Health Consultation

Cancer Investigation for Three Neighborhoods Surrounding
J.H. Baxter & Company and Other Industrial Sites

EUGENE, OREGON

EPA FACILITY ID: ORD009032400

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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

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Prepared By:
Oregon Department of Human Services
Under Cooperative Agreement with the
The U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

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SUMMARY

The Environmental Health Assessment Program (EHAP), part of the Oregon Department of Human Services Public Health Division, developed this health consultation in response to a request from residents of the Bethel, River Road and Trainsong neighborhoods in northwest Eugene, Oregon. These densely populated neighborhoods border the J.H. Baxter wood treatment plant and are near several other industrial sites. Residents were concerned about the possible health impacts from contaminants released by these businesses.

While collecting community concerns about the J.H. Baxter site in 2003, EHAP was petitioned by community members living in this area to investigate the incidence of acute myeloid leukemia (AML) and brain cancer. Residents were concerned that there were excess rates of these two types of cancers, possibly caused by contaminants released from the wood treatment facility along with the other nearby industries.

EHAP prepared an initial report, released in September 2006, which reviewed cancer incidence rates in this area to determine if the number of cases of AML, brain, lung, and nasal cancers was higher than expected for the period of 1996 to 2003, the years for which data were available from the state cancer registry. Since the time of that initial report, additional data have become available, and this follow-up report includes data for the period of 1996-2004.

The cancer investigation focused on the rates of these cancers in the six census tracts that make up the Bethel (census tracts 26 and 43), River Road (census tracts 27, 28 and 41), and Trainsong (census tract 42) neighborhoods. Rates of AML and brain cancer were reviewed because these were the cancers residents thought were occurring at higher rates. Rates of lung and nasal cancers were added to the review because of the close proximity of the wood treatment plant and the association noted in the scientific literature between exposure to the wood preservative creosote and these cancers. In addition, EHAP also reviewed the rates of “all cancers” and “all other cancers” (cancers other than AML, brain, lung and nasal) in these neighborhoods.

There were no statistically significant elevations for brain and nasal cancers for the 1996-2004 period. However, cases of brain cancer in census tract 26 were significantly elevated for the years 1996-2002. The absence of cases in this census tract in 2003 and 2004 is somewhat reassuring, and suggest that there is no on-going cluster. Because of the small number of cases involved, EHAP was unable to determine whether the statistical results from 1996-2002 represents an actual increase in cancer rates. Therefore, EHAP will monitor the incidence of brain cancer in census tract 26 in 2005 and 2006.

There were statistically significant elevations in lung cancer cases in census tract 42 and the six census tracts overall for the years 1996-2004, and in census tract 26 for the years 1996-2003. Further investigation found a strong link to tobacco smoking among those affected. However, given that approximately half of the cancer cases had lived in these census tracts for at least 10 years, a common environmental exposure could be a contributor to lung cancer cases in these neighborhoods. Because many of the cases had more than one risk factor, investigators were unable to determine a cause for the significant elevations in these neighborhoods.

There were no significant elevations in the number of cases of AML over the entire study period (1996-2004). When the analysis was restricted to the period 2002-2004, the number of observed cases in census tract 43 was significantly above the number expected. Because this might represent the early development of a cluster, cases of AML in census tract 43 will be monitored using 2005 and 2006 data. However, because all observed AML cases had known risk factors for AML, including tobacco use and a history of chemotherapy, EHAP was unable to draw conclusions about the origin of the illnesses.

EHAP makes every attempt to thoroughly investigate the relationship between environmental contaminants and disease given the data and scientific means available. However, it is often impossible to link environmental exposures to disease because the needed environmental data do not exist. Cancer investigations are particularly difficult because most cancers have multiple risk factors or causes that can include a family history of cancer, occupational exposures, behavioral risk factors such as tobacco use and diet, and environmental exposures. Any investigation into the causes of a cancer case or cluster requires detailed case histories that include information on the above risk factors, as well as residential, occupational and medical histories. It often is difficult or impossible to obtain this needed information, particularly if the individuals are deceased or are too ill to provide the information. Therefore, while EHAP may be able to determine whether there are elevations of disease in a given area, a lack of data often limits researchers' ability to determine the causes of these elevations.

In conclusion, EHAP identified statistical elevations of AML and brain and lung cancers in the Bethel, River Road and Trainsong neighborhoods. It is likely, given the known risk factors for these cancers, that many of these cases arose from the effects of tobacco use. Because of limited information on individual case histories and environmental exposures, EHAP is unable to determine the role that environmental contaminants from a single or multiple sources might have played in these apparent clusters.

EHAP considers tobacco smoke to be an important environmental contaminant and a major risk factor for cancer, and recommends the implementation or expansion of tobacco prevention and cessation programs in these neighborhoods. EHAP also recommends the review of available air monitoring data to determine if they can be used to evaluate health risks related to environmental exposures in these neighborhoods.

PURPOSE AND HEALTH ISSUES

The Environmental Health Assessment Program, part of the Oregon Department of Human Services Public Health Division, prepared this health consultation to address whether certain types of cancer are elevated in the neighborhoods of Bethel, River Road, and Trainsong located in Northwest Eugene, Oregon. While collecting community concerns for the J.H. Baxter site in 2003, EHAP was petitioned by community members living in this area to investigate the incidence of acute myeloid leukemia (AML) and brain cancer.

In 2003, EHAP completed a health consultation for J.H. Baxter, which concluded that there were not enough data to evaluate whether contaminants being released from J.H. Baxter posed a public health risk. The document stated that although the low-level concentrations of

contaminants from J.H. Baxter were not likely to be associated with elevated cancer rates, an investigation should be conducted to address the residents' concerns [1]. EHAP recommended that the Oregon State Cancer Registry (OSCaR) and EHAP collaborate to complete this investigation. Residents expressed concern specifically about AML and brain cancer rates during a public meeting related to J.H. Baxter.

In 2004, OSCaR performed an initial investigation into the rates of AML and brain cancer in Northwest Eugene near J.H. Baxter. That investigation used data reported at the Zip Code level, and produced no evidence of increased rates for the cancers of concern. At that time, OSCaR was in the process of adding data to their database that allowed them to analyze the data for individual census tracts, which are smaller geographic areas than Zip Code areas. OSCaR and EHAP concluded that when the complete data set became available, another review of the data would be performed. This health consultation summarizes the results of the census tract-level cancer investigation performed by OSCaR in collaboration with the EHAP program.

The follow-up census tract-level cancer investigation began in the winter of 2005. The focus was on census tracts 26, 27, 28, 41, 42, and 43 because they make up the majority of the area in the Bethel, River Road, and Trainsong neighborhoods. In addition to AML and brain cancer, EHAP requested that OSCaR expand the investigation to include lung and nasal cancer because these cancers have been linked to exposure to creosote, which is used for wood treatment by J.H. Baxter.

In September 2006, EHAP released an initial health consultation which evaluated cancer data for the six census tracts from 1996-2003. Both the initial health consultation released in September 2006 and the present document focus on answering the specific question about cancer rates in these neighborhoods. This version of the health consultation uses additional data which have since become available and evaluates the data for the six census tracts for the period of 1996-2004. EHAP is aware that, in addition to cancer rates, residents in the three neighborhoods have expressed concerns about other potential health effects from exposure to contaminants released by J.H. Baxter and the other industrial sites in the immediate area. Although there are many sources of contamination near the three neighborhoods, we are unable to draw conclusions about the public health impacts related to the individual or collective contaminant sources at this time. EHAP also released a report (*Follow-up J.H. Baxter Health Assessment Based on New Air Monitoring Data)* in April 2007 to re-evaluate the public health impact posed by air emissions from J.H. Baxter.

BACKGROUND

In 2003, residents of the Bethel, River Road, and Trainsong neighborhoods expressed concern to EHAP staff about the possibility of increased rates of AML and brain cancer due to chemicals released by industrial sites closely bordering the densely populated neighborhoods. A map of the area of interest can be seen in Figure 1. According to the U.S. 2000 Census (Table 1), approximately 27,000 people live in Bethel, River Road, and Trainsong neighborhoods. The primary census tracts that make up the three neighborhoods are 26, 27, 28, 41, 42, and 43 (Figure 1).

The concerns about cancer rates were raised while EHAP was evaluating the health risk posed by emissions from J.H. Baxter and Company, a wood treatment plant. The original complaint from community members was the unpleasant odor coming from the wood creosoting plant. During a public meeting they described their frustration with the odors coming from the plant, and their concerns that exposure to the chemicals coming from this plant could be causing health effects, specifically cancer, in local residents. The chemical compounds used as preservatives at J.H. Baxter include pentachlorophenol, creosote, and ammonia copper zinc arsenate (ACZA). Polycyclic aromatic hydrocarbons (PAH's), a primary constituent of creosote, have been associated with lung and nasal cancer. EHAP prepared an initial health consultation to evaluate public health risks related to emissions from the J.H. Baxter plant. Inhalation was identified as a completed exposure pathway. The initial consultation concluded that there was an *indeterminate public health hazard* because of a lack of data. The health consultation recommended that more data on emissions from the site be gathered to better assess chemicals released by the plant. The health consultation also concluded that an investigation should be conducted to address the cancer concerns raised by community members although it was unlikely that the wood preservative emissions from the plant could be associated with increased cancer rates. It was suggested that the cancer investigation be conducted in coordination between the community, EHAP, and the Oregon State Cancer Registry (OSCaR).

The Lane Regional Air Pollution Agency (LRAPA) conducted air sampling between 2005 and 2006, and a follow-up health consultation was prepared to re-assess public health risks from exposure to contaminants from J.H. Baxter. EHAP concluded that the air monitoring data did not indicate people will become chronically ill from the emissions from J.H. Baxter, but recommended that J.H. Baxter take additional actions to reduce the creosote-related odors emitted into nearby neighborhoods.

