Health Effects of Pesticides on Children

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UNIVERSITY HONORS PROGRAM
SENIOR PROJECT - PROSPECTUS

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Faculty Mentor: Mary Rogge, Ph.D.

PROJECT TITLE: Health Effects of Pesticides on Children

PROJECT DESCRIPTION (Attach not more than one additional page, if necessary):

This project will research three specific health effects of pesticides on children. These include childhood cancer, endocrine disruption, and developmental problems. The final product will consist of a manuscript jointly written by Dr. Rogge and me. It will be submitted to the Journal "Health and Social Work."

Projected completion date: April 2003

Signed: Sadaf Shaikh

I have discussed this research proposal with this student and agree to serve in an advisory role, as faculty mentor, and to certify the acceptability of the completed project.

Signed: Mary Rogge, Ph.D., Faculty Mentor

Date: 10-8-03

Return this completed form to The University Honors Program, F101 Melrose Hall, following your first presentation in the Senior Project Seminar.
Health Effects of Pesticides on Children

Our environment is becoming increasingly exposed to various types of chemicals. Pesticides are a group of compounds that have proven to contribute to health problems in adults. They are commonly used in homes, lawns, schools, and day-cares. Furthermore, acute and chronic exposure to these chemicals can lead to a variety of health problems. Often times, children are overlooked when it comes to forming legislation and public guidelines. This paper will emphasize, through established literature, the great risk posed to children by these chemicals. The purpose of this project is to prove that children are affected in a different manner, and usually more severely. The three health effects that will be explored in this paper include endocrine disruption, developmental abnormalities, and childhood cancer.
UNIVERSITY HONORS PROGRAM

SENIOR PROJECT - APPROVAL

Name: Sadaf Shaukat
College: Arts & Sciences  Department: Microbiology
Faculty Mentor: Mary E. Rogge, Ph.D.

PROJECT TITLE: Health Effects of Pesticides on Children

I have reviewed this completed senior honors thesis with this student and certify that it is a project commensurate with honors level undergraduate research in this field.

Signed: Mary E. Rogge, Faculty Mentor

Date: 5/8/03

Comments (Optional): I am confident that Sadaf’s thesis will be completed by June 15. She and I will continue to collaborate on a manuscript that incorporates her thesis work.

Mary E. Rogge 5/8/03
Endocrine Disruption

Peer-Reviewed Articles

Pesticides are designed to poison and kill. It is no surprise that scientists have been substantiating a link between numerous health problems and the use of pesticides. Children, more so than adults, are uniquely susceptible to the harmful effects of these chemicals. Common hand-to-mouth behavior and an increased tendency to play close to the ground are unique routes of exposure for children. Also, a child’s diet is a likely route of exposure (Landrigan, 2001). Childrens’ metabolic pathways are maturing and may not be able to detoxify chemicals as well as adults. In a recent study, Scheuplein, Charnley, and Dourson have found that the child’s immature metabolic system usually produces higher toxin blood levels for a longer period of time (2002).

Of the 20,000 emergency room visits from 1990 to 1992 due to exposure to organophosphates and other pesticides, nearly 61% were by children (Landrigan, 2001). The 2001 Annual Report published by the American Association of Poison Control Centers documented 46,929 cases of pesticide exposure of children under 6. In addition, 19,495 people were treated in a health care facility in 2001 due to pesticide exposure. Seventeen people died as a result of exposure to fungicides, metam sodium fumigants, paraquat herbicides, carbamate, chlorinated hydrocarbon, organophosphate, and pyrethrin insecticides, and strychnine and zinc phosphide rodenticides (Litovitz et al, 2002).

Routes of Exposure

Children encounter pesticides at schools and on public and private lawns and gardens. The United States General Accounting Office published a report in November
1999 documenting that from 1993-1996, 2,300 pesticide-related exposures at public schools were reported. Approximately 90% of homeowners apply pesticides inside the home or on lawns, making this the largest route of potential exposure to children (Landrigan, 2001). According to a 1997 EPA report, homeowners used 74 million pounds of pesticides in one year in their gardens and homes (EPA, 1997). Most pesticides contain chemicals that have a high persistence. Persistence is a measure of how long a chemical can survive in the environment before being naturally destroyed. Dioxin and dioxin-like compounds, for example, have persistence in soil ranging from 1.5 years to 20 years (EPA, 2000).

