Childhood Lymphohematopoietic Cancer Incidence and Hazardous Air Pollutants in Southeast Texas, 1995–2004

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BACKGROUND: Cancer is the second leading cause of death among U.S. children with few known risk factors. There is increasing interest in the role of air pollutants, including benzene and 1,3-butadiene, in the etiology of childhood cancers.

OBJECTIVE: Our goal was to assess whether census tracts with the highest benzene or 1,3-butadiene ambient air levels have increased lymphohematopoietic cancer incidence.

METHODS: Our ecologic analysis included 977 cases of childhood lymphohematopoietic cancer diagnosed from 1995–2004. We obtained the U.S. Environmental Protection Agency’s 1999 modeled estimates of benzene and 1,3-butadiene for 886 census tracts surrounding Houston, Texas. We ran Poisson regression models by pollutant to explore the associations between pollutant levels and census-tract cancer rates. We adjusted models for age, sex, race/ethnicity, and community-level socioeconomic status (cSES).

RESULTS: Census tracts with the highest benzene levels had elevated rates of all leukemia [rate ratio (RR) = 1.37; 95% confidence interval (CI), 1.05–1.78]. This association was higher for acute myeloid leukemia (AML) (RR = 2.02; 95% CI, 1.03–3.96) than for acute lymphocytic leukemia (ALL) (RR = 1.24; 95% CI, 0.92–1.66). Among census tracts with the highest 1,3-butadiene levels, we observed RRs of 1.40 (95% CI, 1.07–1.81), 1.68 (95% CI, 0.84–3.35), and 1.32 (95% CI, 0.98–1.77) for all leukemia, AML, and ALL, respectively. We detected no associations between benzene or 1,3-butadiene levels and lymphoma incidence. Results that examined joint exposure to benzene and 1,3-butadiene were similar to those that examined each pollutant separately.

CONCLUSIONS: Our ecologic analysis suggests an association between childhood leukemia and hazardous air pollution; further research using more sophisticated methodology is warranted.

KEY WORDS: 1,3-butadiene, air toxics, benzene, childhood cancer, epidemiology, hazardous air pollution.
and mobile sources of exposure to benzene and 1,3-butadiene, Harris and surrounding counties provided an ideal location to further study potential childhood cancer risks associated with levels of benzene and 1,3-butadiene in ambient air.

**Materials and Methods**

**Study population.** We identified 997 cases of lymphohematopoietic cancer among children < 20 years of age from the Texas Cancer Registry (TCR), a North American Association of Central Cancer Registries (2007) gold-certified population-based registry. All cases were diagnosed between 1995 and 2004 and resided in one of the following eight counties surrounding Houston, Texas, at the time of diagnosis: Harris, Montgomery, Liberty, Chambers, Fort Bend, Brazoria, Waller, and Galveston. The specific lymphohematopoietic cancers identified were leukemia [International Classification of Diseases, 10th Revision (ICD-10) (World Health Organization 1993) codes C91–C95; C96–C97, 10] (World Health Organization 1993) codes.

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exposure variable, we ranked census tracts in the highest quartile for both 1,3-butadiene and benzene levels as "high," and those in the lowest quartile as "low," which served as the referent category. We assigned all other census tracts to the middle category. Using this joint exposure variable, we reran analyses for all cancer subtypes. We completed all analyses in SAS (version 9.1; SAS Institute Inc., Cary, NC).

Results

Table 1 presents the number of cases of each type of lymphohematopoietic cancer included in our study by age, sex, race/ethnicity, and cSES. Cases of ALL and AML were diagnosed at younger ages, whereas Hodgkin’s disease cases were generally diagnosed at older ages. Overall, there were a higher proportion of male than female cases. A greater proportion of ALL and AML cases were Hispanic, whereas most Hodgkin’s disease and NHL cases were non-Hispanic white. Additionally, regardless of cancer type, our population had a higher proportion of cases in the highest quartile of cSES.