Several other industrial sites also exist near residents' homes in or near the Bethel, River Road, and Trainsong neighborhoods (Figure 1), including Union Pacific Railroad (UPRR), many of which release chemicals in the area that are known or suspected carcinogens. Although there are many sources of contamination in these neighborhoods, we are unable to draw conclusions about the public health impacts from the individual or collective contaminant sources at this time.

COMMUNITY HEALTH CONCERNS

EHAP has had many opportunities to collect and listen to concerns expressed by residents in Bethel, and River Road and Trainsong neighborhoods over the past several years. Concerns have ranged from the immediate effects of breathing in airborne emissions from J.H. Baxter to long-range health effects, particularly cancer. Other long-term concerns include endocrine disruption, and damage to the respiratory and immune system. Residents have expressed concern regarding the contamination of air, soil, and water. Several residents have questions about how contaminants released from heavy automobile traffic and the numerous industrial sites may interact and affect the health of neighborhood residents.

Residents' specific concerns related to a potential cancer cluster stemmed from a number of AML cases within a small area in the Bethel neighborhood. Residents also learned about what seemed to them an unusual number of brain cancer cases in the neighborhood. Because of the

odors from J.H. Baxter and knowledge about chemicals released by the different industrial sites, residents came to believe that these cancer cases were related to environmental exposures. This document is intended to address some of those concerns.

EUGENE NEIGHBORHOOD CANCER INVESTIGATION HISTORY

In 2004, information on cancer rates was only available at the Zip Code level (for Zip Code 97402) at the time the initial Eugene cancer investigation was requested for AML and brain cancer [2]. Because of the data limitations, a more detailed review of cancer incidence at the neighborhood level was not possible in 2004. Residents thought there were an unusual number of AML cancer cases within the Bethel neighborhood, a much smaller locality than the Zip Code area. One community member consulted with a local physician, who stated that he thought the number of AML cases in the small geographic area seemed elevated. Other residents were aware of a former employee of J.H. Baxter who died from a brain malignancy, and indicated that they felt there were an unusually large number of brain cancer cases in Northwest Eugene. The decision was made to conduct a more detailed cancer investigation at the census tract level, once geocoded information became available, in order to better address area residents' specific concerns.

Figure 1. Map of Census Tracts and Industrial Sites in Bethel, River Road, and Trainsong Neighborhoods, Eugene, OR.

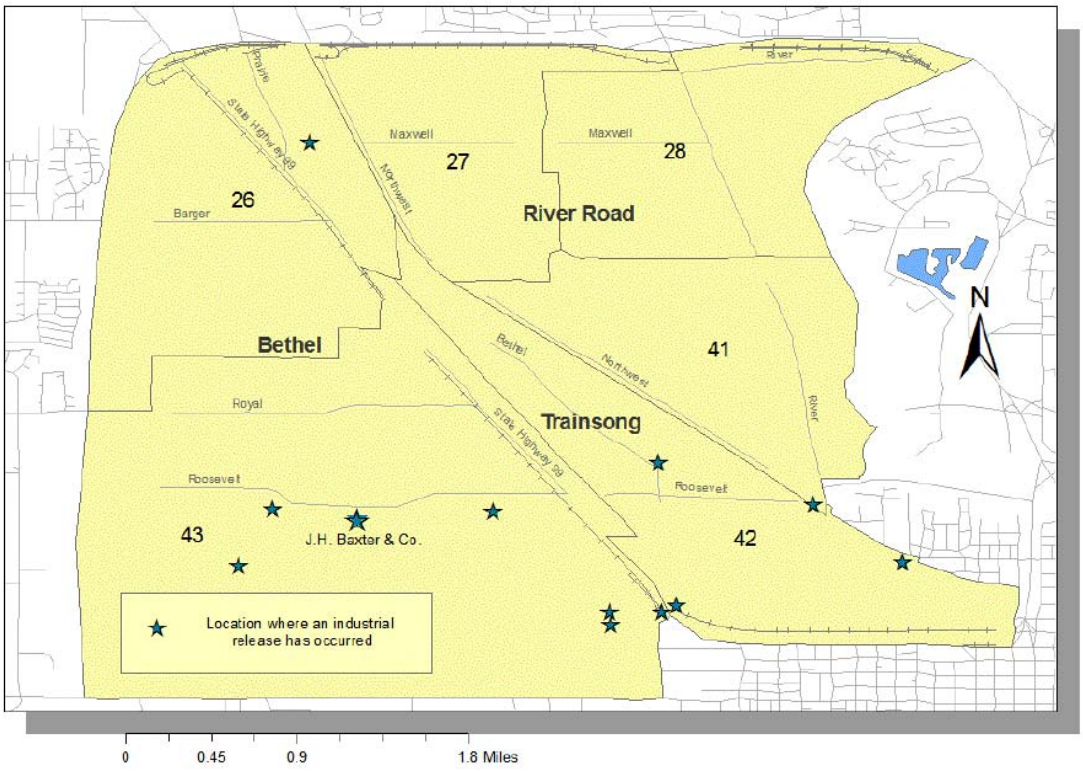

Table 1. Demographic Information for Bethel, River Road, and Trainsong Neighborhoods (Based on 2000 Census).

	Bethel		**River Road**			**Trainsong**
	Census Tract 26	Census Tract 43	Census Tract 27	Census Tract 28	Census Tract 41	Census Tract 42
Total population	5482	6515	3854	3960	3906	4066
Percent of Area Population	19.7%	23.4%	13.9%	14.2%	14.1%	14.6%
Male	2732	3147	1930	1910	1895	2337
Female	2750	3368	1924	2050	2011	1729
Age Group						
Under 5	415	475	272	247	234	308
5-14	845	977	671	548	524	453
15-24	788	821	443	539	615	752
25-34	932	916	561	554	520	899
35-44	852	1,011	590	658	657	750
45-54	650	803	542	650	709	548
55-64	346	455	280	315	292	184
65-74	300	456	273	224	183	92
75+	354	601	222	225	172	80
Median Age	32.5	35.5	34.6	36.3	36.1	30.1

Race or Ethnicity						
Caucasian	4968	5847	3410	3532	3491	3205
Black or African American	51	62	51	32	40	63
American Indian or Alaska Native	66	72	67	65	45	80
Asian	91	72	41	22	35	67
Native Hawaiian or Pacific Islander	5	22	13	8	3	15
Hispanic or Latino	278	434	232	254	235	654
Total Households	2076	2624	1407	1595	1534	1627
Owner Occupied	1284	1507	1041	957	1043	360
Renter Occupied	792	1117	366	638	491	1267
% Below Poverty Level 1999	11.42%	12.57%	8.28%	15.33%	13.95%	36.03%

Data are specifically for the 6 census tracts that make up the majority of Bethel, River Road and Trainsong Neighborhoods.

METHODS

After the data for census tracts became available for the years 1996 to 2004, the Oregon State Cancer Registry (OSCaR) reviewed cancer incidence for the census tracts that make up the majority of the area in the Bethel, River Road, and Trainsong neighborhoods. Information available from OSCaR about cancer in Oregon comes from a variety of sources including hospital cancer registries/medical records departments, ambulatory surgical centers, physician offices, pathology laboratories, other state cancer registries, and death certificates.

The number of observed cancer cases in each identified census tract was compared with the expected number of cases for each census tract during the years between 1996 and 2004 (See Table 1). OSCaR used current cancer rates in the State of Oregon to calculate the expected number of cases of AML, brain, lung, and nasal cancers in these census tracts. The Oregon Cancer Registry calculated the expected number of cases of each type of cancer for each census tract. For a detailed description of how expected rates were calculated, see Appendix A. They also did a comparison for all other cancers, and all cancers combined to determine whether cancer in general was elevated in those census tracts. Background information on the four cancers of specific interest can be found in Appendix C.

Some Cancer Facts

Cancer is a group of diseases characterized by the uncontrolled growth and spread of abnormal cells. If the spread is not controlled, it can result in death. Cancer is caused by both external (tobacco, chemicals, radiation and infectious organisms) and internal factors (inherited mutations, hormones, immune conditions, and mutations that occur from metabolism). These causal factors may act together or in sequence to initiate or promote carcinogenesis. Ten or more years often pass between exposure to external factors and detectable cancer.

- American Cancer Society

Comparison of Observed and Expected Cancers

The method for calculating the expected number of cases in a small geographic area often produces some odd effects. Specifically, it is not uncommon that the number of expected cases at the census tract level would be expressed as a fraction of a person (i.e. 2.4 expected cases). This is because the number of expected cases is based on the number of cases in the larger population, and cancer at the population level is expressed in terms of the number of cases per 100,000 people. For example, if the rate for the number of bladder cancer cases in Oregon in 1996 was 24/100,000 and we were looking at a geographic area that only included 1,000 people, we would say the number of expected cases of bladder cancer was 0.24 - or roughly ¼ of a case. This happens because there is a relatively low rate of bladder cancer at the population level and because the local population is small (1,000). This is important to understand because of the way that we express the excess number of observed cases. For instance, if we expect 0.24 cases, and we observe 1 case, mathematically we would say we have four times the number of cancer cases than expected. This is misleading because it suggests that we have a much larger problem than we actually do, when what we actually have is a mathematical effect from a small number of cases.

One way to address this problem caused by small numbers is to test the numbers statistically. A statistical test is used to test the possibility that increases in observed vs. expected cases could happen simply as a matter of chance. When a condition is relatively rare (as many cancers are) we use a test called a Poisson distribution, which is used when the probability of an event happening is very low. This test helps us evaluate whether the difference between the expected and observed numbers is statistically significant and not likely to be the result of chance or coincidence. Many scientific and public health studies use a 5% significance level ($p=0.05$), which was the level used for this analysis. Therefore, we can be 95% confident that statistically significant findings ($p<0.05$) are not due to chance or coincidence. It should be noted that while these tests can tell us whether there are statistically significant differences, they cannot tell us why these differences exist.

