

Chapter 10 — Pesticide Health Effects and Children

Children are ubiquitously exposed to low levels of pesticides in their food and environment, yet there has been a paucity of studies on the long-term health effects of these exposures (10, 26, 32). Many pesticides persist in the environment, are often transferred long distances from their original area of application, are routinely detected in human tissue, and are transferred across the placenta and via breast milk (1, 19).

Relative to adults, children eat more in proportion to their body weight, resulting in more concentrated exposures. Intakes by children of the four primary pesticides (chlorpyrifos, malathion, diazinon, and atrazine) appear to come primarily from the ingestion of solid food (2). Another common exposure source is indoor and outdoor home pesticide applications, where children may be exposed by playing on floors, treated lawns and play areas, or by handling treated pets (8). Agricultural uses of pesticides may expose children inadvertently from spray drift or farm work (31).

Children present a number of unique characteristics with regard to risks from exposure to pesticides and other environmental pollutants. The most vulnerable time is during fetal development when the brain is known to be subject to environmental influences at all phases of development, with critical windows at different points (6). Since in the female, ova are formed in the fetal stage, and environmental contaminants have been found in follicular fluid, the next generation of children born may be affected by their grandmother's exposures (6). The newborn child has low levels of the enzyme paraoxanase-1, which detoxifies organophosphate pesticides (7).

Environmental contaminants may pose a greater risk to children than adults for another reason: children have a longer life expectancy in which to develop diseases with long latency periods. For example, if a 70-year-old adult and a 5-year-old child are exposed to a carcinogen with a 40-year latency period, the child has a much higher lifetime risk of developing adverse health consequences (20).

Studies in children have so far demonstrated subtle neurotoxic effects of low level, intrauterine, or early childhood exposures to a variety of environmental agents including lead, methyl mercury, and PCBs. While studies of pesticide health effects in children are still lacking, it is possible that a parallel model may emerge for low-level exposures to pesticides, some of which are by design neurotoxic (28, 32, 33). A range of developmental disabilities including learning disabilities, attention deficit hyperactivity disorder, developmental delays, autism, and behavioural disorders are of great importance due to possibly increasing incidence, and personal and public health costs. (12, 17, 18, 33). These are disorders of unknown etiology with a link between genetic susceptibility and environmental factors, perhaps including pesticides in some small proportion of cases (18, 27). Research is urgently needed to fill in the many gaps in this area.

Summary of Findings Concerning Children

The few studies we found which addressed children's health effects from exposures to pesticides have been discussed in detail in each relevant chapter and will be summarized here.

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 (11). Four studies found associations with brain cancer: two found associations with indoor household use of pesticides (9, 30), one with parental farming occupation (16), and one with parental occupational exposure to pesticides (34).

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 (5), and several studies found elevated childhood leukemia rates with pesticide exposure (16, 21, 23, 24). An excellent study by Ma (23) showed an association between maternal pesticide exposure and childhood leukemia. More detailed information on these studies is in Chapters 3–5.

In the genotoxicity or immunotoxicity area (Chapter 6) there were two studies relevant to children. In the first, children with poor metabolizer polymorphisms, 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 (14). Phillips (29) found that children exposed to chlordane and/or heptachlor had more cytokine panel abnormalities than matched controls.

Neurodevelopmental effects (Chapter 8) 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 (13). The only other study of effects on children (15) 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 (Chapter 9), findings 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 hypospadias, 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 (3, 4). Men with birth defects are twice as likely to produce children with birth defects (22).

Future Studies

There have been some plans to develop a parallel Canadian cohort study that would be complementary to the US National Children's Study, a study that will follow a cohort of 100,000 children from the prenatal period to adulthood to study environmental influences on health and development. Scientists from Health Canada were involved in the planning along with the US National Institute of Child Health and Human Development, the US Environmental Protection Agency, the Centers for Disease Control and Prevention, and the National Institute of Environmental Health Sciences. The Canadian involvement is dependent on federal funding which as of February 2004 is still uncommitted. Such a large and comprehensive prospective study is vital and would finally provide sufficient data to inform whatever policy decisions are necessary to protect our children and their futures.

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References

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