	0	0.73	0.00	-
1985	8.16	4.65	≤3	0.91	2.21	0.25, 7.98
1986	2.19	4.91	≤3	0.77	1.30	0.02, 7.21
1987	0.00	5.72	0	0.93	0.00	-
1988	2.18	4.41	≤3	0.78	1.29	0.02, 7.15
1989	2.08	5.65	≤3	1.03	0.97	0.01, 5.38
1990	2.29	4.97	≤3	1.03	0.97	0.01, 5.38
1991	1.79	4.65	≤3	0.87	1.14	0.01, 6.36
1992	0.00	4.20	0	1.19	0.00	-
1993	1.80	5.03	≤3	1.04	0.96	0.01, 5.36
1994	41.68	5.08	≤3	1.15	2.61	0.52, 7.61
1995	2.20	5.26	≤3	1.28	0.78	0.01, 4.36
1996	3.43	4.65	≤3	1.24	1.61	0.18, 5.80
1997	0.00	4.57	0	1.21	0.00	-
1998	1.65	4.11	≤3	1.28	0.78	0.01, 4.36
1999	10.03	3.76	≤3	1.21	2.48	0.50, 7.25
2000	8.52	5.45	≤3	1.72	1.75	0.35, 5.10
2001	2.09	5.22	≤3	1.49	0.67	0.01, 3.74
2002	0.00	5.36	0	1.84	0.00	-
2003	3.03	5.07	≤3	1.71	1.17	0.13, 4.23

[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, 2003.

Table 5. Annual age-adjusted **soft tissue** incidence by single-year periods comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973	0.00	2.55	0	0.17	0.00	-
1974	0.00	2.17	0	0.12	0.00	-
1975	3.86	2.09	≤3	0.12	8.13	0.11, 45.26
1976	0.00	2.02	0	0.19	0.00	-
1977	0.00	2.32	0	0.18	0.00	-
1978	0.00	1.11	0	0.14	0.00	-
1979	0.00	1.23	0	0.14	0.00	-
1980	0.00	1.78	0	0.28	0.00	-
1981	0.00	1.76	0	0.27	0.00	-
1982	0.00	1.72	0	0.26	0.00	-
1983	36.92	3.51	≤3	0.46	2.17	0.03, 12.07
1984	0.00	2.59	0	0.33	0.00	-
1985	3.05	2.38	≤3	0.36	2.80	0.04, 15.59
1986	0.00	1.55	0	0.32	0.00	-
1987	0.00	2.10	0	0.33	0.00	-
1988	0.00	1.77	0	0.31	0.00	-
1989	3.15	1.51	≤3	0.37	2.70	0.04, 15.04
1990	0.00	1.64	0	0.41	0.00	-
1991	0.00	2.12	0	0.49	0.00	-
1992	1.84	2.30	≤3	0.50	2.00	0.03, 11.15
1993	0.00	2.05	0	0.49	0.00	-
1994	0.00	2.32	0	0.57	0.00	-
1995	5.07	3.53	≤3	0.99	2.01	0.23, 7.26
1996	4.27	2.43	≤3	0.74	2.69	0.30, 9.72
1997	0.00	2.56	0	0.79	0.00	-
1998	6.18	2.91	3	0.75	3.98	0.80, 11.61
1999	1.60	3.55	≤3	1.30	0.77	0.01, 4.28
2000	9.09	2.09	3	0.80	3.77	0.76, 11.00
2001	0.00	2.33	0	0.85	0.00	-
2002	4.81	2.17	≤3	0.80	1.25	0.02, 6.94
2003	4.12	2.79	≤3	1.07	1.86	0.21, 6.72

[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, 2003.

Table 6. Age-adjusted **brain** cancer incidence rates by five-year periods comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973-1977	2.64	5.16	≤3	2.51	1.20	0.24, 3.50
1978-1982	1.29	5.85	≤3	3.55	0.56	0.06, 2.03
1983-1987	6.35	6.07	8	5.36	1.49	0.64, 2.94
1988-1992	9.30	6.48	***14****	7.42	1.89	1.03, 3.17
1993-1997	11.68	5.96	14	9.08	1.54	0.84, 2.59
1998-2003	10.40	5.95	***22****	12.92	1.70	1.07, 2.58
[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, 2003.						

Table 7. Age-adjusted **lung and bronchus** cancer incidence rates by five-year periods and one six-year period comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973-1977	18.24	28.85	9	6.01	1.50	0.68, 2.84
1978-1982	23.10	30.14	8	9.52	0.84	0.36, 1.66
1983-1987	60.00	30.43	19	15.01	1.27	0.76, 1.98
1988-1992	34.64	27.47	25	18.80	1.33	0.86, 1.96
1993-1997	36.21	27.91	*36**	25.17	1.43	1.01, 1.98
1998-2003	29.30	25.96	46	36.70	1.25	0.92, 1.67
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
* Statistically significant increase (p = <0.05) from the expected number of cases.						
Data Source: Utah Cancer Registry, 2003.						