There are many risk factors that can contribute to the development of cancer (see "Some Cancer Facts" on page 9), and the presence of multiple risk factors can make it difficult to associate a cancer case or cluster with a single exposure. Statistical analysis methods sometimes can be used to examine an exposure-disease relationship in the presence of multiple risk factors (sometimes referred to as "controlling" for the other risk factors); however, these analyses often require detailed and accurate information on individual cases. In this investigation, we were not able to control for key factors such as smoking, industrial and occupational exposures, years of residence in a census tract and other potential risk factors. This limits our ability to associate any identified cancer clusters with potential environmental exposures. However, the data and methods used were appropriate for the purpose of this investigation, which was to determine whether the rates of certain cancers were higher than expected within the Bethel, River Road and Trainsong neighborhoods.

RESULTS

Table 2 summarizes cancer cases for all 6 census tracts from 1996 to 2004 and compares the actual number of cases (the "observed") to the number of cases expected, based on the rates of these cancers in Oregon. Statistical significance was evaluated by comparing the observed cases with the expected cases using a Poisson distribution. As described above, the Poisson distribution is an appropriate test of significance when the disease occurrence is rare (a small number of cases relative to the size of the population). Detailed data tables reporting the observed and expected number of cases for the cancers of concern for all available years can be found in Appendix B along with the numbers summarizing the statistical significance.

Table 2. Summary of Cancer Cases in Six Census Tracts in Northwest Eugene, OR for the Period 1996-2004.

	Acute Myeloid Leukemia		Brain Cancer		Lung Cancer		Nasal Cancer		All Other Cancers		All Cancers	
Census Tract	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp
26	1	1.6	6[(b)]	3.3	41	31.4	1	0.3	206	197.5	255	234.1
27	0	1.1	0	2.5	31	24.6	0	0.2	131	150.6	162	179.0
28	2	1.1	4	2.5	19	23.6	0	0.2	147	150.9	172	178.3
41	2	1.0	4	2.3	23	19.8	0	0.2	137	134.1	166	157.5
42	0	0.7	4	1.9	29 [(c)]	11.0	0	0.1	66	88.8	99	102.5
43	5	2.3	5	4.4	50	48.2	0	0.4	254	289.5	314	344.7
Total	10[(a)]	7.9	23	16.8	193[(d)]	158.6	1	1.4	941	1011.4	1168	1196.2

(a) $p = .271$, not statistically significant
(b) $p = .117$, not statistically significant
(c) $p < .001$, statistically significant
(d) $p = .004$, statistically significant

All Cancers Combined

The observed number of cases for all cancer combined between 1996 and 2004 for all census tracts was below the expected number of cases. "All other cancers" included all cancers from 1996 to 2004 except AML, brain, lung, and nasal cancer. The observed number of cases for all other cancers was also less than the expected number of cases for this time period.

Brain and Nasal Cancer

After running the appropriate statistical tests, we learned that there were no statistically significant elevations in rates for brain or nasal cancer in any of the census tracts during the period 1996-2004 in the three Eugene neighborhoods. In census tract 26 there were 6 cases of brain cancer when we expected to see 3.3. While this finding was not statistically significant, we further examined brain cancer cases in this census tract by time period. We determined that in one instance (from 1996 to 2002) the number of cases of brain cancer was significantly greater than the number of cases we expected to find. The highest number of cases of brain cancer in census tract 26 (n=2) were diagnosed in 2001, but there was no elevation in the observed number of cases in that census tract before 2001 or after 2002.

While we are always concerned with possible evidence of higher than expected cases of cancer, it is difficult to draw conclusions from these findings because small increases in cancer rates in small geographic areas are not uncommon. This is especially true when an increase occurs over a one or two year period rather than consistently over a several year period. At this time, the data indicate that the measurable increase in brain cancer may be due to chance, a possibility strengthened by the fact that we see very low numbers of cases in the years before 2001 and no cases after 2002.

Lung Cancer

Data from OSCaR indicate a statistically significant elevation in lung cancer in census tract 26 for the period 1996-2003, and in census tract 42 for the period 1996-2004. In census tract 26 for the period 1996-2003, the number of cases of lung cancer (n=39) significantly exceeded the expected number of cases (n=27.8). Based on a review of vital statistics from the Oregon Center for Health Statistics, of the 41 people diagnosed with lung cancer for the period 1996-2004, vital records indicate that tobacco contributed or probably contributed to 29 of the deaths.

In census tract 42, the number of cases of lung cancer (n=29) significantly exceed the expected number of cases (n=11) for the period 1996-2004. Based on a review of vital statistics from the Oregon Center for Health Statistics, 28 persons with lung cancer in census tract 42 died from their illness. In 27 of the 28 deaths, vital records indicate that tobacco contributed or probably contributed to the person's death. Tobacco use is strongly associated with lung cancer, and is considered to be the cause of eighty percent of all lung cancer cases.

OSCaR conducted an additional review of the cases to determine if people lived within the two census tracts for a period of time that would make plausible the possibility that a common environmental exposure could be associated with the development of lung cancer. Lung cancer is thought to have a latency period between exposure to a carcinogen and development of clinical disease of 10-30 years according to ATSDR.

Table 3. Length of residence in Census Tracts 26 and 42 for lung cancer patients at time of diagnosis.

Census Tract 26		**Census Tract 42**	
Number of Years	Number of patients	Number of Years	Number of patients
30+	2	30+	0
20-29	6	20-29	2
10-19	12	10-19	5
5-9	7	5-9	6
<5	8	<5	6
unknown	6	unknown	9
Total	**41**	**Total**	**29**

OSCaR learned that of the 41 people diagnosed with lung cancer in census tract 26 for the period 1996-2004, 35 had documented residence in that census tract prior to diagnosis. In census tract 42, twenty had a documented residence in that census tract prior to diagnosis. Documented period of residence for the cases in census tract 26 ranged from less than one year to 46 years, and in census tract 42 period of residence ranged from less than one year to 24 years.

In summary, we were able to determine residency in the census tracts prior to diagnosis for approximately 78% of cases. Where residence information is known, 57% of patients lived in census tract 26 for 10 or more years, and 40% of patients lived in census tract 42 for 10 or more years prior to diagnosis. The high rates of smoking in the group of cancer patients indicate that tobacco could be a primary contributor to the development of lung cancer in this group of residents. However, a significant proportion of these patients lived in these two neighborhoods

for ten years or longer, making it possible that a common environmental exposure could be a primary or secondary contributor to the development of lung cancer.

Acute Myeloid Leukemia (AML)

Ten (10) cases of AML were diagnosed in the 6 census tracts in the period of 1996-2004 when 7.9 cases were expected in that period (See Table 4). This is not a statistically significant elevation. A closer look at the data show that only 2 of those cases occurred before the year 2000. Between 2002 and 2004, 8 of the 10 cases occurred, when 3.3 were expected. This represents a statistically significant elevation. Further, 5 of the 10 total cases occurred in census tract 43, including 4 of the 8 cases reported between 2002 and 2004.

Table 4. Summary of AML Cases by Location and Time Period. (See Appendix B for Detailed Data Tables)

	1996-2004			2002-2004		
Cases of AML	*Obs*	*Exp*	*p-value*	*Obs*	*Exp*	*p-value*
All Census Tracts	10	7.9	0.271	8	3.3	0.020*
Census Tract 43	5	2.3	0.083	4	0.8	0.009*

**Statistically Significant*

When the years 2002-2004 are considered in isolation, OSCaR data indicate statistically significant increases in AML across all of the 6 census tracts, and in census tract 43 specifically. In general, focusing on an isolated time period in this fashion is not a standard approach in conducting a multi-year cancer data review. However, in this case, the decision was made to do so since the apparent increase in observed cases involved the most recent years for which data are available.

OSCaR conducted an additional review of the observed cases from census tract 43 between the years 1996 to 2004 to identify possible risk factors that may have contributed to the development of AML. In order for a plausible connection to be made between these cases and an environmental exposure, the individuals must have lived in the census tract for at least five years (the approximate latency period for AML). OSCaR learned that 4 of the 5 people with AML in census tract 43 lived in the area for nine years or longer. All 5 of the AML patients had a history of tobacco use, and 1 patient had a history of chemotherapy treatment. Both tobacco use and chemotherapy are associated with development of AML[8]. Based on the residency information, it is plausible that the affected individuals may have shared a common environmental exposure. However, because all of the cases had known risk factors for AML, it is unclear whether a common shared exposure contributed to the development of AML in these cases.

CANCER CLUSTER INVESTIGATIONS

Cancer cluster investigations are complex and difficult for several reasons, and when they are undertaken, they are implemented in a careful and methodical way. First, as in this case, a comparison is made between the observed and the expected number of cases in a specific geographic area during a specific period of time. When this comparison shows an excess number of cases, a cancer cluster investigation seeks to determine if the people with cancer in a specific area could have been exposed to something in their environment that may have caused their cancer. If a large proportion of people with cancer moved into the area after (or shortly before) they were diagnosed with cancer, it reduces the likelihood that an environmental exposure in that area is responsible for the excess cases of cancer.

If a plausible connection is made between the cases of cancer and the length of time it would take between exposure to an environmental contaminant and the cancer diagnosis, the investigation will continue. At that point more specific information is needed that would help determine if there is a way to measure the level and type of environmental exposure the people affected may have had, and if other factors such as occupational exposure, tobacco use, or family history of cancer could play a role. Determining the cause of cancer is a challenge because exposure to cancer-causing agents may have occurred many years earlier.

What is a Cancer Cluster?

A cancer cluster is a greater-than-expected number of cancer cases that occurs within a group of people in a geographic area over a period of time. Cancer cases are more likely to represent a cancer cluster if they involve (1) one type of cancer, (2) a rare type of cancer, or (3) a type of cancer in a group not usually affected by that cancer.

