

REPRODUCTIVE AND DEVELOPMENTAL EFFECTS OF PESTICIDES



While further research is necessary, existing epidemiological studies link pesticides to adverse reproductive and developmental effects. Scientific and medical community's available data demonstrate a clear need to follow the Precautionary Principle, that is, to avoid use and exposure where possible. Unfortunately, the state of knowledge is less than optimal because of the difficulty in measuring pesticide exposures years after the damage may have been done. Until pesticides have been proven safe for children and adults, it only makes sense to minimize exposure to the greatest extent possible.

A recent study finds that pesticides are used in three-fourths of all households, most of which are not aware of the potential health risks.¹

INFANTS AND CHILDREN AT RISK

Recent research indicates that the developing fetus, infant and child may suffer disproportionately from the health effects of pesticides due to:

- ❖ Heightened vulnerability during the growth and development processes.
- ❖ Greater exposure to toxics because children eat, drink and breathe more in proportion to body weight than do adults.²
- ❖ Playing in areas where pesticides are commonly applied, such as floors and lawns.
- ❖ Frequent hand-to-mouth contact.
- ❖ Dietary patterns that increase their exposure to pesticides.³ For example, they consume proportionally larger quantities of products containing pesticide residues, such as applesauce and fruit juice, than do adults.
- ❖ Accidental ingestion of pesticides due to careless storage or handling. EPA receives about 16,000 pesticide hotline calls each year from parents concerned about pesticide risks to their children, because children may have accidentally ingested pesticides that were carelessly stored or handled.⁴
- ❖ Greater susceptibility to airborne health hazards than adults in part because children have a greater average activity level with more rapid respiratory rates. Additionally, because terminal airways of the lung are not fully developed in infants or toddlers, they have a greater risk of respiratory hazards.⁵

HEALTH EFFECTS OF PESTICIDES ON FETUS AND BREASTFED BABIES

Pesticides can also accumulate in body tissues, and may be eliminated in breast milk or by crossing the placenta to a developing fetus. Studies indicate pesticides that cross the placenta can directly affect the developing child, even at low doses.

“[P]regnant women should take extra care to avoid exposure to pesticides.”

— *National Environmental Education and Training Foundation (2003).*

“National Pesticide Practice Skills for Medical and Nursing Practice.”

Pregnant women should be discouraged from using any pesticides, especially aerosol foggers (“bombs”) and other pesticide sprays.

A mother's exposure to pesticides may result in birth defects, low birth weight, or spontaneous abortion.^{6,7,8,9} Epidemiological studies suggest that children exposed to flea and tick products as fetuses may have a higher risk of developing brain tumors by age five.¹⁰

Numerous epidemiological studies and case reports demonstrate an increased risk of congenital malformations where there is pesticide exposure at work or home. A report entitled "*Pesticides and Human Health*" summarizes some of the birth defects that may be associated with exposure to pesticides:¹¹

- ❖ Cleft lip and palate—exposure during the first trimester may double the risk;^{12,13,14}
- ❖ Limb defects—garden or workplace exposures may cause a 3-4-fold increased risk;^{15,16,17,18}
- ❖ Cardiovascular malformations—the Baltimore/Washington Infant Study found a 2-3-fold greater risk;¹⁹
- ❖ Spina bifida and hydrocephaly—residence within a quarter-mile of an agricultural field may be associated with a 50 percent increased risk;^{20,21}
- ❖ Cryptorchidism and hypospadias—agricultural areas may produce 2-3-fold greater rates of orchidopexy and a 50 percent increase in hypospadias.^{22,23}

HEALTH EFFECTS OF PESTICIDES ON CHILDREN

Some pesticides on the market today are known to have significant toxicities for children, including cancer, acute and chronic injury to the nervous system, lung damage, reproductive dysfunction, and possibly dysfunction of the endocrine and immune systems.^{24,25} Along with the increased vulnerability due to developing organs, their enzymatic, metabolic, and immune systems are immature, providing them with less natural protection than adults.²⁶

The possible effects of pesticide exposure on fetal development and young children range from increased learning disabilities, attention deficit and other behavioral disorders, to cancers such as leukemia, soft-tissue sarcomas, and pediatric brain tumors. According to one study, children living in homes where yards are treated with pesticides may have up to four times greater risk of developing soft-tissue sarcoma and are three times more likely to develop leukemia.²⁷



MORE TESTING IS NEEDED

Less than one percent of pesticides used on crops, fabrics, lawns, in schools, hospitals, and homes have been tested to determine their effects on children's developing nervous systems.²⁸ It may take up to several decades to identify these hazards, as EPA only recently began requiring developmental neurotoxicity testing of pesticides. Additional tests to understand the health effects of exposure to mixtures of chemicals are also needed.