Table 8. Age-adjusted **Non-Hodgkin's lymphoma (NHL)** incidence rates by five-year periods and one six-year period comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973-1977	6.13	9.89	≤3	2.81	1.07	0.21, 3.12
1978-1982	4.97	10.57	4	3.91	1.02	0.28, 2.62
1983-1987	14.94	11.41	7	6.73	1.04	0.42, 2.14
1988-1992	11.50	13.63	11	11.06	0.99	0.50, 1.78
1993-1997	12.39	14.50	14	16.05	0.87	0.48, 1.46
1998-2003	15.97	15.95	27	26.47	1.02	0.67, 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, 2003.						

Table 9. Age-adjusted **lymphocytic leukemia** incidence rates by five-year periods and one six-year period comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973-1977	1.56	4.88	5	1.61	3.10	0.99, 7.24
1978-1982	5.06	5.71	4	2.48	1.61	0.43, 4.12
1983-1987	7.81	5.33	4	4.03	0.99	0.27, 2.54
1988-1992	1.67	4.78	4	4.91	0.81	0.22, 2.09
1993-1997	9.82	4.92	7	5.93	1.18	0.47, 2.43
1998-2003	4.22	4.83	10	9.24	1.08	0.52, 1.99
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
Data Source: Utah Cancer Registry, 2003.						

Table 10. Age-adjusted **soft tissue** incidence rates by five-year periods and one six-year period comparing Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) to Utah – 1973-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1973-1977	0.77	2.23	≤3	0.79	1.27	0.02, 7.06
1978-1982	0.00	1.52	0	1.10	0.00	-
1983-1987	8.00	2.42	≤3	1.79	1.11	0.13, 4.02
1988-1992	1.00	1.87	≤3	2.08	0.96	0.11, 3.47
1993-1997	1.87	2.58	4	3.59	1.11	0.30, 2.85
1998-2003	4.30	2.64	10	5.58	1.79	0.86, 3.30
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
[3] Observed cases are presented as ≤3when cases are less than or equal to three in order to protect the confidentiality of the cases						
Data Source: Utah Cancer Registry, 2003.						

Table 11. **SatScan** results for **cancer of the brain** by the most likely period of time where significant clustering occurred in Layton (census tracts 1251.03, 1258.04, 1259.04, and 1258.05), 1997-1999.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	P-Value[2]
1997-1999	24.40	N/A	*19**	3.84	4.69	0.009
[1] Standardized Incidence Ratio						
[2] P-value – p = <0.01						
* Statistically significant increase (p = <0.05) from the expected number of cases.						
Data Source: Utah Cancer Registry, 2003.						
Number (N) of cases per year – 1997 N=8, 1998 N=5, and 1999 N=6.						

Table 12. **Rapid Inquiry Facility (RIF)** rates for **cancer of the brain** by five-year periods and one six-year in Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) from 1988-2003.

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1988-1992	4.00	6.48	*14**	7.17	1.95	1.03, 3.17
1993-1997	7.78	5.96	14	9.15	1.53	0.84, 2.57
1998-2003	7.83	5.95	*22**	12.90	1.88	1.15, 2.91
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
* Statistically significant increase from the expected number of cases.						
Data Source: Utah Cancer Registry, 2003.						

Table 13. **Rapid Inquiry Facility (RIF)** rates for **lung and bronchus cancer** by five-year periods and one six-year period in Layton (census tracts 1251.03, 1251.04, 1258.04, 1258.05, 1258.06, 1259.04, 1259.05, and 1259.06) from 1988-2003).

Time Period	Layton Rate per 100,000	Utah Rate per 100,000	Layton Observed number cases[3]	Layton Expected number cases	SIR[1]	95% CI[2] LL UL
1988-1992	23.06	27.47	*25**	15.68	1.59	1.03, 2.35
1993-1997	24.82	27.91	*36**	23.03	1.56	1.09, 2.16
1998-2003	20.02	25.96	*46*	33.99	1.35	0.99, 1.81
[1] Standardized Incidence Ratio						
[2] 95% Confidence interval						
* Statistically significant increase from the expected number of cases.						
Data Source: Utah Cancer Registry, 2003.						

APPENDIX E

International Classification of Diseases for Oncology – 3rd Edition

Listed are the cancers and International Classification of Diseases for Oncology (3rd edition) codes that were used to select the cancers included in this study. Cancer types that are starred (*) have been associated with the contaminant of concern.

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

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)