-Centers for Disease Control and Prevention

SENSITIVE POPULATIONS

Several factors put people at greater risk for developing cancer. Some people are more susceptible to developing cancer because they inherit altered genes, a weak immune system, or altered hormone levels [3]. Exposure to a cancer-causing chemical, behavioral choices, health, age, and gender can put people at greater risk for developing different types of cancer in addition to inherited conditions or genes. Occupational exposure to certain substances may also put workers at greater risk for developing cancer.

CHILD HEALTH CONSIDERATIONS

In general, EHAP and ATSDR recognize that infants and children may be more vulnerable than adults to exposures to contaminants in air, water, soil, or food. For this investigation, expected rates of cancer were calculated for all age groups, and the observed number cases were compared to expected number of cases by age group as well as geographic area. These comparisons indicated that children in this area were no more likely to have increased rates of cancer than their adult counterparts.

CONCLUSIONS

In summary, preliminary data from OSCaR for the years 1996-2004 were reviewed for six census tracts in northwest Eugene in order to determine whether the numbers of observed cases of specific cancers were higher than might normally be expected, based on random variation.

The data indicate that for the years 1996-2004, the number of lung cancer cases was higher than expected in the six census tracts combined and in census tract 42 specifically.

When the analysis was limited to shorter time periods, statistically significant elevations were also seen for lung cancer in census tract 26, brain cancer in census tract 26, and AML in census tract 43 (Table 5). Elevations in lung cancer were strongly associated with tobacco use among those affected. EHAP and the state cancer registry will monitor brain cancer and AML in the relevant census tracts and in the study area overall to determine if elevations have persisted in more recent years.

Table 5. Summary of Cancer Cluster Investigation Findings.

Cancer	Significant Elevations		Considerations
	Census Tract	Time Period	
AML	43*	2002-2004	• 80% of cases resided in census tract for 9+ years prior to diagnosis (latency period ~5 years) • Significant elevations based on data from a short time period (three years) and a small number of cases (4) • All cases had known risk factors for AML (tobacco use, chemotherapy)
Brain Cancer	26	1996-2002	• Reliability of statistical tests limited due to small numbers • No cases in 2003-2004
Lung Cancer	26	1996-2003	• About half of cases (49%) resided in census tracts for 10+ years prior to diagnosis (latency period ~ 10-30 years) • In the majority of cases (81%) in these tracts, physicians recorded on the death certificate that tobacco contributed or probably contributed to lung cancer • Tobacco use/other factors not controlled in analysis
	42*	1996-2004	
Nasal Cancer	None	None	• None
All Other Cancers	None	None	• None
All Cancers	None	None	• None

*Significant elevations in these census tracts caused significant elevations in all census tracts combined during the indicated time period.

EHAP is unable to determine at this time if there is a relationship between the excess number of cases of AML, brain cancer or lung cancer and exposure to environmental contaminants from a single or multiple sources. The public health impact of individual or multiple contaminants in these census tracts cannot be rigorously assessed due to limited information about individual case histories and past environmental exposures.

RECOMMENDATIONS

Tobacco smoke is a potent environmental contaminant that is a known risk factor for many types of cancer, including lung cancer and AML. This investigation identified tobacco use as a common risk factor for the majority of lung cancer and AML cases in the neighborhoods with statistically significant elevations. EHAP recommends the implementation or expansion of tobacco prevention and cessation programs in these neighborhoods, and health education and outreach to answer concerns about tobacco use and cancer.

LRAPA routinely collects air quality data in this area and surrounding areas as part of their regional air monitoring activities. EHAP recommends a review of this information to determine if they include data on any contaminants of concern. While these data will not allow EHAP to link observed cases of disease to environmental contamination, they may provide information on whether the residents of these neighborhoods experience exposures that increase their risk for possible health effects.

PUBLIC HEALTH ACTION PLAN

Past public health actions that EHAP conducted include the presentation of health consultation and health assessment findings of concern to the community at public meetings in May and November 2007.

EHAP will work with OSCaR to review rates of AML and brain and lung cancers in the Bethel, River Road and Trainsong neighborhoods as case information becomes available from OSCaR for the years 2005 – 2006. This information will help to determine whether there continue to be statistically significant elevations of these cancers in these neighborhoods.

EHAP will review existing air monitoring data collected by LRAPA. Based on this review, EHAP may support additional air monitoring in the Bethel, River Road and Trainsong neighborhoods. EHAP will provide facilitation and consultation services to LRAPA and other regulatory agencies as needed.

EHAP will continue to accept and respond to community concerns about environmental contaminants and their impacts on public health in the neighborhoods of Bethel, River Road, and Trainsong.

PUBLIC COMMENT

The revised health consultation report released in April 2007 was made available for public comment for 30 days. EHAP received comments from 1 citizen. The comments and our responses are reflected below.

Comment A
Thank you for undertaking this investigation of cancer in three of our west Eugene neighborhoods. It is interesting that multiple cancer clusters were found in our neighborhoods closest to Eugene's industrial area. However, it is disappointing that there was not enough data available to assess common environmental exposures that might be linked to these cancer clusters. It is also disappointing that recommendations call only for continued monitoring of cancer incidence data and assessment of individual exposure factors, and do not call for additional effort to gather data on common environmental exposures that might explain these excesses of cancer. And, it is dismaying that the report reassures residents that environmental exposures are unlikely to be the cause of the excess lung cancers despite the complete lack of data on environmental exposures. This conclusion seems unwarranted and perhaps falsely reassuring.

Response A
EHAP acknowledges that there are several limitations to this investigation, which in turn limit the types of conclusions that we can draw at this time. Many of these limitations are related to the availability of data for this investigation. The data that were available allowed us to examine and provide some answers to the question that prompted the investigation (whether there are elevated rates of certain cancers in the Bethel, River Road, and Trainsong neighborhoods). However, because we cannot obtain data on past environmental exposures, we are limited in our ability to scientifically examine whether these cancer clusters can be linked to one or more environmental contaminant. The conclusions and recommendations of this investigation are not intended to be falsely reassuring; they are based on our assessment of the data that were available and current scientific evidence on the causes of cancer.

Comment B
In **Table 1, Demographic Information**, something is amiss with the line of data that is labeled "Percent of Total Eugene population". Obviously these three neighborhoods do not make up 86% of Eugene's total population of 130,000.

Response B
Thank you. The table has been corrected. The title that line has been corrected to read “Total Area Population”

Comment C
It would have been helpful to have the age distribution of the population in each neighborhood listed in Table 1, since that information is used to calculate expected number of cancer cases in each area and could help the reader do a gross cross-check of the validity of the "expected" values for the different neighborhoods.

Response C
Table 1 has been updated to include the age distributions of each neighborhood.

Comment D
On page 9, in **Comparison of Observed and Expected Cancers**, you note that the Poisson test "helps us evaluate whether the difference between the expected and observed numbers is significant..." It would be helpful if the report elaborated just a little more on this test, including:
1) your choice of P value used as a breakpoint to determine statistical significance (presumably the conventional P=.05, but I was confused after studying Table 2, and also seeing that some data in the appendix tables with P values less than .05 (see lung cancer below) was not flagged or discussed at all in the text. I wondered if you were using P < .01 or P < .001; and
2) some acknowledgement that whatever P value was used is a convention and choice, not some immutable law of nature or statistics, and that a decision about whether findings are important in the real world involves questions of judgment that can go beyond a mechanistic "above/below p=.05" criterion.

Response D
The test of significance that was used was the conventional p=.05. This means that that we can have confidence that in no more than 5% of cases, random chance accounted for a statistical association between place of residence and cancer diagnosis (a "one in 20 chance"). This significance level is less conservative than 1% (p. <.01) or 0.1% (p. <.001), levels. We have included more detail about this test in the methods section on page 10. The table for lung cancer in the appendix has been corrected so that all significant values are in bold.

Comment E
The number of expected lung cancer cases in Trainsong (tract 42)--looks unusually low relative to figures calculated for other neighborhoods. Is it really true that only 11 lung cancers were expected among the 4066 residents in Trainsong, while more than double that number of cases (23.8) were expected among the 3960 residents in census tract 28 (NE River Road)? Is the population that much younger in Trainsong than in tract 28?

Response E
US Census data indicate that the population in Trainsong is younger when compared to the State of Oregon (which has a median age of 36.3), and to the other five census tracts included in this investigation. The median age for each of the six census tracts has been included in the demographic information in Table 1. The younger population in Trainsong probably explains why the number of expected cancer cases in this tract is lower than the expected cases in census tract 28, despite Trainsong having a larger population size.

Comment F
When using P<=.05 as the cutoff for statistical significance, the figures in the lung cancer data table in Appendix B (page 25) indicate a significant increase in lung cancer cases in census tract 26 (Bethel, north of Trainsong) during the periods 96-02 (P=0.016) and 96-03 (P=0.025)--yet these findings are not noted in the **Results** section. Instead, the text discusses only the statistically significant increase in tract 42 (Trainsong). The **Conclusion** section also states that

"It appears that the significant excess in observed cases in all census tracts is attributable to the excess cases in census tract 42", disregarding the significant increase in tract 26.

Response F
Excess cases of lung cancer in census tract 26 were not reported in the initial release of this document. This was an oversight and has been corrected in this version.

Comment G
EHAP says vital records indicate that tobacco was the underlying cause or probable cause of the person's death in 27 of the 28 lung cancer cases among Trainsong residents. Also, only 8 of the 29 residents with lung cancer--where residence information is known--were documented to have lived in Trainsong for more than 10 years (the suspected latency for lung cancer). For these reasons, EHAP concludes it is unlikely that this cluster of cancer cases are attributable to environmental causes, and does not seem to recommend any additional follow-up investigation (except for strongly encouraging residents to abstain from smoking).

