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Cluster of Hodgkin’s lymphoma in residents near a non-operational petroleum refinery

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This report examines the prevalence rate of Hodgkin’s disease in an American mid-west town located directly south of a non-operational oil refinery. The refinery has a history of benzene-containing gasoline leaks dating back to the early 1900s. Exposure data were assessed through the Toxic Release Inventory (TRI) data as published by the Environmental Protection Agency (EPA) and supplemented by exposure simulations using variations of residential exposure times and odour levels and the benzene content of the gasoline. Prevalence rates depended on the size of the population in question. The population size varied greatly between sources, with the more conservative and consistent estimates being reported by the local government and United States Census Bureau and the highest population figure being reported by the Agency for Toxic Substances Disease Registry. The prevalence of Hodgkin’s disease for the residents within 1 mile from the refinery was found to be elevated for every population figure, ranging from 72.11 cases per 100,000 using the ATSDR’s population to 182.34 per 100,000, whereas the prevalence for Hodgkin’s disease in all the United States is only 22 cases of Hodgkin’s disease per 100,000 people. The prevalence value reported in this report should be given greater weight than what would have been calculated using data from the ATSDR. Because of its significantly increased value compared with the rest of the United States, it provides evidence of benzene’s role as a causative agent in the etiology of Hodgkin’s disease. Toxicology and Industrial Health 2008; 24: 683–692.

Key words: benzene; cluster; Hodgkin’s disease; lymphoma; petroleum refinery; prevalence

Introduction

The former American Oil Company (AMOCO) refinery located immediately to the north of the Sugar Creek, Missouri community operated for about 80 years before closing in the early 1980s. The plant has a documented history of benzene-containing gasoline leaks dating back to its first years of operation. The gasoline produced at this refinery that was shown to have leaked into water sources and flowed through the neighbourhood contained 0.7–5% benzene. It is estimated that more than 6 million gallons of unrecovered leachate remains in the ground beneath and surrounding the plant, including the residential community of Sugar Creek (Petty, 2005).

In this benzene-contaminated town, we have discovered a cluster of Hodgkin’s disease among the residents, all of which lived within 1 mile from the refinery. Since 1978, nine residents have been diagnosed with Hodgkin’s disease. Seven are still living. The average age of diagnosis is 26.77 years. The average time between initial exposure to the chemicals emanating from the refinery and diagnosis is...
14.55 years. Five of the seven subjects were exposed in utero, and their average age of diagnosis is only 20.4 years. See Table 1 for profiles of the residents diagnosed with Hodgkin’s disease. The most notable findings on this cluster are a known exposure to chemicals associated with lymphohematopoietic malignancies, the severely high prevalence of Hodgkin’s disease, and the fact that the subjects are generally diagnosed at a much younger age than compared with the rest of the United States.

The production of fatal aplastic anaemia in workers exposed to benzene was originally recognized in the 19th century (Santesson, 1897). Reports of benzene’s carcinogenicity have been accumulating (Selling, 1910) since the 1920s (Boweditch and Elkins, 1939; Hine, 1950; Selling and Osgood, 1935; Wilson, 1942; Winslow, et al., 1926).

In 1948, the American Petroleum Institute (API) published a toxicological review on benzene, wherein the API advised that there were well-documented instances of the development of leukaemia as a result of chronic benzene exposure. The API also recognized that the occurrence of delayed toxic effects, years after the initial exposure, appears likely, and concluded that in as much as the body develops no tolerance to benzene, and as there is a wide variation in individual susceptibility, it is generally considered that the only absolute safe concentration for benzene is zero (API, 1948).

