

International Journal of Global Health and Health Disparities

Volume 4 | Number 1

Article 6

2005

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Recommended Citation

Hill, Sara Jo (2005) "Childhood Cancer Rates and Exposure to Vehicle Pollutants," *International Journal of Global Health and Health Disparities*: Vol. 4 : No. 1 , Article 6.

Available at: <http://scholarworks.uni.edu/ijghhd/vol4/iss1/6>

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CHILDHOOD CANCER RATES AND EXPOSURE TO VEHICLE POLLUTANTS

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Cancer is the second leading cause of death among children ages zero to 15 in the United States (Gouveia-Vigeant & Tickner, 2003, p. 1). Rates for childhood cancer have increased nearly 21 percent between 1975 and 1998 (Gouveia-Vigeant & Tickner, p. 1). Multiple factors can be attributed to this increase. Possible explanations include: increased surveillance, changes in diagnostic criteria, improvements in medical imaging and diagnostic techniques, genetic predisposition as well as environmental factors. Experts recently concluded that genetic predisposition accounts for no more than 20 percent of all childhood cancers (Gouveia-Vigeant & Tickner, p. 1). While this demonstrates a significant contribution, it leaves the majority of cancer etiology unexplained.

Environmental factors have been proposed to cause anywhere from five to 90 percent of newly diagnosed pediatric cancer cases depending on the specific type of cancer (Gouveia-Vigeant & Tickner, p. 1). A series of reports have shown a significant associa-

tion between elevated cancer risk and exposure to vehicle exhaust (Crosignani, Tittarelli, Borgini et al., 2004; Feychting, Svensson, & Ahlbom, 1998; Knox & Gilman, 1997; Pearson, Wachtel, & Ebi, 2000; Savitz & Feingold, 1989). Some of the factors evaluated in these studies included exposure to traffic density and estimated nitrogen dioxide concentration in outdoor air. The assumption in these studies was that traffic density is correlated with the volume of petrochemical combustion byproducts, including benzene and nitrogen dioxide. Researchers in these studies concluded that clusters of childhood cancers within areas of high vehicular pollution must indicate a causal relationship.

Subsequent researchers have not been as eager to accept these results as conclusory, citing the small scale of the studies and pointing out potential errors in experimental design that may have led to false conclusions that hazardous air pollutants (HAPs) are causally linked to childhood cancer rates. After taking into account confounding variables and increasing sample size, subsequent reports have failed to demonstrate an association between cancer rates and traffic pollutants. For example, one study examined the association between nitrogen dioxide and benzene concentrations in the air and childhood cancer rates, but failed to find a significant correlation (Raaschou-Nielsen, Hertel, Thomsen, & Olsen, 2001, p. 433). A related study focusing on childhood cancer rates among patients living near oil refineries emitting volatile organic compounds also failed to demonstrate a significant relationship (Wilkinson et al., 1999).

It is important to ascertain the implications of these conflicting studies. If childhood cancer rates are significantly associated with environmental pollutants, there is obvious potential for changes in national pollution standards not to mention the opportunity for early intervention to stop future cases. The purpose of this review is to synthesize the relevant epidemiological literature related to childhood cancer rates and hazardous air pollutants.

REVIEW OF STUDIES

To date, 18 peer-reviewed studies have been published addressing the link between air quality and childhood cancer. The first studies were conducted in Denver, Colorado in the late 70s and through the 80s (Savitz & Feingold, 1989; Wertheimer & Leeper, 1979). Most of the studies focused on the risks associated with residential traffic exposure, and just over half of the studies were conducted in the United States (Reynolds, Von Behren, Gunier, Raaschou-Nielsen, 2004, p. 1). Eleven of the studies found positive associations between childhood cancer and poor air quality, while seven failed to find a causal linkage. Studies that found no association were usually much larger, more recent, and more comprehensive (accounting for foreseeable confounders) than the studies that found a statistically significant association. The most notable studies will be discussed below.