Table 2 presents the distribution of the U.S. EPA ASPEN model estimates for benzene and 1,3-butadiene in the study area. Across the 886 census tracts in our study area, ambient air levels ranged from 0.42 to 9.05 µg/m³ for benzene and from 0.01 to 2.41 µg/m³ for 1,3-butadiene. The median benzene level in the eight-county study area (1.72 µg/m³) is approximately 10 times the median level of 1,3-butadiene (0.16 µg/m³).

We found that census tracts with the highest ambient air levels of benzene had elevated rates of all leukemia (RR = 1.37; 95% CI, 1.05–1.78) and AML (RR = 2.02; 95% CI, 1.03–3.96) compared with census tracts with the lowest levels of 1,3-butadiene. We found a borderline significant trend between rates of ALL (p = 0.06) and 1,3-butadiene levels. We observed no statistically significant trend between rates of AML and 1,3-butadiene levels. We detected no association between estimated 1,3-butadiene levels and incidence of Hodgkin’s disease or NHL.

In examining the effect of cocontaminant exposure to benzene and 1,3-butadiene, for each specific cancer type examined, the estimated RRs comparing the highest exposure group to the lowest were similar in magnitude to the results from the analyses of the independent effects of benzene and 1,3-butadiene (data not shown). However, only the point estimate for all leukemias remained statistically significant.

Additionally, these census tracts had 1.24 (95% CI, 0.92–1.66) times the rate of ALL compared with census tracts with the lowest levels, although this estimate was not statistically significant. We detected a statistically significant trend of increasing incidence rates with increasing estimated levels of benzene for all leukemias combined (p = 0.03) and a borderline significant trend for AML (p = 0.06). We observed no trend for ALL. No associations were observed between benzene levels in ambient air and Hodgkin’s disease or NHL.

Relative to census tracts with the lowest levels, we observed significantly increased rates of all leukemia among census tracts with the highest levels of ambient 1,3-butadiene (RR = 1.40; 95% CI, 1.07–1.81) as well as a significant trend (p = 0.01) (Table 4). We also observed 32% (RR = 1.32; 95% CI, 0.98–1.77) and 68% (RR = 1.68; 95% CI, 0.84–3.35) higher (albeit insignificant) rates of ALL and AML, respectively; in these census tracts compared with census tracts with the lowest levels of 1,3-butadiene. We found a borderline significant trend between rates of ALL (p = 0.06) and 1,3-butadiene levels. We observed no statistically significant trend between rates of AML and 1,3-butadiene levels. We detected no association between estimated 1,3-butadiene levels and incidence of Hodgkin’s disease or NHL.

Discussion

We found significantly higher rates of leukemia in census tracts with the highest ambient levels of benzene and 1,3-butadiene, as estimated from the U.S. EPA ASPEN model for 1999 (U.S. EPA 2007). We also observed elevated rates of the two most common types of childhood leukemia, ALL and AML, associated with benzene levels. We detected similar results for 1,3-butadiene, although these results were not statistically significant. Our results are consistent with the only other study to have examined childhood cancer rates with ambient air levels of HAPs (Reynolds et al. 2003). Similar to our investigation, Reynolds et al. (2003) used the U.S. EPA ASPEN model for 25 HAP’s to create a single exposure score; they did not, however, examine rates in relation to individual pollutants. Reynolds et al. (2003) reported significantly higher rates of childhood leukemia (RR = 1.21; 95% CI, 1.03–1.42) and increased rates of ALL and AML (ALL: RR = 1.19; 95% CI, 1.00–1.43; AML: RR = 1.46; 95% CI, 0.97–2.19) among census tracts with the highest HAP exposure score.

The few other studies that have explored the potential association between childhood cancer and air pollution have produced equivocal results. Of four studies reporting statistically significant positive results, three used measures of traffic density (Crosignani et al. 2004; Nordlinger and Jarvholm 1997; Pearson et al. 2000), and one study examined residential distance from roadways (Knox 2006) to assess exposure. In contrast, two case–control studies found no association between traffic density and childhood leukemia (Reynolds et al. 2002; Steffen et al. 2004), and one case–control study found no significant risk of leukemia associated with residence within 100 m of either a main road or a gas station (Harrison et al. 1999).