### Table 1. Hodgkin’s disease subject summaries

<table>
<thead>
<tr>
<th>Subject</th>
<th>Date of birth</th>
<th>Date of diagnosis</th>
<th>Age of diagnosis</th>
<th>Pre HD diagnosis (addresses)</th>
<th>Years 1 mile away pre HD diagnosis</th>
<th>Exposed in utero</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7/16/81</td>
<td>11/11/1997</td>
<td>16</td>
<td>11420 Scarrit</td>
<td>3</td>
<td>Yes</td>
<td>Born in SC, moved out in 1984, at age 3</td>
</tr>
<tr>
<td>2</td>
<td>7/12/52</td>
<td>12/96</td>
<td>43</td>
<td>219 N. Huttig</td>
<td>9</td>
<td>No</td>
<td>In 1962, moved to SC at age 10, left in 1971 (9 years)</td>
</tr>
<tr>
<td>3^a</td>
<td>04/04/1964</td>
<td>7/80</td>
<td>26</td>
<td>3001 N. Osage</td>
<td>13</td>
<td>No</td>
<td>In SC from age 5 to 18; diagnosed 8 years later</td>
</tr>
<tr>
<td>4</td>
<td>07/11/1958</td>
<td>10/84</td>
<td>25</td>
<td>301 Marcia St.</td>
<td>20</td>
<td>No</td>
<td>In SC from age 3 to 23 (1961–1981)</td>
</tr>
<tr>
<td>5</td>
<td>7/8/51</td>
<td>02/01/1980</td>
<td>28</td>
<td>10836 Scarrit</td>
<td>25</td>
<td>Yes</td>
<td>In SC for 50 years and was diagnosed age 28</td>
</tr>
<tr>
<td>6^b</td>
<td>8/29/34</td>
<td>10/78</td>
<td>44</td>
<td>221 North Ash</td>
<td>8</td>
<td>No</td>
<td>In SC from 1970 until the time of his death</td>
</tr>
<tr>
<td>7</td>
<td>21/02/1961</td>
<td>8/1979</td>
<td>18</td>
<td>117 Novack</td>
<td>16</td>
<td>Yes</td>
<td>In SC from birth to age 16; was diagnosed 2 years later</td>
</tr>
<tr>
<td>8</td>
<td>26/03/1976</td>
<td>3/6/1997</td>
<td>20</td>
<td>120 N Oxford</td>
<td>21^b</td>
<td>Yes</td>
<td>In SC from birth to age 26; was diagnosed at age 20</td>
</tr>
<tr>
<td>9</td>
<td>08/11/1962</td>
<td>1/1983</td>
<td>20</td>
<td>705 Marcia St</td>
<td>16</td>
<td>Yes</td>
<td>In SC from birth to age 16; was diagnosed 4 years later</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td></td>
<td>26.77</td>
<td></td>
<td>14.55</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

^aDeceased as of 1998 and therefore not factored into 2007 prevalence.

^bDiagnosed just before birthday.

### Methods and materials

#### Exposure to benzene within 1 mile from the AMOCO refinery

We obtained the AMOCO plant’s emissions data from the US Environmental Protection Agency (EPA) – Toxic Release Inventory (TRI) Web site. The US EPA requires that companies estimate the amount of hazardous material they release into the environment beyond the plant’s borders. The EPA TRI data from the AMOCO Plant (Facility Name: BP Products North America Inc. Sugar Creek Terminal; Mailing Address: 1000 N Sterling, Sugar Creek, MO 64054; TRI Facility ID Number: 64054BPMCS1000N; RCRA ID Number: MOD007161425; Latitude 39-07-18.2568 Longitude 94-26-35.2248) notes that thousands of pounds of toxic chemicals, benzene included, have been released into the environment over recent years despite the plant being closed for over a decade (U.S. EPA, 2005).

Exposure estimates for four residents of Sugar Creek were also determined using reports by a Certified Industrial Hygienist (CIH). He calculated exposure estimates and utilized Monte Carlo simulations of benzene levels based on recollections of gasoline odours in air, odour thresholds, and the benzene content of gasoline. The homes of these
four residents are depicted in Figure 1. One of these residents for whom exposure estimates were calculated was diagnosed with Hodgkin’s disease in 1980. The calculations of these residents’ exposures are assumed to be similar to those of the subjects with Hodgkin’s disease, as they all lived within the same air and soil plume area.

Benzene in air concentration was determined by the use of gasoline odour threshold data of Drinker, et al. (1943). Human exposure data from this era was used because no such data are available in more recent years, because of ethical issues of exposure to individuals without their knowledge to chemicals containing known carcinogens.

Based on the Drinker data (1943), the following values were used to determine gasoline vapour concentrations by odour response in modelling an individual’s benzene exposure: With a weak, detectable odour and no physical responses, the gasoline vapour concentration is estimated to be 150 ppm. With a moderate to strong odour and slight to moderate eye irritation, the gasoline vapour concentration is estimated to be 300 ppm. With a strong to very strong odour and moderate eye and throat irritation, the gasoline vapour concentration is estimated to be 600 ppm. Because no gasoline vapour-related physiological response was noted in any of the four cases, a conservative gasoline vapour concentration of 150 ppm was used whenever one reported smelling the odour of gasoline.