STUDIES FINDING A POSITIVE CORRELATION

A particularly startling case-control study published in 1989 found that childhood cancers were significantly linked to high traffic density near the subject's home (Savitz & Feingold, 1989). Cases were defined as children ages zero to 14 who were diagnosed with cancer between 1976 and 1983 (Savitz & Feingold, 1989). The study was very

small, with a total of 162 childhood cancer cases and 100 exposed to vehicle pollutants (Savitz and Feingold, 1989). Researchers calculated an odds ratio for children aged zero to 14 living on streets with traffic densities of higher than 10,000 vehicles per day of 3.1 (95 percent confidence interval [CI] 1.2-8.0) for total childhood cancers and 4.7 (95 percent CI 1.6-13.5) for leukemia (18 and 8 cases respectively for the high risk categories) (Pearson, 2000, p. 176; Reynolds, Von Behren, Gunier, Raaschou-Nielsen, 2004, p. 1).

The authors noted limitations to the study, including the crude measure of exposure based solely on the subject's residence at time of diagnosis, exposure misclassification, small sample size and unmeasured confounders. Selection bias was a major issue in this study because a prerequisite for controls was that they be living in the same home they had lived in the year the case was diagnosed, whereas cases had no such requirement (Wartenberg, 1998). Thus, 57 percent of the cases had moved since the year the case was diagnosed, while none of the controls relocated (Wartenberg). Regardless of the design flaws, a study suggesting that children living in highly trafficked areas are three to five times more likely to get cancer than children who live on less dense streets would obviously cause quite a stir in the academic community. Other researchers were quick to conduct their own studies to investigate whether these results were legitimate.

A few years later, a follow-up study was conducted in Denver, Colorado, and it also found an association between proximal high traffic streets and childhood cancer. Researchers in this study employed the case-control childhood cancer epidemiologic data from the previous Savitz and Feingold study (Pearson, Wachtel, & Ebi, 2000, p. 176). Cases were defined as children aged zero to 14 who were diagnosed with cancer from 1976 to 1983 and who lived in the greater Denver metropolitan area (Pearson et al., pg 176). Cases were primarily found through the Colorado Central Cancer Registry of the Colorado Department of Health (Pearson et al., p. 176). Approximately 95 percent of the cases were confirmed by direct visualization or radiography (Pearson et al., p. 176). Of the 320 identified cases, 97 were leukemias, 59 brain cancers, 30 lymphomas, 32 soft tissue cancers and 102 other forms of cancer (Pearson et al., p. 176). The controls were selected by random digit dialing with individual matching of gender, age \pm three years and telephone exchange area (Pearson et al., p. 176).

Traffic density data was obtained from the Colorado Department of Transportation for the years corresponding to the collection of cancer data (1979 and 1990) (Pearson et al., p. 177). The distance was measured from the center line of the street to the center of the home where the subject resided (Pearson et al., p. 177). This measure was used to calculate airborne exhaust dispersion from motor vehicles traveling on the street. Possible confounders for this part of the experiment included prevailing winds, turbulence generated by passing traffic and the dispersion blocking effect of buildings and hillsides (Pearson et al., p. 177).

The study found a statistically significant correlation between highly trafficked streets (20,000 vehicles per day) and childhood cancer, but only for children living within 750 feet of the street (Pearson et al., p. 179). An odds ratio of 5.90 (95 percent CI 1.69-20.56) for all cancers and 8.28 (95 percent CI 2.09-32.80) for leukemia was calculated, significantly higher values than the previous study (Pearson et al., p. 175). Confounders for homes close to the street could include noise, increased light exposure or socioeco-

The study was good because it improved upon the design flaws of the Savitz and Feingold study (1989), such as taking into account the presence of other busy streets near the home. However, the study still suffered from several limitations. First, the number of cases and controls were very small, leading to a wide confidence interval, and second, data was not collected on the residential history during the relevant aetiological period of the child. The researchers conceded to flaws in the study and would not draw conclusions based on their evidential association between living near a high-traffic street and increased incidence of childhood cancer.

Knox and Gilman conducted a third case-control study in England, Scotland and Wales in which spatial clustering patterns were examined among 22,458 children aged zero to 15 from 1953 to 1980 (Knox & Gilman, 1997, p. 151-9). Abnormally high occurrences of childhood leukemia were found in children living near motorways and industrial plants (Knox & Gilman, 1997, p. 151-9). The highest percentages of cases were found within one km or less from motorways (Knox & Gilman, p. 151-9). Knox and Gilman concluded that a geographical association existed between childhood cancers and petroleum-derived volatiles, effluent from internal combustion engines and kiln and furnace smoke and gases.