Our study has the benefit of being population based and not subject to selection bias. With more than a decade of cancer incidence data for the greater Houston area, we also had sufficient power to evaluate risk associated with...
HAP exposure. Additionally, we chose to study childhood cancers, which have a shorter latency period than adult cancers and therefore present fewer methodologic challenges when studying environmental exposures. We used an innovative method of assigning cases with “difficult” or “nongeocodable” addresses, most of which were post office boxes, to census tracts by “matching” them to another case with similar demographic characteristics and reporting the same residential ZIP code. This method of assignment is an improvement over discarding data for subjects for whom an exact match cannot be made or by assigning these cases to the ZIP code centroid, which may create an artificial cluster of cases in one location and result in nondifferential exposure misclassification (Hurley et al. 2003).

A limitation of our study is the potential for an ecologic fallacy, which may result when using grouped versus individual-level data. Nonetheless, an ecologic study design is efficient when little is known about the association under study (Friis and Sellers 2004), as is the case for childhood cancer and HAPs. Moreover, the smaller the geographic unit of analysis, the more likely bias resulting from aggregation will be minimized (Diggle and Elliott 1995). We chose to analyze data at the census tract level, which we felt to be the smallest possible spatial resolution at which to aggregate without sacrificing so much power that we could no longer detect an association. Because of the aggregate nature of our composite measure of socio-economic status, there was likely misclassification of this variable. Also, the U.S. Census does not provide intercensal estimates at the subcounty level. Moreover, in our study, it was necessary to have population totals by race/ethnicity, age, and sex for each census tract (32 groups in total), and the accuracy of population projections for small areas has been called into question (Smith and Shahidullah 1995). Given these difficulties, we chose to develop our 10-year population projections for small areas using the 2000 U.S. Census (U.S. Census Bureau 2006) even though we recognize that there was likely intercensal variability in population growth or decline during this period.

We based our exposure assignment on address at diagnosis, which may not represent the address at which the case or case mother resided during the prenatal or postnatal periods, which may be etiologically more relevant. Few studies have examined the degree of residential stability in epidemiologic investigations (Canfield et al. 2006; Hurley et al. 2003), and we are aware of none that have examined this issue in studies of childhood cancer. To examine possible effects of exposure misclassification due to residential mobility, we ran sensitivity analyses restricting cases to those diagnosed at ages < 5 years. For NHL, which had a majority of cases diagnosed at later ages, we found increased rates compared with the analyses including children of all ages (benzene: RR = 1.49; 95% CI, 2.04–4.54; 1,3-butadiene: RR = 1.61; 95% CI, 0.53–4.90). We also repeated the sensitivity analysis for all leukemias and each subtype (there were too few cases of Hodgkin’s disease diagnosed before 5 years of age to analyze) and found results similar to those presented among all children. These results indicate that mobility is likely not as large a problem for childhood cancers that are diagnosed earlier, such as leukemia, compared with those that are diagnosed at later ages (e.g., NHL).

The ASPEN data provide the advantage of a complex dispersion modeling approach using myriad information about sources and fate and transport of pollutants. The ASPEN model accounts for multiple factors that influence HAP levels in the environment (e.g., meteorologic conditions, emission height, deposition, and secondary formation) and represents a more robust measure of potential exposure than has previously been used in studies of childhood cancer and HAPs. Despite this advantage, our exposure assessment is subject to error. Limitations of the modeling approach include uncertainties in the emissions inventory and the inherent inability to capture variability within a census tract (Ozkaynak et al. 2008). Additionally, we relied upon levels of benzene and 1,3-butadiene in ambient air in 1999. Because cases were diagnosed in 1995–2004, the etiologic window of exposure varied over calendar time, so some misclassification of exposure is likely. On the one hand, data for a single year in the midpoint of this

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### Table 3. Adjusted RR for the association between 1999 U.S. EPA ASPEN modeled estimates of ambient benzene levels and lymphohematopoietic cancer incidence among children (<20 years of age).

