Risks from Lawn-Care Pesticides

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…Five of the most popular pesticides in the U.S. home and garden sales market (2,4-D, glyphosate, MCPP, dicamba, and diazion) have been associated with non-Hodgkin's lymphoma (NHL) in epidemiological studies…Non-Hodgkin's lymphoma is the sixth most common malignancy in America, with nearly 54,000 cases estimated to have occurred in 2002. Between 1973 and 1997, the incidence increased by 80 percent. According to scientists at the National Cancer Institute, "Since the use of pesticides, particularly phenoxy herbicides, has increased dramatically preceding and during the time period in which the incidence of HNL has increased, they could have contributed to the rising incidence of NHL."

Children are often more susceptible to the toxic effects of pesticides than adults; they take in more pesticides relative to body weight than adults, and have developing organ systems that are more vulnerable and less able to detoxify toxic chemicals. In addition, the likelihood of developing cancer is greater if exposure occurs early in life, since cancer develops over time.

Children are especially vulnerable to carcinogens before the age of five, when their cells are normally reproducing most rapidly, may be more susceptible to loss of brain function if exposed to neurotoxins, and may be more susceptible to damage to their reproductive systems.

Children can be exposed to lawn-care pesticides by playing near an area where pesticides are being applied or by playing outside following a pesticide application, drinking or bathing in water contaminated with lawn-care pesticides, or from parental exposre to lawn-care chemicals during the child's gestation or prior to conception. Exposure to lawn-care pesticides can even occur inside a child's home. Studies have found that 2,4-D can be tracked from lawns into homes, leaving residues of the herbicide in carpets, on surfaces, and in indoor air. Estimated post-application indoor exposure levels for young children from non-dietary ingestion may be as high as 30 micrograms/day from contact with tabletops. By comparison, dietary ingestion of 2,4-D is approximately 1.3 micrograms/day.

Childhood malignancies linked to pesticides in studies include leukemia, neuroblastoma, Wilm's tumor, soft-tissue sarcoma, Ewing's sarcoma, NHL, and cancers of the brain, colorectum, and testes. Many of the reported increased risks are greater than those noted in studies of pesticide-exposed adults, indicating that children may be particularly sensitive to the carcinogenic effects of pesticides.

Pesticides and Childhood Cancer: An Update of Zahm and Ward's 1998 Review

Journal of Toxicology and Environmental Health, Volume 10, Issue 1 & 2 December 2006, pages 81 – 99

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Abstract:
Children are exposed to pesticides through a number of sources, including residential and agricultural applications. Parental occupational exposure to pesticides is also a concern because exposures occurring during pregnancy and carry-home residues also contribute to children's cumulative burden. A number of epidemiological studies consistently reported increased risks between pesticide exposures and childhood leukemia, brain cancer, neuroblastoma, non-Hodgkin's lymphoma, Wilms' tumor, and Ewing's sarcoma. An extensive review of these studies was published in 1998 (Zahm & Ward, 1998). Fifteen case-control studies, 4 cohort studies, and 2 ecological studies have been published since this review, and 15 of these 21 studies reported statistically significant increased risks between either childhood pesticide exposure or parental occupational exposure and childhood cancer. Therefore, one can confidently state that there is at least

some association between pesticide exposure and childhood cancer. However, an unambiguous mechanistic cause-and-effect relationship between pesticide exposure and childhood cancer was not demonstrated in these studies, and modifying factors such as genetic predisposition, rarely considered in the reviewed studies, likely play an important role. While the time window of exposure may be a crucial determinant for biological effects associated with pesticide exposure on children, studies have not contributed definitive information on the most vulnerable period. Accurate exposure assessment remains a challenge; future epidemiological studies need to assess gene-environment interactions and use improved exposure measures, including separate parental interviews, specific pesticide exposure questions, and semiquantitative exposure measures that can be used to confirm information obtained through questionnaires.

Residential Exposure to Polychlorinated Biphenyls and Organochlorine Pesticides and Risk of Childhood Leukemia

Environmental Health Perspectives January 2009

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Abstract:

BACKGROUND: Incidence of childhood leukemia in industrialized countries rose significantly during 1975-2004, and the reasons for the increase are not understood.

OBJECTIVES: We used carpet dust as an exposure indicator to examine the risk of childhood leukemia in relation to residential exposure to persistent organochlorine chemicals: six polychlorinated biphenyl (PCB) congeners and the pesticides alpha- and gamma-chlordane, p,p'-DDT (dichlorodiphenyltrichloroethane), p,p'-DDE (dichlorodiphenyldichloroethylene), methoxychlor, and pentachlorophenol.