This study, too, suffered from limitations and confounders. Matched controls were only available for two out of three cases (Knox & Gilman, 1997, p. 151-9). In addition, no data was available regarding subject's residence at conception and microenvironmental exposures. Also, only crude exposure measurements were used.

A 1998 small-scale Swedish study conducted by Feychting, Svensson and Ahlbohm, also found an association between motor vehicle exhaust and childhood cancer. Exposure data was obtained by estimating nitrogen dioxide levels from traffic data and road type (Feychting et al., p. 8-11). Cases were defined as children aged 15 or younger who had lived within 300 meters of high voltage power lines (for at least one year) during the time period of 1960-1985 (Feychting et al., p. 8-11). Only 142 cases and 550 controls were included in the study. Results in the highest exposure cases, with levels greater than or equal to 80 microg/m³ indicated a relative risk of 3.8 [1.2-12.1] (Feychting et al., p. 8-11). The results are limited because of the extremely small number of cases and the possibility of confounders such as exposure misclassification.

A recent population based case-control study in Italy (120 cases) found a nearly four-fold increase in leukemia diagnoses in high-exposure areas (Crosignani, Tittarelli, Borgini et al., 2004, p. 596). Concentrations of benzene were estimated by Gaussian diffusion model which considered vehicle emissions, distances from highly trafficked roads, traffic density in surrounding areas and weather conditions (Crosignani et al., p. 596-9). Researchers improved upon their initial study design by later obtaining information regarding the subject's benzene exposure history, especially focusing on the natal and perinatal time period (Crosignani et al., p. 596-9). Parents were interviewed by telephone about their lifestyle, residential history and means of transportation (Crosignani et al., p. 596-9). However, despite the improvements, the small sample size limited the application of results.

A new U.S. population based case-control study published in 2003 expanded the

scope of the previous studies to examine the effects of census tract hazardous air pollutant (HAP) levels, which were modeled by the United States EPA (Reynolds et al., 2003, p. 663-8). Cases were defined as children under the age of 15 living within a specified area in California (Reynolds et al., 2003). Researchers identified 7,143 cases, of which 2,443 were diagnosed with leukemia. No association of childhood cancer in general was found in the highest exposure levels of HAPs, however, the risks for leukemia were elevated by 21 percent (RR=1.21 95 percent CI 1.03-1.42). In areas where high HAP levels were caused mainly by industrial facilities, the association was even higher (RR=1.32 95 percent CI 1.11-1.57) (Reynolds et al., p. 663). In these areas, the HAPs were mainly benzene and perchloroethylene. Researchers found a higher association in children aged zero to four years and suspect that this is due to the fact that this age group may spend more time at home than older subjects. Confounders that were not considered included other sources of HAPs, especially those commonly found within the home.

STUDIES FINDING NO CORRELATION

A case-control study conducted in Denmark is arguably the most comprehensive and largest scale study thus performed on the association of vehicle pollution exposure and childhood cancer. Researchers measured the level and extent of past exposure to exhaust fumes at the residences of children with the most common types of cancer compared to a population-based, random sample of children without cancer (Raaschou, Hertel, Thomsen, Olsen, 2001, p. 433). Cases were defined as those children born after December 31, 1959, who were diagnosed with leukemia, tumor of the central nervous system or malignant lymphoma when they were under the age of 15, and between the period of January 1, 1968, and December 31, 1991 (Raaschou et al., p. 433). Cases were identified through the Danish Cancer Registry and totaled 1,989 children (Raaschou et al., p. 433). Controls were identified through the Danish Central Population Registry and were matched by sex and age (exact), totaling to 5,506 children (Raaschou et al., p. 433).

The average concentrations of benzene and nitrogen dioxide were measured at the front door of each subject's dwelling through the use of an Operational Street Pollution Model (Raaschou et al., 2001, p. 434). The model took into account such factors as street width, height of buildings and their distance from the street, traffic density (vehicles per day), the proportion of vehicles weighing more than 3,500 kg, average traffic speed, neighboring high density streets, meteorological variables such as wind speed, temperature and solar radiation and background air pollution (Raaschou et al., p. 434). The data was collected by municipal highway departments who were blinded as to whether the residences were those of the case or control subjects (Raaschou et al., p. 434).