<table>
<thead>
<tr>
<th>Cancer type</th>
<th>Benzene level</th>
<th>No. of census tracts</th>
<th>No. of cases</th>
<th>Adjusted RR (95% CI)</th>
<th>p-Value</th>
<th>p-Value for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hodgkin’s disease</td>
<td>Low</td>
<td>221</td>
<td>36</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>222</td>
<td>42</td>
<td>1.17 (0.74–1.85)</td>
<td>0.500</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>31</td>
<td>1.07 (0.66–1.72)</td>
<td>0.789</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>222</td>
<td>37</td>
<td>1.52 (0.92–2.52)</td>
<td>0.101</td>
<td>0.163</td>
</tr>
<tr>
<td>NHL</td>
<td>Low</td>
<td>221</td>
<td>41</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>222</td>
<td>40</td>
<td>0.96 (0.62–1.48)</td>
<td>0.853</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>27</td>
<td>0.84 (0.52–1.36)</td>
<td>0.484</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>222</td>
<td>28</td>
<td>1.06 (0.60–1.87)</td>
<td>0.852</td>
<td>0.936</td>
</tr>
<tr>
<td>All leukemia</td>
<td>Low</td>
<td>221</td>
<td>157</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>222</td>
<td>183</td>
<td>1.13 (0.91–1.40)</td>
<td>0.269</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>156</td>
<td>1.10 (0.87–1.40)</td>
<td>0.415</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>222</td>
<td>174</td>
<td>1.37 (1.05–1.78)</td>
<td>0.019</td>
<td>0.034</td>
</tr>
<tr>
<td>ALL</td>
<td>Low</td>
<td>221</td>
<td>116</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>222</td>
<td>147</td>
<td>1.23 (0.97–1.56)</td>
<td>0.092</td>
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</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>123</td>
<td>1.14 (0.87–1.49)</td>
<td>0.349</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>222</td>
<td>124</td>
<td>1.24 (0.92–1.66)</td>
<td>0.153</td>
<td>0.186</td>
</tr>
<tr>
<td>AML</td>
<td>Low</td>
<td>221</td>
<td>21</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>222</td>
<td>20</td>
<td>0.93 (0.50–1.72)</td>
<td>0.813</td>
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</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>20</td>
<td>1.11 (0.59–2.10)</td>
<td>0.747</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>222</td>
<td>31</td>
<td>2.02 (1.03–3.96)</td>
<td>0.040</td>
<td>0.063</td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, race/ethnicity, and cSES.

### Table 4. Adjusted RR for the association between 1999 U.S. EPA ASPEN modeled estimates of ambient 1,3-butadiene levels and lymphohematopoietic cancer incidence among children (<20 years of age).