METHODS: We conducted a population-based case-control study in 35 counties in northern and central California in 2001-2006. The study included 184 acute lymphocytic leukemia (ALL) cases 0-7 years of age and 212 birth certificate controls matched to cases by birth date, sex, race, and Hispanic ethnicity. We collected carpet dust samples from the room where the child spent the most time before diagnosis (similar date for controls) using a specialized vacuum. RESULTS: Detection of any PCB congener in the dust conferred a 2-fold increased risk of ALL [odds ratio (OR) = 1.97; 95% confidence interval (CI), 1.22-3.17]. Compared with those in the lowest quartile of total PCBs, the highest quartile was associated with about a 3-fold risk (OR = 2.78; 95% CI, 1.41-5.48), and the positive trend was significant (p = 0.017). Significant positive trends in ALL risk were apparent with increasing concentrations of PCB congeners 118, 138, and 153. We observed no significant positive associations for chlordane, DDT, DDE, methoxychlor, or pentachlorophenol. The associations with PCBs were stronger among non-Hispanic whites than among Hispanics despite similar distributions of PCB levels among controls in each racial/ethnic group.

CONCLUSIONS: Our findings suggest that PCBs, which are considered probable human carcinogens and cause perturbations of the immune system, may represent a previously unrecognized risk factor for childhood leukemia.

Childhood Brain Tumors, Residential Insecticide Exposure, and Pesticide Metabolism Genes

Environmental Health Perspectives January 2010

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Abstract:

BACKGROUND: Insecticides that target the nervous system may play a role in the development of childhood brain tumors (CBTs). Constitutive genetic variation affects metabolism of these chemicals.

METHODS: We analyzed population-based case-control data to examine whether CBT is associated with the functional genetic polymorphisms PON1C-108T, PON1Q192R, PON1L55M, BCHEA539T, FMO1C-9536A, FMO3E158K, ALDH3A1S134A, and GSTT1 (null). DNA was obtained from newborn screening archives for 201 cases and 285 controls, <or= 10 years of age, and born in California or Washington State between 1978 and 1990. Conception-to-diagnosis home insecticide treatment history was ascertained by interview.

RESULTS: We observed no biologically plausible main effects for any of the metabolic polymorphisms with CBT risk. However, we observed strong interactions between genotype and insecticide exposure during childhood. Among exposed children, CBT risk increased per PON1-108T allele [odds ratio (OR) = 1.8; 95% confidence interval (CI), 1.1-3.0] and FMO1-9536A (*6) allele (OR = 2.7; 95% CI, 1.2-5.9), whereas among children never exposed, CBT risk was not increased (PON1: OR = 0.7; 95% CI, 0.5-1.0, interaction p = 0.005; FMO1: OR = 1.0; 95% CI, 0.6-1.6, interaction p = 0.009). We observed a similar but statistically nonsignificant interaction between childhood exposure and BCHEA539T (interaction p = 0.08). These interactions were present among both Hispanic and non-Hispanic white children.

CONCLUSION: Based on known effects of these variants, these results suggest that exposure in childhood to organophosphorus and perhaps to carbamate insecticides in combination with a reduced ability to detoxify them may be associated with CBT. Confirmation in other studies is required.

Agricultural Exposures and Childhood Cancers

Rev Environ Health. Volume 24 Issue 4 Page 339-44 October 2009

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Abstract:
Cancers occurring in children are rare but tragic occurrences. Sadly, the causes of these events are poorly understood, and because of their rarity, also difficult to study. Because, with a few exceptions, there are not well established causal genetic or behavioral factors for these diseases it is not surprising that impacted families and communities have concerns about the potential role of ambient exposures in the physical environment. Although a number of fledgling studies have begun to systematically evaluate potential risks proximity to agricultural pesticide use, challenges posed by balancing the interplay of exposure mechanisms, host susceptibility, scale, and timing make this a daunting task. Future progress will require support for leveraging large and heterogeneous population studies, assembling transdisciplinary investigative teams, and for promoting creative research strategies. Such efforts will be informative for studying a host of health risks for both adults and children. In the meantime, regulatory actions such as requiring pre-market testing of new chemical compounds used in agriculture for potential adverse health effects and minimizing exposure opportunities for children and women of childbearing age to agents with known toxic properties would be a prudent course of action. We owe it to our children and our future to seriously address these issues.

Prenatal and Childhood Exposure to Pesticides and Neurobehavioral Development: Review of Epidemiological Studies

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Abstract:

Objectives: Conventional pesticides comprise a diverse group of substances intended to destroy, repel or control organisms identified as pests. Compared to the studies on lead, mercury, and PCBs, few epidemiological studies have assessed the developmental neurotoxicity of pesticides. Materials and

Methods: Epidemiological studies focused on the neurobehavioural development of children exposed to pesticides were identified by searching the PubMed, Medline, EBSCO, Agricola and TOXNET databases.