**Endocrine Disruption**

Research studies have shown that several persistent chemicals found in pesticides are linked to disruption of the endocrine system (Reiter, DeRosa, Kavlock, et al, 1998). The human endocrine system is of great importance, especially in regard to children because their bodies are continually developing and maturing. The endocrine system controls the body's hormonal processes. Hormones such as epinephrine, estrogen, and testosterone are essential for our well-being. Epinephrine, also known as adrenaline, stimulates the heart and increases blood pressure, metabolic rate, and blood glucose concentration (Campbell, 1993). Estrogen is a steroid hormone found in males, but in a higher concentration in females. Estrogen plays an important role in the development of female secondary sexual characteristics and influences growth, behavior, and female reproductive cycles (Campbell, 1993). Testosterone is a central hormone secreted in the male testis and is responsible for sperm production and the development of male secondary sexual characteristics (Campbell, 1993).
Research studies indicate that many chemicals found in everyday pesticides can either mimic or interfere with the actions of these hormones (Paigen, 1995). Pesticide compounds that mimic hormones can “fool” the body into processing the chemical compound as an authentic hormone. Another mode of endocrine disruption is interference of normal hormonal processes. This differs from hormone mimicking in that the chemical compound is not necessarily structurally similar to the hormone, but blocks the natural activity and effects of the hormone (Paigen, 1995). Because most hormones act as messengers within the body to activate or deactivate biological cascades, that is a series of enzymatic events, hormone-mimicking and interfering compounds pose a significant risk to normal hormonal processes. Examples of both types of compounds are provided in the following sections.

**Hormone-Mimicking Compounds**

Pesticide compounds such as methoprene and pyriproxyfen mimic the juvenile hormone that regulates normal development in insects. Juvenile hormone diminishes as the insect matures, but the application of an increased amount of this hormone can disrupt the normal developmental process (Oberlander, Leach, Shaaya, et al, 1999). Hormone disrupting insecticides eliminate insects quite efficiently, but the unintended effects on animals and humans are often unknown. Rachel Carson (1962) elucidated in *Silent Spring* about the harmful effects of dichlorodiphenyltrichloro-ethane (DDT) exposure in wildlife. More recently, the heavily contaminated Lake Apopka in Florida was tested for health effects of DDT and its metabolites. Scientists found in 1994 that the alligator population had decreased by 90% and that the male alligators had abnormally smaller penises (Guillette, Gross, Masson, et al, 1994). The chemical dicofol was the agent in
this case. Scientists asserted that dicofol employed a hormone-mimicking mechanism to cause the gross defects in the alligators (Guillette, 1994).

Research has linked endocrine mimicking activity to human health across the lifespan. A research study conducted in 1999 showed that certain pyrethroid compounds found in pesticides can mimic the activity of estrogen and thus proliferate breast cancer cells. The use of anti-estrogen compounds halted the activity of this chemical, indicating that estrogen-like activity was indeed observed (Go, Garey, Wolff, et al, 1999). In addition, a worldwide sperm density decline was reported in 1992 and researchers in 1997 linked this phenomenon to endocrine-mimicking chemicals in the environment (Swan, Elkin, Fenster, 1997).

Although little is known of exact health effects, it is reasonable and precautionary to conclude that pesticides pose a significant threat to children. Fetuses are especially at risk because of their developing organs. Hormones play a crucial role during fetal development and any disruption by chemicals could result in permanent damage. Dioxin, for example, is a chemical compound that has been found to modulate sex steroid hormone activity in fetuses. Vreugdenhil, Slijper, Mulder, et al found that higher prenatal exposure to dioxin can influence behavior that distinguishes males from females, such as play behavior (2002). A number of researchers have linked endocrine disruption in children to developmental and neurobehavioral problems (Damstra, 2002).

**Hormone-Interfering Compounds**

Animals and humans are susceptible to anti-hormonal effects that interfere with the normal processes of the endocrine system. Vinclozolin, a fungicide used on field crops and small fruits blocks the male sex hormone androgen. Vinclozolin’s metabolites,
or byproducts, bind to the androgen receptor and prevent androgen from binding. Research with male rats has shown that this blockage leads to reduced sperm numbers and reduced prostate weight (EPA, 2000). Due to the high potency of hormones, small levels can activate these processes. In addition, many thiocarbamide and sulfonamide-based pesticides have anti-thyroid effects (Birnbaum, 1995).