<table>
<thead>
<tr>
<th>Cancer type</th>
<th>1,3-Butadiene level</th>
<th>No. of census tracts</th>
<th>No. of cases</th>
<th>Adjusted RR (95% CI)</th>
<th>p-Value</th>
<th>p-Value for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hodgkin’s disease</td>
<td>Low</td>
<td>221</td>
<td>36</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>221</td>
<td>43</td>
<td>1.16 (0.73–1.82)</td>
<td>0.530</td>
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</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>32</td>
<td>1.10 (0.69–1.76)</td>
<td>0.694</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>223</td>
<td>35</td>
<td>1.41 (0.85–2.34)</td>
<td>0.186</td>
<td>0.236</td>
</tr>
<tr>
<td>NHL</td>
<td>Low</td>
<td>221</td>
<td>42</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>222</td>
<td>36</td>
<td>0.85 (0.54–1.34)</td>
<td>0.485</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>33</td>
<td>1.01 (0.63–1.61)</td>
<td>0.965</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>222</td>
<td>26</td>
<td>0.99 (0.57–1.71)</td>
<td>0.960</td>
<td>0.986</td>
</tr>
<tr>
<td>All leukemia</td>
<td>Low</td>
<td>221</td>
<td>147</td>
<td>1.00</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>222</td>
<td>164</td>
<td>1.22 (0.98–1.52)</td>
<td>0.081</td>
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</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>172</td>
<td>1.25 (0.99–1.58)</td>
<td>0.066</td>
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</tr>
<tr>
<td></td>
<td>High</td>
<td>223</td>
<td>167</td>
<td>1.40 (1.07–1.81)</td>
<td>0.013</td>
<td>0.013</td>
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<tr>
<td>ALL</td>
<td>Low</td>
<td>221</td>
<td>108</td>
<td>1.00</td>
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</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>221</td>
<td>145</td>
<td>1.31 (1.02–1.68)</td>
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<tr>
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<td>Medium-high</td>
<td>221</td>
<td>136</td>
<td>1.31 (1.00–1.71)</td>
<td>0.052</td>
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<tr>
<td></td>
<td>High</td>
<td>223</td>
<td>121</td>
<td>1.32 (0.98–1.77)</td>
<td>0.064</td>
<td>0.056</td>
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<tr>
<td>AML</td>
<td>Low</td>
<td>221</td>
<td>20</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium-low</td>
<td>222</td>
<td>23</td>
<td>1.12 (0.61–2.06)</td>
<td>0.721</td>
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</tr>
<tr>
<td></td>
<td>Medium-high</td>
<td>221</td>
<td>22</td>
<td>1.20 (0.64–2.27)</td>
<td>0.570</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>223</td>
<td>27</td>
<td>1.68 (0.84–3.35)</td>
<td>0.142</td>
<td>0.163</td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, race/ethnicity, and cSES.
interval may be suitable surrogates for levels over longer periods because the relative ranking of geographic areas of high versus low ambient air pollutant levels (because of their proximity to roadways and point sources) likely remains the same. On the other hand, significant changes in air pollutant emissions due, for example, to the opening or closing of a large industrial facility during the study period could potentially affect the relative ranking of census tracts and result in some misclassification.

Because the ASPEN data provide levels of HAPs in the outdoor environment, data regarding levels in the home or other indoor environments were not available. Because children tend to spend a great deal of time indoors (Adgate et al. 2004), indoor sources of benzene and 1,3-butadiene, such as environmental tobacco smoke, may be a significant contributor to their personal exposure. However, parental smoking has been weakly and inconsistently associated with childhood leukemias in the epidemiologic literature (Belson et al. 2007). Nonetheless, we attempted to control for smoking by creating a census-tract–level variable using information from county-level rates on smoking by ethnicity, available from the Texas Department of State Health Service’s Behavioral Risk Factor Surveillance System (Texas Department of State Health Service 2005). However, effects associated with air pollutant levels were essentially unchanged with the inclusion of this variable in the model (data not shown), and we chose to report on the results from the more parsimonious model.

Because of the high correlation between estimated ambient air levels of benzene and 1,3-butadiene, we were limited in our ability to tease apart the effects of each pollutant. We attempted to address this issue by creating a single variable representing their joint effect. It is of interest that the comparison of census tracts with high levels of both benzene and 1,3-butadiene levels to census tracts with low levels of both pollutants resulted in RRs similar to, although more imprecise than, those produced by separate analyses of each pollutant. These results suggest that childhood leukemia risk may be related to one, but likely not both, pollutants. Given the overwhelming evidence of the carcinogenic potential of benzene, we therefore cannot exclude the possibility that the associations we observed between childhood cancer and 1,3-butadiene were actually attributable to benzene, and more research is needed to elucidate the cancer risks arising from complex air pollutant mixtures.

Conclusion

Our exploratory analysis suggests that estimated ambient levels of benzene and 1,3-butadiene may contribute to increased rates of childhood leukemia in census tracts with the highest estimated levels. To our knowledge, this is the first epidemiologic study to have examined childhood cancer incidence rates associated with these two HAPs in Texas. Although data from the U.S. EPA ASPEN model provide a unique source of potential exposure data for researchers interested in the health effects of air pollution, and it is a logical source of data for exploratory analyses, they represent modeled levels rather than actual measured concentrations of HAPs and carry with them some limitations, as discussed above. Given that Houston, Texas, is one of the most densely monitored cities in the nation (Texas Commission on Environmental Quality 2005), work is under way to apply spatial analysis techniques to the existing monitoring data to further explore associations between childhood lymphohematopoietic cancers and hazardous air pollution.

References