The Monte Carlo simulation looks at the effect of varying key parameters to determine their impact on results. To accomplish this, a program called Crystal Ball was utilized. It is an add-on program to Microsoft Excel and is accessed as a custom tool bar.

Variables used for the Monte Carlo Analysis allowed to vary, and the degree to which they were varied include percent time at home (mean ± 5%), percent time smelled gasoline (mean ± 5%), and for

![Figure 1](image-url)  
*Figure 1* Satellite Image of 1 mile buffer zone surrounding plant. Illustrates the residences of the Hodgkin’s subjects in reference to the AMOCO refinery and the zero to low population density in the area north of the refinery.
a gasoline concentration of 150 ppm the range used was 100–200 ppm. Benzene percentage in gasoline for means of 2.75%, range used was 0.5–5%. Each range of values was considered to be distributed normally. The simulation was run 10,000 times.

**Prevalence of Hodgkin’s disease in Sugar Creek**

The current value for the prevalence of Hodgkin’s disease in Sugar Creek, MO, depends on how the population is determined. The 2000 census obtained a population of 3,839 residents (Census, 2000). The Sugar Creek city clerk estimated the town’s population to be no greater than 4,000 residents, a value ascertained during a phone conversation on 23 July 2007 (personal communication with city clerk, Jana Oliverez-Dickenson). The Agency for Toxic Substances and Disease Registry (ATSDR) estimated the population to be 9,708 (ATSDR AmocoSugarCreek092404-HC). Upon scrutiny however, this is a very unrealistic value. Instead of using the 3,839 census value for the population of Sugar Creek made available to them, the ATSDR used the population density value in the same census report and multiplied that value by the area within a 1 mile radius of the AMOCO plant. What they neglected to factor in is that only half of the area is residential while the latter half is farmland. The satellite images of the area provided by Google maps confirm this low or no density value. See Figure 1. Therefore the value the ATSDR should have reported is half of their 9,708 value, which equals 4,854. Although this is still 25% greater than the 2000 census, it more closely approximates the other population values within 1 mile from the AMOCO plant.

Finally, in an effort to check all of these numbers, we used Google Maps to obtain a highly detailed satellite image of the Sugar Creek community and counted by hand the number of homes within a 1 mile radius of the plant. Any structure on the map that was not obviously a public enterprise was counted as a home. We multiplied this value by the average number of persons per US household (2.4) and estimated the population to be 5,318 within 1 mile from the plant, as we counted 2,216 homes. Based on the 2003 census and the conversation with the city clerk, this figure, although clearly an overestimate for the town of Sugar Creek, better represents the population within 1 mile from the AMOCO refinery than the value reported by the ATSDR. As stated above, the town of Sugar Creek’s population according to the 2000 Census and the city clerk is approximately 4,000 residents. To account for the difference in populations between these figures and that by counting via satellite is the explanation that approximately 550 homes (1,300 residents) fall outside of Sugar Creek’s official border. These homes are south of RT 24 Independence Avenue. One limit of our technique is that we did not factor in the possibility of apartment complexes, which generally hold more residents than the average household, but by the same token counted properties with possible separate garages and guest homes as two homes as opposed to one.

The population used in the calculations of prevalence of Hodgkin’s disease was the average of the populations reported in the 2000 census, city hall’s highest estimate, half the ATSDR value, and the estimated population from the satellite image estimation. The ATSDR value of 9,708 was ignored. The population used therefore in the calculations is the average of 3839, 4000, 4854 and 5318, which equals an estimated 4502.75 people within 1 mile from the refinery.

The prevalence was determined for each population value along with a prevalence for the average of the population values. Values were then broken down by age. Given that seven still living residents of Sugar Creek in 2007 have been diagnosed with Hodgkin’s disease, the following equation for prevalence used was:

\[
\text{Prevalence} = \frac{(7 \div N)}{100,000}, \text{ where } N = 4502.75 \text{ people.}
\]

Statistical analysis was performed using Stata 9.0 Statistical Software.

**Results**

**Exposure to benzene within 1 mile from the AMOCO refinery**

**TRI data**

Based on the data published on the EPA-TRI website, the AMOCO facility’s average for total on- and off-site disposal or other releases for benzene was in excess of 586 pounds of benzene per year with a range of 351–960 pounds per year.