EHAP is taking liberties with these assumptions and this conclusion. First, it is possible that all 9 of the Trainsong residents for whom residency information could not be determined had in fact lived in Trainsong more than 10 years prior to diagnosis. If so, then almost 60% of the cancer cases might have occurred in persons who were long-time residents of the neighborhood. Also, EHAP did not investigate the residency (or smoking history) of lung cancer patients in the second (evidently missed?) census tract with elevated lung cancer.

Response G
This version includes the available information on residency and smoking history for lung cancer patients in census tract 26.

Comment H
Second, the fact that vital records say tobacco was the underlying cause of nearly all the lung cancer deaths (in one of the two tracts) is not all that compelling. There is no information about whether the persons were heavy smokers, or light smokers years ago. I would guess that when doctors fill out a death certificate of a person who dies of lung cancer, they simply assume smoking to be the cause if the person is or was ever a smoker. I suspect they do not ask whether they had other exposures to potential carcinogens, such as working with chemicals or living near a railyard or highway or chemical factory. While smoking is a clear and obvious cause of lung cancer, it is also true that a large majority of smokers do not get lung cancer. Cancer is a complex, multi-stage disease process. Individuals have differing genetic susceptibility, and different chemicals can act as either initiators or promoters. Doctors were unlikely to have investigated any of these other factors or causes, but simply made a biased guess. This kind of data is not adequate to rule out other contributing environmental causes of the cancers.

Finally, for EHAP to selectively investigate smoking histories but not other potential exposures to lung carcinogens suggests that the intent is to try to rule out a common environmental exposure instead of to investigate if one exists. It is possible that though smoking may have initiated the lung cancers in these individuals or made them susceptible to disease that additional environmental exposures contributed as cancer promoters. Smokers who live in the area may

experience lung cancer in excess of normal rates in the population of smokers because of additional exposures to co-carcinogens in their environment. The fact that the expected number of lung cancers in Trainsong is relatively low to begin with suggests that the population is relatively young. This could support an hypothesis of a secondary contributing factor that accelerated the otherwise long latency of development of lung cancer in this young population.

Indeed, some of the constituents of cigarettes that are thought to affect development of lung cancer (naphthalene, benzo(a)pyrene, other PAHs) are also found as pollutants in air from industrial emissions or from other sources. Residents' exposures to these chemicals in air pollution are a potential cause of lung cancer that deserves to be investigated. JH Baxter is an emitter of naphthalene and other PAHs.

Diesel particulate matter is another pollutant in our neighborhoods' air that deserves investigation. According to Oregon DEQ, "Long term exposure to diesel particulate likely increases the chance of lung cancer." A 2004 study at the Union Pacific railyard in Roseville, CA concluded: "The study results indicate there are elevated concentrations of diesel particulate matter and associated cancer risk impacting a large area surrounding the rail yard." Given that the two census tracts with elevated lung cancer abut the Eugene Union Pacific railyard, a major source of diesel particulate emissions, additional investigation seems warranted. The railroad is responsible for a large amount of air pollution, and has done relatively little compared to other sources to reduce their emissions.

Comment I

The **Results** section says "based on residential history, it is unclear whether there are additional common exposures that could also be associated with AML cases." The **Conclusions** say that "additional investigation is needed to determine if these cases of AML are attributable to other or additional risk factors including an environmental exposure." However, the recommended next steps EHAP suggests are only that all cases of AML be reviewed for information on personal exposures to other known potential causal agents--smoking, radiation, chemotherapy, or occupational or other exposure to benzene.

EHAP needs to investigate the potential common environmental exposures that might contribute to this cancer cluster--and not just benzene, but formaldehyde, PAHs, and other potential leukemogens. As with lung cancer and smoking history, just focusing on the personal exposure history of individuals suggests a bias toward trying to rule out a common environmental cause rather than investigate whether such a cause might exist. In the case of AML, the latency period is much shorter than that for lung cancer (only 1-5 years), which should make it easier to assess actual exposures that might have contributed.

Weyerhaeuser MDF (just west of the western boundary of census tract 43) emitted 135,859 pounds of formaldehyde in 2002--making it one of the largest emitters to air of the chemical of all TRI-reporting facilities in the US. This facility and its pollution would seem a potential "culprit" contributing to risk of AML in tract 43 (Bethel) residents.

The years 2002-2004 (when AML rates were high) also correlate with a period when there were a high number of odor complaints about JH Baxter. Baxter's odors are assumed to correlate with

naphthalene content of the emissions. Naphthalene is also used as a surrogate for measuring total PAHs, suspected to cause cancer, including leukemia. More investigation of naphthalene and PAH exposure to area residents is needed, including bio-monitoring to look for PAH-DNA adducts that are a marker of exposure, and examination of potential sources of PAH exposure.

Comment J
Just looking at smoking histories and personal occupational or medical exposures to toxic chemicals among cancer patients is not sufficient. Much more could be done, and needs to be done, to investigate potential environmental exposures and connections to the cancer clusters that were found in our neighborhoods.

Response H-J
EHAP and OSCaR are very limited in the kinds of information that are available to assess past and current environmental exposures. Most environmental exposures are rarely identified or recorded in a person's medical history or charts. The environmental data from previous years that would allow us to estimate the environmental exposures that residents of these neighborhoods may not exist, though EHAP will review the data that are available to verify this. The available information on residency indicate that exposure to an environmental contaminant may have been a contributing factor in cases of lung cancer and AML. However, because we were unable to control for tobacco use in this analysis, we cannot rule out that tobacco use likely was the primary contributor to these cancers.

The available data also suggest that tobacco use is closely associated with the cases of lung cancer observed in both census tracts 26 and 42, and with the cases of AML in census tract 43. Tobacco smoke can be a significant environmental contaminant with known carcinogenic effects. Since we do not have and cannot acquire environmental data from previous years, or know the types and levels of environmental contaminants those who developed these cancers may have been exposed to, it is impossible to assess the relationship between other environmental exposures and cancer cases.

As mentioned previously, most environmental exposures are not identified or recorded in a person's medical history or charts. Tobacco use and occupational exposures are more commonly recorded and can sometimes help in understanding an individual's potential exposures. For this analysis, we were unable to obtain detailed information on history of tobacco use for each person; therefore, we had to rely on the information available to us through vital records. Again, we did not intend to provide false reassurance (*Comment A*) by stating that these cases of cancer are likely associated with tobacco use; the intention was to offer, based on the weight of evidence, the assessment that given the data available, exposure to tobacco and its associated contaminants appear to be a likely cause.

Comment K
The report states that in census tract 26 "in one instance" the number of brain cancer cases was significantly greater than the number expected, and that the highest number of brain cancer cases were diagnosed in 2001, but there was no elevation before 2001 (or after 2002). A few sentences later, the report implies that the increase occurred over a one or two year period only, and says the data indicate that the increase may be due to chance. The tone seems intended to downplay

this cancer cluster finding. Yet, the data in Appendix B show that the statistical significance of the elevation of brain cancer in census tract 26 remains over the period from 1996-2002. This seven-year period does not seem like "one instance" or one to two years. And if only one brain cancer case was diagnosed in this tract in 2005 and in 2006, it appears this would remain a statistically significant cluster over the nine-year period, and over the most recent seven years. Also, this number of brain cancers in this census tract was enough to cause the overall number of brain cancers in all 6 census tracts examined to be elevated and within a hair's breadth of (the arbitrary convention for) statistical significance. It is a rare enough cancer that even a single additional case per census tract in a year alters the results significantly. While no brain cancer cases were identified in this tract in 2003 and 2004, I am not sure it is fair to suggest this is a trend--2 brain cancer cases each were diagnosed in 2004 in two adjacent tracts (42 and 43). Given the arbitrary boundaries of the tracts, this is hardly reassuring. Overall, I am not sure why EHAP would try to spin this cancer cluster as due to chance, when it seems just as possible that the trend of elevation of brain cancer could re-emerge as data comes in for 2004 and 2005.

Comment L
According to a peer reviewed analysis of brain cancer: "One study in France reported an association between work with wood preservatives such as pentachlorophenol and gliomas. Although the initial increased risk reported in this study was 60%, the researchers subsequently identified additional cases of glioma in the wood preserving industry (Cordier *et al.* 1988). Cordier S, Poisson M, Gerin M, Varin J, Conso F, Hemon D. 1988. *Gliomas and exposure to wood preservatives*. Br J Ind Med 45:705-709."
(http://www.healthandenvironment.org/brain_cancer). Given that there is an industrial facility that emits pentachlorophenol in the census tract where a statistically significant excess of brain cancer was found, the evidence of a potential association between penta exposure and brain cancer seems important to mention. Were the brain cancer cases found in census tract 26 gliomas? Or other kinds of brain tumors?

Response L
All of the brain cancers in census tract 26 were gliomas or astrocytomas (a specific type of glioma).

Comment M
One or more permanent **air toxics monitoring station(s)** needs to be sited in West Eugene downwind of the industrial belt, to help identify and establish levels of carcinogens in the ambient air. Since the wind changes predominant direction during the year, and the industrial belt spans a large area, two or more monitors really are needed to provide for a reasonable sampling of the air contamination experienced by neighborhood residents.

Special air monitors need to be used to measure semi-volatile compounds and diesel particulate in air in these neighborhoods, and special **environmental monitoring studies** need to be undertaken to measure specific contaminants in the air (or house dust or soil) in the vicinity surrounding particular facilities. Some compounds can only reliably be measured with special methods, and effort needs to be made to use methods that are sufficiently sensitive and specific.

Biologic sampling needs to be undertaken to explore residents' actual exposure to various chemicals. Such data can be used to compare with baseline human exposure data and known environmental exposure sources, to increase the potential for detection of environmental exposure and disease relationships. Followup could also be done to investigate chromosomal aberrations (such as PAH-DNA adducts) in cancer patients or the local population to help "fingerprint" any chemicals that might be linked to the development of DNA damage or blood disorders or cancers.

