# Prenatal exposure to air pollution boosts childhood cancer risk

By: MICHELE G. SULLIVAN, Ob.Gyn. News Digital Network

12-4-2013

WASHINGTON – Children whose mothers were exposed to air pollution during pregnancy may have a small, but significant, increase in pediatric cancer risk.

A 9-year case-control study found that children who were exposed to the highest levels of traffic-related pollution prenatally and during the first year of life were 19% more likely to develop bilateral retinoblastoma and 17% more likely to develop germ cell tumors than were children who were exposed to the lowest level.

"This is the first study to report upon traffic pollution in relation to retinoblastoma or germ cell tumors, and since both of these are rare, the findings need to be replicated in other studies," Julia E. Heck, Ph.D., said in an interview at the annual meeting of the American Association for Cancer Research.

Dr. Heck, an epidemiologist at the University of California's Fielding School of Public Health, Los Angeles, used a state air pollution modeling system to detect the patterns in her study. The California Line Source Dispersion Modeling system, version 4 (CALINE4) uses a test fleet of motor vehicles to predict traffic-related carbon monoxide emissions from vehicles in idle, cruise, acceleration, and deceleration.

Her study cohort comprised 3,590 children with cancer who were included in a California cancer registry. All were born between 1998 and 2007 and were aged younger than 6 years. She compared these children to a random selection of 80,224 children drawn form California birth records. The CALINE4 system generated estimates of local traffic exposure based on where the mothers were living during each trimester of pregnancy.

The model imputed emissions from gasoline and diesel vehicles within a 1,500-meter radius of the address. It included data on traffic volume, roadway geometry, vehicle emission rates, and weather patterns, and divided the cohort into quartiles according to exposure. Dr. Heck looked specifically at carbon monoxide measurements in increments of 53 parts per billion.

Each increase in CO emission raised the risk of acute lymphoblastic leukemia by 4%. Each quartile was associated with an overall 14% increased risk of a retinoblastoma, with the risk significantly elevated for bilateral tumors rather than unilateral tumors (a 19% increase). Dr. Heck also saw a 17% increased risk for germ cell tumors for every quartile increase in CO.

Associations with other cancers, including acute myeloid leukemia, non-Hodgkin's lymphoma, ependymoma, astrocytoma, neuroblastoma, Wilms tumor, hepatoblastoma, and rhabdomyosarcoma, were nonsignificant.

Because Dr. Heck's study was a retrospective analysis, it cannot prove causation. "The results need to be confirmed in other studies," she said in an interview. "But our findings do support a link between traffic pollution and some childhood cancers."

The field of childhood cancers and air pollution has not been well studied in recent years, Dr. Heck said, although there were a number of such studies in the late 1990s and early 2000s.

One was a British case-control analysis of birth and death records of 12,018 children in the United Kingdom who had been born and died from leukemia and other cancers from 1955 to 1980. The cancer-related deaths were linked to locations of rail and bus stations, ferry terminals, railways, roads, canals, and rivers (J. Epidemiol. Community Health 2005;59:755-60).

"The most striking result is the extraordinary concentration of cancer births within 0.3 km of bus/coach stations (OR = 12.5)," wrote Dr. E.G. Knox, professor emeritus at the University of Birmingham, England. "This was followed by hospitals (OR = 2.6) and heavy transport centers (OR = 1.6)."

Of the individual pollutants examined, both carbon monoxide and 1,3-butadiene more than doubled the risk of cancer.

Because the study encompassed the child's location at birth, it demonstrated a link to prenatal exposure. "Childhood cancers are strongly determined by prenatal or early postnatal exposures to oil-based combustion gases, especially from engine exhausts," he noted. "The chief carcinogenic agent is probably 1,3-butadiene."

The National Institute for Occupational Safety and Health has recommended that 1,3-butadiene be treated as a potential occupational carcinogen, teratogen, and as a reproduction hazard.

In 2006, Dr. Knox published another study on the same group, in which he attributed about 24% of the cancers to prenatal or early life proximity to areas of high airborne pollutants. "Child cancer initiations are strongly determined by prenatal or early postnatal exposures to engine exhaust gases, probably through maternal inhalation and accumulation of carcinogens over many months," he noted. (J. Epidemiol. Community Health 2006;60:136-41).

Dr. Heck's study was funded by the National Institutes of Health. She had no financial disclosures

**LSOG 2013**