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Based on the data published on the EPA-TRI website, the AMOCO facility’s average for total on- and off-site disposal or other releases for benzene was in excess of 586 pounds of benzene per year with a range of 351–960 pounds per year.
It is important to note the following points when analyzing the data:

- The EPA-TRI exposure data for the AMOCO refinery in Sugar Creek is not available for the years before 1997 and after 2005 online.
- The exposure data that are available is based on company reports and are not validated in any way.
- The AMOCO-refinery has not been in operation since the early 1980s so all emissions are from residual waste, either underground or still inside storage vessels, not removed following cessation of operations.

Supplemental exposure calculations
Detailed historic benzene exposure assessments were completed for four residents of Sugar Creek. In these cases, inhalation exposure in ppm-years were determined. This summary provides background information and an overview of results of these exposure assessments. These exposures are listed in Table 2 and were based ultimately on the time gasoline odours were smelled, gasoline odour threshold levels, and the benzene content of gasoline.

Prevalence of Hodgkin’s disease in sugar creek
The prevalence was determined to be 155.46 cases of Hodgkin’s disease per 100,000 people. Given that the prevalence of Hodgkin’s disease in the United States is 22 per 100,000 among whites (SEER, 2003), the value of 155.46 exceeds the prevalence of a generally non-overexposed population by a multiple of greater than seven. Using the US prevalence all ages of 22 and Sugar Creek prevalence of 155.49 a test for the difference in the rates yields a p-value = 0.034 which is significant at the 5% level. A 95% (Poisson Exact) confidence interval for Sugar Creek prevalence rate is (62.5, 320.4).

For the subjects diagnosed with Hodgkin’s disease, the average age of diagnosis among the Sugar Creek residents is 26.77 years with a range of 16 to 45 years (see table 1). All of the subjects with Hodgkin’s disease lived within one mile of the AMOCO plant for an average of 14.55 years before the onset of their respective Hodgkin’s lymphomas, with a range of 3 to 25 years.

Discussion
With regard to exposure, benzene levels were still elevated in the late 1990s and 2000s in Sugar Creek despite the plant being closed since the early 1980s. The main source of benzene in Sugar Creek therefore could be due to off-gassing of the 6 million gallons of unrecovered gasoline leachate in the soil.

Table 2. Background and exposure histories of four residents of Sugar Creek for whom exposures were calculated

<table>
<thead>
<tr>
<th>Case #</th>
<th>Exposure time interval</th>
<th>Address</th>
<th>Exposure time (years)</th>
<th>Monte carlo range of exposure (inhalation)</th>
<th>Total Exposure (ppm-yrs)</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>1981–1985</td>
<td>101 N Brent</td>
<td>4</td>
<td>27.2–45.8</td>
<td>33.3</td>
<td>Leukemia</td>
</tr>
<tr>
<td>4</td>
<td>1951–1980</td>
<td>1110 Scarritt</td>
<td>29</td>
<td>47.0–163.8</td>
<td>102</td>
<td>Hodgkin’s Lymphoma</td>
</tr>
</tbody>
</table>

Table 3. Benzene metabolites and lymphohaematopoietic response

<table>
<thead>
<tr>
<th>Source</th>
<th>Metabolite</th>
<th>Lymphohaematopoietic response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kolachana, 1993</td>
<td>Phenol</td>
<td>Induced oxidative stress to DNA in bone marrow</td>
</tr>
<tr>
<td>Pellack-Walker and Blumer, 1986</td>
<td>Benzoquinone</td>
<td>Produced breaks in DNA</td>
</tr>
<tr>
<td>Robertson, et al., 1990</td>
<td>Catechol and Hydroquinone</td>
<td>Produced a synergistic genotoxic effect in human lymphocytes</td>
</tr>
<tr>
<td>Wierda and Irons, 1982</td>
<td>Catechol and Hydroquinone</td>
<td>Selectively inhibited the maturation of LPS-activated marrow progenitors into end-stage PC-PFC</td>
</tr>
<tr>
<td>Witz, et al. (1989)</td>
<td>Trans, trans- muconaldehyde</td>
<td>Results in cell membrane changes, including loss of activity in NADPH-dependent oxidase and decreased in membrane lipid fluidity</td>
</tr>
<tr>
<td>Zhang, et al., 1993</td>
<td>1, 2, 4 Benzenetriol</td>
<td>Causes strand breaks, microtubule damage, micronuclei, and aneuploidy in human cells</td>
</tr>
<tr>
<td>Zhang, et al., 2005</td>
<td>Hydroquinone and benzenetriol</td>
<td>Affect the ploidy status of specific chromosomes</td>
</tr>
</tbody>
</table>
Blood disorders, such as Hodgkin’s disease, are extremely well documented in Gist and Burg’s review of benzene toxicity, citing the toxin as a ‘known haematopoietic poison’. They show the earliest known blood disorders in response to benzene exposure were reported in 1897 (Santesson, 1897). The more recent literature they referenced provides unequivocal evidence for benzene’s ability to damage the human haematologic and lymphatic systems (Gist and Burg, 1997).