Comment N
EHAP and OSCAR should continue to monitor lung cancer rates in our neighborhoods.

The data in this report show lung cancer to be significantly elevated in two census tracts just west of the railyard, and high (but at a slightly lower level of significance) in a third tract abutting the northern part of the railyard to the east (River Road/Maxwell area). Railyards are significant sources of diesel particulate matter, a known lung carcinogen. I would think that **future tracking of lung cancer rates in all of the tracts abutting the railyard would be warranted**, as well as **investigation of actual air pollutants** at the present time. **Monitoring of the levels of diesel particulate matter** in the air in neighborhoods surrounding and downwind from the railyard should be done, and/or **modeling of risks** (as is being done in neighborhoods surrounding many California railyards). **Biologic sampling** should be done to look for biomarkers of diesel exposure in nearby residents.

And just as EHAP urges residents to stop smoking, your agency should **urge Union Pacific Railroad** to take steps now--such as those underway in California--**to reduce its emissions of diesel particulates** to our neighborhoods' airshed. EHAP should also **urge JH Baxter to reduce naphthalene emissions** to the public airshed. Scientists tell us there is no "safe" level of exposure to a carcinogen. Residents' (involuntary) exposures to naphthalene and diesel particulate from these sources is potentially as harmful as exposure to these carcinogenic compounds from (voluntary or second-hand) smoking.

Comment O
EHAP and OSCAR should **continue to monitor AML rates** in our neighborhoods.

EHAP should also **investigate reported or measured emissions** of known or suspected leukemogens--such as pentachlorophenol, benzene, formaldehyde, and PAHs (which cause chromosomal aberrations thought to lead to cancer)--from nearby industrial facilities such as JH Baxter, **Weyerhaeuser MDF on Danebo,** and others. If air monitoring data cannot be collected, Toxics Release Inventory (TRI) data on estimated emissions can be used. TRI data is available back to 1986.

A **special study of formaldehyde exposure** of residents in this tract and/or nearest the Weyerhaeuser MDF facility may be warranted.

More **investigation of naphthalene and PAH exposure** to area residents is needed, including **bio-monitoring** to look for PAH adducts that are a marker of exposure, and examination of potential sources of PAH exposure (including the JH Baxter wood preserving facility).

Also, a **special study** of the emissions of **pentachlorophenol (and its chlorinated dibenzodioxin and furan contaminants)** from JH Baxter and LD McFarland, and potential exposure to residents, would be important before ruling out environmental pollutants as causes of cancer in our neighborhoods. The Baxter facility reports suspiciously low emissions of penta to air. An independent assessment of the validity of the company's emissions estimates is important.

A followup health investigation could also look at **incidence of aplastic anemia and other blood disorders** or myeloproliferative diseases in this neighborhood, and **potential environmental exposures** linked to these conditions. Some of these conditions, such as polycythemia vera, have been associated with later development of AML.

Comment P
EHAP should continue to **track brain cancer cases** in all 6 W. Eugene census tracts, not just tract 26.

EHAP should **assess the location of cancer cases within the census tracts**, and **identify any nearby industrial facilities that emit chemicals that are potential risk factors** for brain cancer. A **special study** of pentachlorophenol (and dioxin) exposure of residents surrounding the LD McFarland wood preserving facility may be warranted.

Comment Q
It would be helpful to have information in each paragraph about the latency periods of the specific cancers, if known.

The paragraph on **AML** should include **PAHs and pentachlorophenol** as potential leukemogens. Research in 2005 demonstrated that prenatal exposure to airborne PAHs at relatively low levels causes chromosomal changes that have been linked to leukemia and other cancers (Perera, et al). Also, "Chronic exposure by inhalation to pentachlorophenol in humans has resulted in ... blood effects such as aplastic anemia" and "Case reports suggest a possible association between inhalation pentachlorophenol exposure and cancer (Hodgkins disease, soft tissue sarcoma, and acute leukemia)", according to US EPA Technology Transfer Network, Pentachlorophenol hazard summary (Jan. 2000), http://www.epa.gov/ttn/atw/hlthef/pentachl.html.

The paragraph on **brain cancer should also mention pentachlorophenol** as a possible risk factor, as mentioned previously.

In the **lung cancer** paragraph, exposure to **diesel particulate** should be mentioned as a risk factor. Diesel particulate is a known lung carcinogen, and there is a major emitter, the railyard, that abuts the two census tracts where elevated lung cancer was found.

Response K, M-Q
The findings of this investigation do not rule out the possibility that exposure to environmental contamination may have been a contributing factor to these cancer clusters. Instead, the findings

have helped to identify data gaps and limitations, particularly the need for environmental monitoring data and more accurate information on individual case histories. They also provide some basis to focus further investigations.

EHAP will review rates of AML and brain and lung cancers for the years 2005-2006, and will review available air quality data from LRAPA to determine whether they address any of the identified data gaps. The findings of this follow-up assessment may provide support for recommendations to expand environmental monitoring and conduct additional studies of the health impacts of environmental exposures in these neighborhoods.

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CERTIFICATION

The Environmental Health Assessment Program of the Oregon Department of Human Services prepared this report of a cancer investigation for the Bethel, River Road, and Trainsong neighborhoods under a cooperative agreement with the Agency for Toxic Substances and Disease Registry. This document was completed in accordance with approved methodology and procedures existing at the time the health consultation was initiated. Editorial review was completed by the Cooperative Agreement partner.

Alan Crawford
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I have reviewed this health consultation, as the designated representative of the Agency for Toxic Substances and Disease Registry and concur with its findings.

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APPENDIX A - CALCULATING EXPECTED NUMBER OF CANCER CASES

To calculate the expected number of cases in each census tract, a count was created utilizing indirect adjustment, applying age-specific rates for the state as a whole to 18 age groups in each census tract. The 18 age groups were 0-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, and 85+.

The age-specific rates for Oregon were calculated by dividing the number of cases of cancer in each age group by the number of people in each age group. Cases diagnosed from January 1, 1996 through 2004 were included. Population estimates from the US 2000 Census were used.

As an example, to calculate Oregon's age-specific rate for all cancers during 1996-2004 among people age 50-54, the number of cases of cancer in that age-group between 1996 and 2004 (13,035) was divided by nine times the number of Oregonians aged 50-54 from the 2000 Census (235,840 X 9 = 2,122,560). This number was then multiplied by 100,000 to give an age-adjusted rate of 614.1 per 100,000.

To outline the calculation in general terms: age-specific rate per 100,000 = (number of cases in age group / population in that age group) X 100,000.

To calculate the expected number of cases for a given census tract during 1996-2004, the number of people in a given age group in that census tract was multiplied by the Oregon age-specific rate for that age group. For example, in census tract 42 there were 236 people age 50-54 in the 2000 Census. That number was multiplied by 9 to estimate 9 years of population (236 X 9 = 2,124). That estimate was then multiplied by the state's age-specific rate for all cancers in the 50-54 age group (2,124 X 614.1) and divided by 100,000 to produce the expected number of cases for the 50-54 age group in census tract 42 (13). This same process was carried out for each age group, and the results were added together to produce the total number of expected cancer cases in census tract 42 (102.5).The expected number of cases was compared to the observed number of cases in the same time period (99).

To summarize: calculation of expected count in census tract = (population in age group X age-specific rate) / 100,000.

APPENDIX B – OBSERVED V. EXPECTED RATES OF CANCERS IN 6 LANE CTY. CENSUS TRACTS

Acute Myeloid Leukemia (AML) by census tract and year, 1996-2004

	Year									Year Grouping								Test of Significance			
	1996	1997	1998	1999	2000	2001	2002	2003	2004	1996-2002		1996-2003		1996-2004		2002-2004		poisson			
Lane Cty CT	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	**96-02**	**96-03**	**96-04**	**02-04**
002600	0	0	0	0	0	0	0	1	0	1.2	0	1.4	1	1.6	1	0.6	1	0.301	0.500	0.500	0.451
002700	0	0	0	0	0	0	0	0	0	0.9	0	1.0	0	1.1	0	0.9	0	0.406	0.367	0.332	0.406
002800	0	0	1	0	0	0	1	0	0	0.9	2	1.0	2	1.1	2	0.4	1	0.227	0.264	0.300	0.330
004100	0	0	0	0	0	0	1	0	1	0.8	1	0.9	2	1.0	2	0.4	2	0.500	0.227	0.264	0.062
004200	0	0	0	0	0	0	0	0	0	0.6	0	0.6	0	0.7	0	0.3	0	0.500	0.500	0.496	0.500
004300	0	0	0	1	0	0	1	2	1	1.7	2	2.0	4	2.3	5	0.8	4	0.500	0.142	0.083	**0.009**
TOTAL	0	0	1	1	0	0	3	3	2	6.0	5	6.8	9	7.9	10	3.3	8	0.446	0.245	0.271	**0.020**

**Case definition for AML has been changed from the initial public comment version of this health consultation. Initial case definition included all acute leukemias. This case definition includes only acute myeloid leukemia (AML)*

Brain cancer by census tract and year, 1996-2004

	Year									Year Grouping						Test of Significance		
	1996	1997	1998	1999	2000	2001	2002	2003	2004	1996-2002		1996-2003		1996-2004		poisson		
Lane Cty CT	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	**96-02**	**96-03**	**96-04**
002600	1	1	0	0	1	2	1	0	0	2.4	6	2.8	6	3.3	6	**0.035**	0.065	0.117
002700	0	0	0	0	0	0	0	0	0	1.8	0	2.1	0	2.5	0	0.165	0.122	0.082
002800	1	0	1	0	0	0	2	0	0	1.9	4	2.1	4	2.5	4	0.125	0.161	0.242
004100	0	0	2	0	0	1	1	0	0	1.7	4	2.0	4	2.3	4	0.093	0.142	0.200
004200	1	0	0	1	0	0	0	0	2	1.4	2	1.6	2	1.9	4	0.408	0.475	0.125
004300	0	1	0	0	1	1	0	0	2	3.3	3	3.8	3	4.4	5	0.500	0.473	0.448
TOTAL	3	2	3	1	2	4	4	0	4	12.5	19	14.5	19	16.8	23	0.052	0.147	0.268