Results were contrary to the other studies conducted up to that point. They indicated that there was no correlation between traffic-related air pollution at the subject's residence and increased rates of leukemias or central nervous system tumors (Raaschou et al., p. 439). However, researchers found a correlation between ambient air pollution at the time the child was in utero and risk of lymphomas, although it was restricted to Hodgkin's disease diagnosed before the age of 12 (Raaschou et al., p. 439).

Potential confounders were properly accounted for in the study. Trained employees of electric companies investigated the presence of 50- to 400- kV power lines, underground cables, transformer stations and measured the average electromagnetic field strength at each address (Raaschou et al., 2001, p. 435). Other factors considered included the mother's age and the birth order of the child, as well as the degree of urbanization and the type of residence (Raaschou et al., p. 435). There were slight differences between the numbers of cases and controls that were born in Copenhagen and that lived in single-family homes (Raaschou et al., p. 435).

Another study specific to traffic pollutants and childhood cancer was conducted by the nearly the same team of researchers that later found an association between leukemia and HAPs in California in 2003 (Reynolds et al., 2002; Reynolds et al., 2003). In the 2002 study, researchers identified nearly 6,000 cases, as children under the age of 15 who were diagnosed with cancer between 1988 and 1994 and living in neighborhoods with high density traffic (Reynolds et al., 2002, p. 665-73). Exposure was defined as levels of traffic exhaust, as estimated from three different traffic models (Reynolds et al., 2002, p. 665-73). Researchers failed to find an association between high traffic volume and childhood cancer rates (Reynolds et al., 2002, p. 665-73). However, the study had a number of design flaws that make the results somewhat questionable. First, no information was obtained regarding residential history, which is a very important factor in the study (Reynolds et al., 2002, p. 665-73). Second, no information was gathered regarding other sources of pollution (indoor or outdoor) (Reynolds et al., 2002, p. 665-73). Since the age of subjects included school aged children, it is important to also consider levels of pollution in or near the schools.

The same team of researchers conducted a follow-up study to refine their methods in 2004 (Reynolds et al., 2004, p. 6-12). Cases (5177) were defined as children under the age of five who lived in California and were diagnosed with cancer between 1988 and 1997 (Reynolds et al., 2004, p. 6-12). Controls were matched according to sex and age (Reynolds et al., 2004, p. 6-12). This time, information about residential history was collected, including the mother's residence at the time of birth (Reynolds et al., 2004). Exposure was defined as traffic emissions, estimated by traffic density on streets near subjects' homes (Reynolds et al., 2004). Improvements in this study included a larger sample size and acquisition of information regarding residential history (Reynolds et al., 2004). As in their previous study, researchers failed to find an association between traffic density and childhood cancer rates (OR=.92, 95 percent CI .73-1.15).

SUMMARY AND CONCLUSIONS

Based on the studies conducted thus far, evidence seems to suggest that the correlation between vehicle admissions and childhood cancer is unknown. While a majority of the studies conducted thus far have found a correlation, even a significantly high correlation in some studies, the study designs had flaws which cannot be ignored; whereas, the studies that found no correlation were much more comprehensive and of larger scale. The most notable of these is the Denmark study conducted by Raaschou-Nielsen et al. (2001), which was the only study to take into account both nitrogen dioxide concentrations and traffic density for the entire lifetime of the subject.

All studies are limited by the difficulty in measuring concentrations of potentially carcinogenic pollutants at the neighborhood level (Reynolds et al., 2003, p. 663-8). Other limitations that are most often found in studies on this subject include limited sample size, disease heterogeneity, exposure assessment, and analytic strategies.

Although the strongest evidence suggests a null association between vehicle emissions and childhood cancer rates, researchers should not retire future efforts to solve this puzzle. It is conceivable that future study designs will more precisely assess exposure, as well as accounting for host vulnerability in addition to other confounders discussed above. Childhood cancer is such an important problem that future studies on its relation to pollution are imperative, especially considering the mixed nature of results thus far.

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