Occupational exposures to benzene have been associated with increased risks of Hodgkin’s disease. For example, Aksoy reports 6 out of 94 patients admitted to the 2nd International Clinic of Istanbul Medical School with Hodgkin’s disease had worked with benzene. Their exposure to benzene was in the range of 150–210 ppm with a mean duration of 11 years. “The fact that 6 of 94 patients with Hodgkin’s disease have a history of chronic benzene exposure suggests the possibility of a causal relationship between Hodgkin’s disease and chronic benzene exposure” (emphasized in the article) (Aksoy, et al., 1974). Increased lymphoma occurrence was consistent with earlier studies, which showed 23.5 percent of a group of 217 healthy, benzene exposed workers experienced significant haematologic alterations (Aksoy, et al., 1971). Aksoy then reviews three additional cases of malignant lymphoma associated with chronic exposure to benzene. One of the three patients had Hodgkin’s disease (Aksoy, 1980).

The relative risk in a study by Olsson for Hodgkin’s disease was 6.6 ($P = 0.0005$) in benzene exposed workers (Olsson and Brandt, 1980). Persson reported occupational exposure to organic solvents showed a significant increased risk for Hodgkin’s disease (OR 3.4) (Persson, et al., 1993). Olin reported an increased occurrence of Hodgkin’s disease among chemistry graduate students who routinely used benzene as a solvent in the laboratory (Olin, 1976). Vianna reported a significant excess of deaths caused by lymphomas in men employed in occupations where benzene and/or coal tar fractions are used. The relative risk for Hodgkin’s disease was 1.6 (Vianna and Polan, 1979).

Hardell reported a statistically significant increase in relative risk for malignant lymphomas among workers exposed to benzene or to specific solvents such as trichloroethylene in Sweden,
the etiology of Hodgkin's disease, have pointed to environmental factors in unspecified organic solvents, RR = 2.8 (CI = 95%; 1.6–4.8). Hardell also found a dose response; the higher the exposure the greater the risk. These researchers implicated benzene as a causative factor for malignant lymphoma (Hardell, et al., 1981).

Brandt 1987 reviewed lymphomas associated with solvents, benzene in particular, and noted an odds ratio of 2.8–6.6 for Hodgkin's disease (Brandt, 1987). This article reports on several other studies documenting a significant association between benzene and an increased occurrence of Hodgkin's disease.

La Vecchia found evidence of occupational exposure to chemicals, especially benzene, being related to the risk of Hodgkin's disease as they report increased percentages of cases of Hodgkin's disease for workers exposed to solvents, benzene in particular and calculated a significantly increased relative risk of 1.9 (95% CI = 1.2–3.0). (La Vecchia, et al., 1989).

Berlin reports an elevated standard incidence ratio (SIR) of 6.3 (CI = 95%; 0.8–22.7) for a diagnosis of Hodgkin's lymphoma among Swedish workers with a suspected solvent-related disorder (Berlin, et al., 1995).

Yin compared 74,828 benzene exposed to 35,835 non-exposed workers in China in a study ‘designed specifically to avoid the disadvantages of earlier investigations’. Its extremely large cohort provides statistics of the highest reliability by eliminating differences between the case and control groups and also minimizes the healthy worker effect. Yin reports more than a doubling of the relative risk of all haematolymphoproliferative malignancies RR = 2.6 (CI = 95%; 1.5–5.0) and more than a tripling of malignant lymphoma RR = 3.5 (CI = 95%; 1.2–14.9) (Yin, et al., 1996).