Lung cancer by census tract and year, 1996-2004

	Year									Year Grouping						Test of Significance		
	1996	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**	**2003**	**2004**	**1996-2002**		**1996-2003**		**1996-2004**		**poisson**		
Lane Cty CT	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***EXP***	***OBS***	***EXP***	***OBS***	***EXP***	***OBS***	**96-02**	**96-03**	**96-04**
002600	3	5	7	1	6	5	9	3	2	24.4	36	27.8	39	31.4	41	**0.016**	**0.025**	0.056
002700	2	3	3	6	3	1	8	3	2	19.1	26	21.8	29	24.6	31	0.076	0.080	0.119
002800	1	3	2	2	1	1	3	5	1	18.3	13	20.9	18	23.6	19	0.127	0.309	0.201
004100	2	1	2	5	3	2	4	1	3	15.4	19	17.6	20	19.8	23	0.209	0.314	0.264
004200	3	2	7	2	1	1	6	2	5	8.5	22	9.7	24	11.0	29	**0.000**	**0.000**	**0.000**
004300	5	5	4	4	5	8	4	7	8	37.3	35	42.6	42	48.2	50	0.393	0.500	0.416
TOTAL	16	19	25	20	19	18	34	21	21	122.9	151	140.4	172	158.6	193	**0.007**	**0.005**	**0.004**

Nasal cancer by census tract and year, 1996-2004

	Year									Year Grouping						Test of Significance		
	1996	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**	**2003**	**2004**	**1996-2002**		**1996-2003**		**1996-2004**		**poisson**		
Lane Cty CT	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***OBS***	***EXP***	***OBS***	***EXP***	***OBS***	***EXP***	***OBS***	**96-02**	**96-03**	**96-04**
002600	0	0	0	0	1	0	0	0	0	0.2	1	0.3	1	0.3	1	0.181	0.259	0.259
002700	0	0	0	0	0	0	0	0	0	0.2	0	0.2	0	0.2	0	0.500	0.500	0.500
002800	0	0	0	0	0	0	0	0	0	0.2	0	0.2	0	0.2	0	0.500	0.500	0.500
004100	0	0	0	0	0	0	0	0	0	0.1	0	0.2	0	0.2	0	0.500	0.500	0.500
004200	0	0	0	0	0	0	0	0	0	0.1	0	0.1	0	0.1	0	0.500	0.500	0.500
004300	0	0	0	0	0	0	0	0	0	0.3	0	0.4	0	0.4	0	0.500	0.500	0.500
TOTAL	0	0	0	0	1	0	0	0	0	1.1	1	1.3	1	1.4	1	0.500	0.500	0.500

All other cancers by census tract and year, 1996-2004

	Year									Year Grouping						Test of Significance		
	1996	1997	1998	1999	2000	2001	2002	2003	2004	1996-2002		1996-2003		1996-2004		poisson		
Lane Cty CT	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	96-02	96-03	96-04
002600	21	19	25	17	21	23	24	26	30	150.7	150	172.8	176	197.5	206	0.499	0.414	0.282
002700	11	16	16	14	11	10	19	13	21	115.1	97	131.9	110	150.6	131	0.048	0.029	0.057
002800	15	18	10	16	17	20	20	15	16	115.0	116	131.9	131	150.9	147	0.475	0.492	0.396
004100	18	23	13	15	5	12	19	14	18	102.0	105	117.1	119	134.1	137	0.396	0.443	0.413
004200	9	12	8	6	4	8	7	7	5	67.2	54	77.3	61	88.8	66	0.057	**0.033**	**0.007**
004300	32	36	24	32	20	29	31	29	21	221.2	204	253.6	233	289.5	254	0.130	0.102	0.018
TOTAL	106	124	96	100	78	102	120	104	111	771.3	726	884.5	830	1011.4	941	0.052	**0.034**	**0.013**

All cancers by census tract and year, 1996-2004

	Year									Year Grouping						Test of Significance		
	1996	1997	1998	1999	2000	2001	2002	2003	2004	1996-2002		1996-2003		1996-2004		poisson		
Lane Cty CT	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	*EXP*	*OBS*	96-02	96-03	96-04
002600	25	25	32	18	29	30	34	30	32	179.7	193	205.9	223	234.1	255	0.152	0.111	0.082
002700	13	19	19	20	14	11	27	16	23	137.6	123	157.5	139	179.0	162	0.113	0.073	0.107
002800	17	21	14	18	18	21	26	20	17	136.7	135	156.7	155	178.3	172	0.465	0.467	0.336
004100	20	24	17	20	8	15	25	15	22	120.5	129	138.2	144	157.5	166	0.231	0.322	0.259
004200	13	14	15	9	5	9	13	9	12	78.3	78	89.9	87	102.5	99	0.500	0.406	0.389
004300	37	42	28	37	26	38	36	38	32	264.7	244	303.3	282	344.7	314	0.106	0.115	0.050
TOTAL	125	145	125	122	100	124	161	128	138	917.4	902	1051.6	1030	1196.2	1168	0.325	0.268	0.221

APPENDIX C - GENERAL CANCER INFORMATION

Note – The citations listed in this section can be found in the reference section at the end of the main body of this document

The American Cancer Society (ACS) estimates that approximately one in two men and one in three women will develop cancer in their lifetime [4]. It is a disease associated with increasing age, and nearly eighty percent (77%) of all cancer cases occur in adults 55 years or older. It is the leading cause of death for people under the age of 85 [5], and the second leading cause of all deaths in the United States.

Cancer, a group of over 200 diseases, develops inside the cell and disrupts the normal process of cell development [3]. Cancer causes cells to divide continuously when new cells are not needed.

It is estimated that smoking causes nearly two-thirds of cancers, and 25-30% of cancers are caused by obesity and physical inactivity. [3]. Other environmental factors linked to cancer include viruses, radiation, medications, and chemicals in the air and water. Identifying the factor or factors that act alone or in combination to cause cancer is difficult.

A cancer cluster is defined as a greater-than-expected number of cancer cases that occurs within a group of people in a geographic area over a period of time [6]. It is not uncommon to wonder about the cause of cancers when they are grouped in a geographic area, and people often fear that pollution or environmental contamination is the cause. Cancer clusters can and do occur because of exposures from a common source but they are difficult to document [5]. There are some important considerations to take into account when trying to evaluate whether a cancer cluster exists.

1.) Cancer is the second leading cause of death in the U.S., and consists of about 200 different types that may not share a common cause.
2.) It is difficult to track the cause of most cancers. For some, the cause is unknown. For others, there may be a long period between the time that one or more exposures that trigger the disease and the time cancer is diagnosed.
3.) A person may change residence between exposure and the development of cancer, making it difficult draw connections between environmental exposures and disease.
4.) Occupation and individual behavior (smoking, nutrition, and exercise) play significant roles in the risk of developing cancer.

Possible cancer clusters can initially be evaluated by defining a population (i.e., neighborhood or workplace) and calculating the expected number of cases in that group over a period of time, based on a comparison population. The observed number of cases is then compared to the expected number of cancer cases in that population.

AML

Acute myeloid leukemia (AML) is the most common type of leukemia, a cancer of the blood and bone marrow [7]. It causes the production of abnormal cells including blasts that normally develop into white blood cells, red blood cells, and platelets. The abnormal leukemia cells crowd out normal red and white blood cells and platelets. It is a disease that usually affects older adults (average age at diagnosis is 65 years) and nearly 12,000 new cases are diagnosed in the U.S. each year.

Occupational exposures to certain hazardous substances and specific occupations are associated with an increased risk of developing leukemia [8]. A strong association exists between exposure to benzene, ethylene oxide, and ionizing radiation along with working in boot and shoe manufacturing and repair. An association means there is evidence of a link between an environmental exposure and a disease [9] but it does not assume that exposure to that substance will automatically result in that disease. Other substances or industries that may also be linked to an increased risk of developing leukemia are formaldehyde, non-arsenical (non-arsenic containing) pesticides and the rubber industry or petroleum refining [8].

Brain

Brain cancers are categorized according to the type of cell affected. There are several types of brain cancers since tumors can form in any of the brain tissues, cells, or a mixture of cell types [4]. Only primary malignant brain tumors were included in this investigation, and not benign tumors or tumors that had spread from other sites. There is strong evidence linking brain cancer with pesticide exposure and ionizing radiation [5]. There is some evidence of a link between brain cancer and solvents such as benzene and toluene and metals such as lead, arsenic and mercury.

Lung

Lung cancer is the second most common type of cancer[5]. It is estimated that nearly 175,000 people will develop lung cancer in the U.S. in 2006 [10]. There are two main types of lung cancer: small cell and non-small cell. Several environmental contaminants are associated with lung cancer, in addition to the well-known link between lung cancer and tobacco smoke. Natural fibers such as silica, wood dust, and asbestos are strongly linked with lung cancer as well as exposure to arsenic, beryllium, cadmium, and chromium [5]. Exposure to polycyclic aromatic hydrocarbons (PAHs), ionizing radiation, benzene, toluene, mustard agent, and coal tar pitch are also linked with lung cancer. Lung cancer is thought to have a long latency between exposure to a carcinogen and development of clinical disease (10-30 years according to ATSDR).

Nasal

Nasal cancer is a rare cancer that affects approximately 2,000 people each year in the U.S. [11]. Several different cells make up the nasal cavity resulting in several different types of nasal cancer [4]. The most common type of nasal cancer is squamous cell carcinoma. Occupational exposures that have been linked to nasal cancer include exposure to dusts from wood, textiles, and leather, glues, formaldehyde, solvents used in furniture and shoe production, nickel and chromium dust, mustard agent, isopropyl ("rubbing") alcohol, and radium [4]. Inhalation of naphthalene, a PAH that is a major constituent of coal tar and petroleum, has also been shown to cause nasal cancer in an animal study [12].