Government policy allowing low levels of pesticide contamination in food, water, and air rests on the assumption that such exposures are harmless. Yet, recent studies suggest that in some cases low doses of chemicals once believed safe can affect the endocrine, immune and nervous systems.²⁹ A child with a compromised immune system or inadequate nutrition may be even more susceptible to pesticides.³⁰

REPRODUCTION AND DEVELOPMENT

The Effects of Pesticides on Fertility

Agricultural pesticide applicators exposed to the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) have been shown to have decreased sperm density and increased percentages of abnormal sperm. In a study of over 800 couples undergoing in-vitro fertilization, men who were moderately or highly exposed to pesticides at work had significantly decreased fertilization rates compared with unexposed males. These couples were also much less likely to have successful in-vitro fertilization. After adjusting for all other known exposure variables, such as smoking, alcohol, caffeine, and other chemical use, these effects persisted.³¹



Developmental Abnormalities

Studies of female agricultural workers and wives of agricultural workers have implicated pesticides in spontaneous abortions and stillbirths. One study demonstrates a 70 percent increased risk of stillbirth for home exposure to pesticides, and a 240 percent increased risk of stillbirth for occupational exposure.³² Another study shows that women living in communities where drinking water is contaminated by a variety of herbicides have an 80 percent increased risk of intra-uterine growth retardation when compared with similar communities with uncontaminated water.³³

Endocrine Disruption

Many currently used pesticides are now known, and others are suspected, of being endocrine disruptors. These chemicals can either emulate or interfere with hormones like estrogen, testosterone and thyroid hormones. Endocrine disruption may result in infertility, low sperm counts, testicular and breast cancer, endometriosis, prostate enlargement, altered fetal and child development as well as other disorders.³⁴

Wildlife studies show that exposure to certain pesticides may result in reproductive abnormalities,

animals unable to care for their young and alterations in behavior and growth. Male animals are found to have significantly depressed testosterone, and females have twice the normal levels of estrogen. These problems, resulting from low-level contaminants that persist from birth, may create organizational abnormalities that occur during fetal development and last a lifetime.³⁵

In regard to endocrine disrupting pesticides, it has been established that conventional dose response curves (the “dose makes the poison” assumption) may be wrong for some health effects. In its year 2000 review (at the invitation of EPA) of endocrine disruptors, the National Toxicology Program (NTP) confirmed low-dose effects as well as non-monotonic dose response curves. The NTP concluded that low-dose considerations must be integrated into regulatory science, yet, EPA has not done this in the case of pesticide registrations. Recent studies suggest that the usual rule of an increased effect with an increasing dose may not always be true. This means that low doses formally thought to be safe must be restudied and that may affect safety considerations of low dose exposures. This is yet another reason for precaution.

Note: For sources of this material see back of this page.