Two industry sponsored studies by Divine and Tsai both found elevated standard mortality risks (SMRs) for Hodgkin’s disease, 108 and 199, respectively (Divine, et al., 1985; Tsai, et al., 1993).

A number of articles, not limited to occupational studies, have pointed to environmental factors in the etiology of Hodgkin’s disease. Dworsky described a clustering of Hodgkin’s disease in 5 graduates of the same junior high school (Dworsky and Henderson, 1974). Glaser reports on a Hodgkin’s cluster in San Francisco and writes, “the small, widely dispersed clusters detected here suggest late exposure to a ubiquitous environmental agent involved in the Hodgkin’s disease etiology” (Glaser, 1990). Wilkinson found a positive association between risk of Hodgkin’s lymphoma and proximity to a major petrochemical industry (Wilkinson, et al., 1999).

Raaschou-Nielsen found the risk of lymphomas increased in a dose-dependent manner for air pollution at the residence at the time the child was in utero. When measured against benzene levels in particular, the relative risk was 4.3 (CI = 95% 1.5–12.4) (Raaschou-Nielsen, et al., 2001). This study agrees with previous animal studies in which foetuses and offspring of benzene exposed pregnant mice showed long-term functional changes in haematopoiesis (Keller and Snyder, 1986, 1988, Schardein and Keller, 1989). Other animal studies show effects to haematopoietic stem cells and leukocytes of the exposed subjects (Cronkite, et al., 1982, 1985).

Researchers hypothesized Hodgkin’s disease represented an unusual response to a common environmental agent in individuals with a susceptible immune system (Dworsky and Henderson, 1974). Benzene or a benzene metabolite has reproducible effects on the lymphatic system and in particular on the B lymphocytes that give rise to the cancer cells that are the relevant cell type in Hodgkin’s disease, Reed-Sternberg cells (Aoyama, 1986; Lee, et al., 2005; Young, 1989). Table 4 is an incomplete summary of the literature with regard to the toxicity of several key benzene metabolites. It should be noted that there is a synergistic effect between combinations of metabolites, whereby the toxicity of one is enhanced by the presence of another (Barale, et al., 1990).

Through a series of enzymatic and non-enzymatic chemical reactions having been shown to begin mainly in the liver and then continue in bone marrow, benzene and its metabolites ultimately reach the multipotent stem-cells that subsequently differentiate into the mutated B-lymphocytes that define Hodgkin’s disease. The lymphohaematopoietic system has been shown to be particularly susceptible because benzene and benzene metabolites phenol, catechol and hydroquinone levels are higher in the bone marrow than in other tissues (Rickert, et al., 1979). In addition, the
metabolites of benzene, hydroquinone and catechol, have also been shown to reduce the number of bone
marrow and spleen progenitor B lymphocytes, another piece of evidence documenting benzene’s del-
eterious effect on the lymphohaematopoietic system
(Hseigh, et al., 1990; Wierda and Irons, 1982).

Snyder describes the metabolic pathways for
benzene. One pathway involves the metabolic acti-
vation of benzene to species that covalently bind to
DNA, which produces mutagenic events that are
expressed as malignant lymphoma. The other path-
way following metabolic activation involves oxida-
tive stress, subsequent oxidative damage to DNA,
and a mutagenic effect. Both pathways can cause
cellular change that lead to Hodgkin’s lymphoma
(Snyder and Hedli, 1996).

In conclusion, we have reported on a severely
increased prevalence rate of Hodgkin’s disease
among residents with a known exposure to ben-
zene, which most likely results from off-gassing of
the millions of gallons of benzene known to have
leaked from the refinery during and after its years
of operation. In determining the prevalence rate of
Hodgkin’s disease, our methods are different than
those that would have been calculated based on the
ATSDR’s figures. The dichotomy in reasoning
appears to lie in the differing values used to repre-
sent the relevant population. Despite the inflated
population figure reported by the ATSDR, an
increased prevalence was found for all figures
representing the population and there was a shift
in diagnosis toward a younger age. We believe
our methodology is more accurate because the
ATSDR made an assumption on population den-
sity in an area that is known to have zero or close
to zero inhabitants (see Figure 1). The prevalence
value reported in this report should therefore be
given greater weight than what would have been
calculated using data from the ATSDR and
because of its significantly increased value com-
pared to the rest of the United States provides evi-
dence of benzene’s role as a causative agent in the
etiology on Hodgkin’s disease.

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