APPENDIX D - ATSDR PLAIN LANGUAGE GLOSSARY OF ENVIRONMENTAL HEALTH TERMS.

Absorption	How a chemical enters a person's blood after the chemical has been swallowed, has come into contact with the skin, or has been breathed in.
Acute Exposure	Contact with a chemical that happens once or only for a limited period of time. ATSDR defines acute exposures as those that might last up to 14 days.
Additive Effect	A response to a chemical mixture, or combination of substances, that might be expected if the known effects of individual chemicals, seen at specific doses, were added together.
Adverse Health Effect	A change in body function or the structures of cells that can lead to disease or health problems.
ATSDR	The **A**gency for **T**oxic **S**ubstances and **D**isease **R**egistry. ATSDR is a federal health agency in Atlanta, Georgia that deals with hazardous substance and waste site issues. ATSDR gives people information about harmful chemicals in their environment and tells people how to protect themselves from coming into contact with chemicals.
Background Level	An average or expected amount of a chemical in a specific environment. Or, amounts of chemicals that occur naturally in a specific environment.
Bioavailability	See **Relative Bioavailability**.
CAP	**See Community Assistance Panel.**
Cancer	A group of diseases which occur when cells in the body become abnormal and grow, or multiply, out of control
Carcinogen	Any substance shown to cause tumors or cancer in experimental studies.
CERCLA	See **C**omprehensive **E**nvironmental **R**esponse, **C**ompensation, and **L**iability **A**ct.
Chronic Exposure	A contact with a substance or chemical that happens over a long period of time. ATSDR considers exposures of more than one year to be *chronic*.
Completed Exposure Pathway	

Comparison Value (CVs)	Concentrations of substances in air, water, food, and soil that are unlikely, upon exposure, to cause adverse health effects. Comparison values are used by health assessors to select which substances and environmental media (air, water, food and soil) need additional evaluation while health concerns or effects are investigated.
Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)	**CERCLA** was put into place in 1980. It is also known as **Superfund**. This act concerns releases of hazardous substances into the environment, and the cleanup of these substances and hazardous waste sites. This act created ATSDR and gave it the responsibility to look into health issues related to hazardous waste sites.
Concentration	How much or the amount of a substance present in a certain amount of soil, water, air, or food.
Contaminant	See **Environmental Contaminant**.
Delayed Health Effect	A disease or injury that happens as a result of exposures that may have occurred far in the past.
Dermal Contact	A chemical getting onto your skin. (See **Route of Exposure**).
Dose	The amount of a substance to which a person may be exposed, usually on a daily basis. Dose is often explained as "amount of substance(s) per body weight per day".
Dose / Response	The relationship between the amount of exposure (dose) and the change in body function or health that result.
Duration	The amount of time (days, months, years) that a person is exposed to a chemical.
Environmental Contaminant	A substance (chemical) that gets into a system (person, animal, or the environment) in amounts higher than the **Background Level**, or what would be expected.
Environmental Media U.S.	Usually refers to the air, water, and soil in which chemicals of interest are found. Sometimes refers to the plants and animals that are eaten by humans. **Environmental Media** is the second part of an **Exposure Pathway**.
Environmental Protection Agency (EPA)	The federal agency that develops and enforces environmental laws to protect the environment and the public's health.

Epidemiology	The study of the different factors that determine how often, in how many people, and in which people will disease occur.
Exposure	Coming into contact with a chemical substance. (For the three ways people can come in contact with substances, see **Route of Exposure**.)
Exposure Assessment	The process of finding the ways people come in contact with chemicals, how often and how long they come in contact with chemicals, and the amounts of chemicals with which they come in contact.
Exposure Pathway	A description of the way that a chemical moves from its source (where it began) to where and how people can come into contact with (or get exposed to) the chemical. ATSDR defines an exposure pathway as having 5 parts 1. Source of Contamination, 2. Environmental Media and Transport Mechanism, 3. Point of Exposure, 4. Route of Exposure, and 5. Receptor Population. When all 5 parts of an exposure pathway are present, it is called a **Completed Exposure Pathway**. Each of these 5 terms is defined in this Glossary.
Frequency	How often a person is exposed to a chemical over time; for example, every day, once a week, or twice a month.
Hazardous Waste	Substances that have been released or thrown away into the environment and, under certain conditions, could be harmful to people who come into contact with them.
Health Effect	ATSDR deals only with **Adverse Health Effects** (see definition in this Glossary).
Indeterminate Public Health Hazard	The category is used in Public Health Assessment documents for sites where important information is lacking (missing or has not yet been gathered) about site-related chemical exposures.
Ingestion	Swallowing something, as in eating or drinking. It is a way a chemical can enter your body (See **Route of Exposure**).
Inhalation	Breathing. It is a way a chemical can enter your body (See **Route of Exposure**).

LOAEL	**L**owest **O**bserved **A**dverse **E**ffect **L**evel. The lowest dose of a chemical in a study, or group of studies, that has caused harmful health effects in people or animals.
Malignancy	See **Cancer**.
MRL	**M**inimal **R**isk **L**evel. An estimate of daily human exposure – by a specified route and length of time -- to a dose of chemical that is likely to be without a measurable risk of adverse, non-cancerous effects. An MRL should not be used as a predictor of adverse health effects.
NPL	The **N**ational **P**riorities **L**ist. (This is part of **Superfund**.) A list kept by the U.S. Environmental Protection Agency (EPA) of the most serious uncontrolled or abandoned hazardous waste sites in the country. An NPL site needs to be cleaned up or is being looked at to see if people can be exposed to chemicals from the site.
NOAEL	**N**o **O**bserved **A**dverse **E**ffect **L**evel. The highest dose of a chemical in a study, or group of studies, that did not cause harmful health effects in people or animals.
No Apparent Public Health Hazard	The category is used in ATSDR's Public Health Assessment documents for sites where exposure to site-related chemicals may have occurred in the past or is still occurring but the exposures are not at levels expected to cause adverse health effects.
No Public Health Hazard	The category is used in ATSDR's Public Health Assessment documents for sites where there is evidence of an absence of exposure to site-related chemicals.
PAH	**P**olycyclic **A**romatic **H**ydrocarbons - one of a class of chemical compounds, organic pollutants
PHA	**P**ublic **H**ealth **A**ssessment. A report or document that looks at chemicals at a hazardous waste site and tells if people could be harmed from coming into contact with those chemicals. The PHA also tells if possible further public health actions are needed.
Plume	A line or column of air or water containing chemicals moving from the source to areas further away. A plume can be a column or clouds of smoke from a chimney or contaminated underground water sources or contaminated surface water (such as lakes, ponds and streams).

Point of Exposure	The place where someone can come into contact with a contaminated environmental medium (air, water, food or soil). Some examples include the area of a playground that has contaminated dirt, a contaminated spring used for drinking water, or the backyard area where someone might breathe contaminated air.
PRP	**P**otentially **R**esponsible **P**arty. A company, government or person that is responsible for causing the pollution at a hazardous waste site. PRP's are expected to help pay for the clean up of a site.
Public Health Assessment(s)	See **PHA**.
Public Health Hazard	The category is used in PHA's for sites that have certain physical features or evidence of chronic, site-related chemical exposure that could result in adverse health effects.
Health Hazard Criteria	People who live or work in the path of one or more chemicals, and who could come into contact with them (See **Exposure Pathway**).
Reference Dose (RfD)	An estimate, with safety factors (see **safety factor**) built in, of the daily, life-time exposure of human populations to a possible hazard that is <u>not</u> likely to cause harm to the person.
Relative Bioavailability	The amount of a compound that can be absorbed from a particular medium (such as soil) compared to the amount absorbed from a reference material (such as water). Expressed in percentage form.
Route of Exposure	The way a chemical can get into a person's body. There are three exposure routes – breathing (also called inhalation), – eating or drinking (also called ingestion), and – getting something on the skin (also called dermal contact).
Safety Factor	Also called **Uncertainty Factor**. When scientists don't have enough information to decide if an exposure will cause harm to people, they use "safety factors" and formulas in place of the information that is not known. These factors and formulas can help determine the amount of a chemical that is <u>not</u> likely to cause harm to people.
SARA	The **Superfund Amendments and Reauthorization Act** in 1986 amended CERCLA and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects resulting from chemical exposures at hazardous waste sites.
Sample Size	The number of people that are needed for a health study.

Sample	A small number of people chosen from a larger population (See Population).
Source (of Contamination)	The place where a chemical comes from, such as a landfill, pond, creek, incinerator, tank, or drum. Contaminant source is the first part of an **Exposure Pathway**.
Special Populations	People who may be more sensitive to chemical exposures because of certain factors such as age, a disease they already have, occupation, sex, or certain behaviors (like cigarette smoking). Children, pregnant women, and older people are often considered special populations.
Statistics	A branch of the math process of collecting, looking at, and summarizing data or information.
Superfund Site	A way to collect information or data from a group of people (**population**). Surveys can be done by phone, mail, or in person. ATSDR cannot do surveys of more than nine people without approval from the U.S. Department of Health and Human Services.
Synergistic effect	A health effect from an exposure to more than one chemical, where one of the chemicals worsens the effect of another chemical. The combined effect of the chemicals acting together is greater than the effects of the chemicals acting by themselves.
Toxic	Harmful. Any substance or chemical can be toxic at a certain dose (amount). The dose is what determines the potential harm of a chemical and whether it would cause someone to get sick.
Toxicology	The study of the harmful effects of chemicals on humans or animals.
Tumor	Abnormal growth of tissue or cells that have formed a lump or mass.
Uncertainty Factor	See **Safety Factor**.