Results: The findings of the studies reviewed imply that children's exposure to pesticides may bring about impairments in their neurobehavioral development. Children exposed to organophosphate pesticides (OP), both prenatally and during childhood, may have difficulties performing tasks that involve short-term memory, and may show increased reaction time, impaired mental development or pervasive developmental problems. In newborns, the effects of OP exposure are manifested mainly by an increased number of abnormal reflexes, while in adolescents, by mental and emotional problems. The studies investigating association between exposure to organochlorine pesticides and neurodevelopmental effects show inconsistent results. While some studies report impairments in mental and psychomotor functions, other studies do not confirm the above.

Conclusion: The information deriving from epidemiological studies indicate a need to increase awareness among people and children exposed to pesticides about the association between the use of pesticides and neurodevelopmental impairments. Therefore, the principle of prudence should become a rule.

Pesticides Literature Review

The Ontario College of Family Physicians, April 23, 2004

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Abstract:
In recent years, few environmental issues have aroused the concern of the public as much as pesticides, especially in relation to the health of children. In spite of the many published studies on the subject of pesticides and human health, there remains deep controversy surrounding this issue.

During the 1960s and 1970s, epidemiologists in the USA noted a rise in the incidence of non-Hodgkin's lymphoma (NHL). When plotted on a map of the USA these cases were clearly clustered in agricultural areas. This increase in NHL incidence paralleled the rise in pesticide use, prompting some epidemiologists to theorize that there was a causal link.

Several studies found associations between pesticide exposures and solid tumours in children. An elevated rate of kidney cancer was associated with paternal pesticide exposure through agriculture. Four studies found associations with brain cancer: two found associations with indoor household use of pesticides one with parental farming occupation, and one with parental occupational exposure to pesticides.

Conclusions:

Several studies in this review implicate pesticides as a cause of hematologic tumours in children. One study found an association with childhood non-Hodgkin's lymphoma, and several studies found elevated childhood leukemia rates with pesticide exposure. An excellent study by [Xiaomei Ma (University of California,Berkeley)] showed an association between maternal pesticide exposure and childhood leukemia.

In the genotoxicity or immunotoxicity area there were two studies relevant to children. In the first, children with poor metabolizer polypmorphisms, genotyped at birth and representing just over 40% of the Montreal study group, had overall increased risk of acute lymphocytic leukemia if exposed to pesticides in utero or during childhood, especially for exposure to repellents and sprays for outdoor insects during pregnancy, and exposure to mite and spider killers during pregnancy or between birth and leukemia diagnosis. Herbicide use (mainly 2,4-D) both during pregnancy and in childhood, showed a consistent interaction with poor metabolizer genes and was associated with a 2-fold increase in leukemia incidence. [Terry M.] Phillips found that children exposed to chlordane and/or heptachlor had more cytokine panel abnormalities than matched controls.

Neurodevelopmental effects were found in pre-school children in pervasive pesticide exposure situations in Mexican valley agriculture, and likely resulted from maternal, in-utero, and early childhood exposures. The only other study of effects on children found substantially higher proportions of residents — including adolescents — exposed to pesticides from aerial spraying drift to have mental and emotional symptoms compared to those not exposed by aerial spraying, consistent with other studies of broader nervous system function.

In the reproductive review, finding suggested that occupational exposure to agricultural chemicals including pesticides may cause intrauterine growth retardation, and may increase a woman's risk of giving birth to children with congenital anomalies, such as limb defects, nervous system and musculoskeletal defects, cryptorchidism and hyposadias, cardiovascular defects, oral clefts, and other multiple and specific defects. The adverse reproductive effects that are non-fatal produce future risks for the individual and for the next generation. Intrauterine growth retardation has been shown to increase susceptibility in later life to hypertension, type 2 diabetes, heart disease, and breast and prostate cancer. Men with birth defects are twice as likely to produce children with birth defects.

Adverse Health Effects of Children's Exposure to Pesticides: What Do We Really Know and What Can Be Done About It?

Acta Paediatrica, Volume 95, Issue S453 October 2006, pages 71 - 80

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Abstract:
Children may be exposed to pesticides in several ways, such as by transplacental transfer during fetal life, by intake of contaminated breast milk and other nutrients, or by contact with contaminated subjects and areas in the environment such as pets treated with insecticides, house dust, carpets and chemically treated lawns and gardens. Exposure early in life, and particularly during periods of rapid development, such as during foetal life and infancy, may have severe effects on child health and development by elevating the risk of congenital malformations, cancer, malabsorption, immunological dysfunction, endocrine disease, and neurobehavioural deficiencies. As pesticides can also interfere with parental reproductive health, exposure of parents may have consequences for the offspring leading to reduced chance of male birth and increased risk of childhood cancer.

Conclusions: Current knowledge about tolerable levels and consequences of toxic exposure to pesticides during human development is rather scarce. Owing to the high risk of exposure to pesticides, particularly in less developed countries, further elucidation by well-controlled epidemiological studies in this field it is urgently needed. The Policy Interpretation Network on Children's Health and Environment (PINCHE), which is financed by the EU DG research has suggested actions against pesticide exposure.