MARYLAND PESTICIDE NETWORK

www.mdpestnet.org • info@mdpestnet.org • (410) 849-3909

- 1 Aspelin, AL and AH Grube (1999). "Pesticide Industry Sales and Usage: 1996 and 1997 Market Estimates." Washington, DC. U.S. Environmental Protection Agency, Office of Pesticide Programs.
- 2 National Research Council (1993). "Pesticides in the Diets of Infants and Children." Washington, D.C., National Academy Press.
- 3 Wargo, John (1996). "Our Children's Toxic Legacy: How Science and Law Fail to Protect Us from Pesticides." Yale University Press, New Haven and London.
- 4 U.S. Environmental Protection Agency (1998). "The EPA Children's Environmental Health Yearbook." Available online at www.epa.gov/children/whatwe/ochpyearbook.pdf. Website accessed August 2002. Page 85.
- 5 American Academy of Pediatrics Committee on Environmental Health (1993). "Ambient air pollution: Respiratory hazards to children." *Pediatrics* 91: 6: 1210-13.
- 6 Baker, S.R. and C.F. Wilkinson, eds. (1990). "The effects of pesticides on human health: Advances in modern environmental toxicology XVIII." Princeton, NJ: Princeton Scientific Publishing.
- 7 Arbuckle, T.E. and L.E. Sever (1998). "Pesticide exposures and fetal death: A review of the epidemiologic literature." *Crit Rev Toxicol* 28: 229-70.
- 8 Moses, M. (1993). "Occupational and environmental reproductive hazards: A guide for physicians." *Baltimore: Williams and Wilkins*: 296-305.
- 9 Pastore, L.M., I. Hertz-Picciotto, and J.J. Beaumont (1997). "Risk of stillbirth from occupational and residential exposures." *Occup Env Med* 54 7: 511-18.
- 10 Pagoda, J.M. and S. Preston-Martin (1997). "Household Pesticides and Risk of Pediatric Brain Tumor." *Environmental Health Perspectives*. Vol.105,#11.
- 11 Solomon, Gina (2000). "Pesticides and Human Health: A Resource for Health Care Professionals." Published by Physicians for Social Responsibility and Californians for Pesticide Reform. Available online at <http://www.sfbaypsr.org/publications.html>.
- 12 Nurminen, T., K. Rantala, K. Kurppa, and P.C. Holmberg (1995). "Agricultural work during pregnancy and selected structural malformations in Finland." *Epidemiology* 6: 23-30.
- 13 Gordon, J.E. and C.M. Shy (1981). "Agricultural chemical use and congenital cleft lip and/or palate." *Arch Env Hlth*. 36: 213-20.
- 14 Shaw, G.M., C.R. Wasserman, C.D. O'Malley, et al. (1999). "Maternal pesticide exposure from multiple sources and selected congenital anomalies." *Epidemiology* 10: 60-66.
- 15 Kricker, A., McCredie, J. Elliot, and J. Forrest (1986). "Women and the environment: A study of congenital limb anomalies." *Comm Hlth Stud* 10: 1-11.
- 16 Lin, S., E.G. Marshall, and G.K. Davidson (1994). "Potential parental exposure to pesticides and limb reduction defects." *Scand J Work Env Hlth* 20: 166-79.
- 17 Kristensen, P., L.M. Irgens, A. Andersen, et al. (1997). "Birth defects among offspring of Norwegian farmers, 1967-1991." *Epidemiology* 8 5: 537-44.
- 18 See note 14 above.
- 19 Correa-Villasenor, A., C. Ferencz, J.A. Boughman, and C.A. Neill (1991). "Baltimore-Washington Infant Study Group-Total anomalous pulmonary venous return: Familial and environmental factors." *Teratology* 44: 415-28.
- 20 Kristensen, P., L.M. Irgens, A.Andersen, et al. (1997). "Birth defects among offspring of Norwegian farmers, 1967-1991." *Epidemiology* 8 5: 537-44.
- 21 See note 14 above.
- 22 Garcia-Rodriguez, J., M. Garcia-Martin, M. Nogueras-Ocana, et al. (1996). "Exposure to pesticides and cryptorchidism: Geographical evidence of a possible association." *Environmental Health Perspectives* 104: 1090-95.
- 23 See note 20 above.
- 24 Goldman, L.R. (1995). "Children Unique and Vulnerable: Environmental Risks Facing Children and Recommendations for Response." *Environmental Health Perspectives*. Volume 103 (Supplement 6), Pages 13-18.
- 25 Zahm, S.H. and S.S. Devesa (1995). "Childhood Cancer: Overview of Incidence Trends and Environmental Carcinogens." *Environmental Health Perspectives*. Volume 103 (Supplement 6), Pages 177-184.
- 26 See note 2 above.
- 27 Leiss, JK; DA Savitz (1995). "Home Pesticide Use and Childhood Cancer." *Amer. Journal of Public Health*. Vol. 85.
- 28 Wargo, John and Emily Eveston Wargo (2002). "The State of Children's Health and the Environment, 2002." Available online at www.chechnet.org.
- 29 *Ibid*.
- 30 *Ibid*.
- 31 Lerda, D. and R. Rizzi (1991). "Study of reproductive function in persons occupationally exposed to 2,4-dichlorophenoxyacetic acid (2,4-D)." *Mutat Res* 262 1:47-50. E. Telemans, R. Van Kooij, E.R. te Velde, and D. Heederik (1999). "Pesticide exposure and decreased fertilization rates in vitro." *Lancet* 354: 484-85.
- 32 Pastore, L.M., I. Hertz-Picciotto, and J.J. Beaumont (1997). "Risk of stillbirth from occupational and residential exposures." *Occupational Environmental Medicine*. 54.7:511-18.
- 33 Munger, R.G. , P. Isaacson, S. Hu, et al. (1997). "Intrauterine growth retardation in Iowa communities with herbicide-contaminated drinking water supplies." *Environmental Health Perspectives*. 105: 308-314.
- 34 Solomon, Gina (1998). "Trouble on the Farm: Growing Up with Pesticides in Agricultural Communities." NRDC Report.
- 35 Guillette, Louis (2000). "Impact of Endocrine Disruptors on Wildlife." *Center for Health Effects of Environmental Contaminants*, Chicago, Illinois.

MARYLAND PESTICIDE NETWORK

www.mdpestnet.org • info@mdpestnet.org • (410) 849-3909