**Research on Endocrine Disruptors**

The following table represents recent peer-reviewed studies on pesticides and endocrine disruption. These articles are a subset of a larger collection of articles that were found through an extensive database search for children and pesticides. The articles were then categorized according to health problem, namely endocrine disruption, developmental disabilities, and childhood cancer.

**INSERT CHART**

**Non-Governmental/Governmental Organization Reports**

According to the EPA, more than 100,000 children ingest pesticides every year (EPA, 1996). Nine out of ten American children ingest organophosphates in their food every day. Organophosphates target the brain and nervous system of the insects they kill. Despite this fact, research studies show that children’s exposure to these chemicals far exceeds the amount deemed “safe” by the EPA. Children are exposed through many routes, including drinking water. The EPA does not have adequate data on the amount of pesticides present in drinking water (NRDC: Putting Children First, 1998). Pesticide residues can also be found on foods. Winter squash, canned spinach, frozen strawberries are examples of foods that have been tested and characterized by the Consumers Union as being contaminated above the “safe” dosage level (Consumers Union, 2000).
The most susceptible population to pesticide exposure is farmworkers’ children. These children are surrounded by chemicals outside and inside the home. This is possible through parents’ clothing and chemical traces tracked into the house that can accumulate to over 100 times higher than outdoor levels (NRDC: Trouble on the Farm, 1998).

Furthermore, adult animals are usually used in research studies involving health risks to humans. When the animals are not exposed during crucial windows of development, inadequate data of the health risks to children is produced. Also, failure to follow up with an animal over their lifetime to detect any long-term effects poses a problem in accurately assessing health risks (NRDC: Putting Children First, 1998).

Endocrine disruption has been identified as a potential threat to children’s health. It has been established that many pesticides can cause endocrine disruption in laboratory and wildlife animals. Also, because only a minute amount of the chemical can mimic and/or disrupt an essential human hormone, lasting developmental defects can arise. In addition, endocrine disruptors may be linked to the onset of several reproductive cancers (EPA, 1996).

Some suggestions that have been given to the EPA are to assemble a group of unbiased experts including pediatric neurologists, endocrinologists, toxicologists, and other professionals that could assess the effects of exposure to toxic chemicals on children’s health. This professional board would assist the EPA in developing and revising relevant regulations and standards (NRDC: Putting Children First, 1998).

Other recommendations regarding pesticides in schools include notifying parents and staff prior to pesticide application. Expanding Integrated Pest Management (IPM)
programs would be a great way to reduce overall exposure to toxins. Ultimately, policymakers should stop using pesticides that are known to be carcinogenic or detrimental to reproductive and developmental processes (CPR, 1998).
Works Cited

Peer-Reviewed Articles


Sadaf Shaukat
Undergraduate Honors Thesis


**Government Reports**


**Non-Governmental Organization Reports**


Developmental Disabilities

Developmental Disabilities

Developmental disabilities cover a wide spectrum of problems including physical and mental impairments. Physical impairments include birth defects, genetic mutations, and damage to the body’s reproductive and immune systems. Mental impairments include neurodevelopmental problems that lead to visual, motor, and sensory defects. ADD and ADHD are also examples of developmental disabilities.

Not only can pesticides have an effect on the endocrine system, they can affect normal developmental processes in animals and humans. A number of pesticides have been classified as neurotoxic, including aldicarb, carbaryl, and carbofuran (U.S. EPA, 2003). According to the National Academy of Sciences, “…environmental factors…can cause approximately 25% of all developmental and neurological deficits working in combination with a genetic predisposition” (Commission on Life Sciences, 2000). One in every 200 children is estimated to have a developmental or neurological problem due to exposure to environmental toxic substances (National Environmental Trust, 2000). An estimated 24 billion pounds of neurological and developmental toxins are released in the U.S. each year; only 1.2 billion are reported to the EPA (Bogo, 2001). Pesticides account for a significant portion of toxins released into the environment each year.

Effects on Animals

Several research studies have shown that pesticides harm animal development. For example, Vartiainen found that exposure to dioxins and furans can result in decreased birth weight (1997). Dioxin, a toxic chemical used in the manufacturing of pesticides, has been linked to the onset of endometriosis of Rhesus monkeys. Endometriosis is a
condition in which endometrial cells proliferate at a site outside the uterus. Severe pain, infertility, and effects on the female gonads often result from this condition (Rier, 1993).

Organophosphate pesticide exposure has been linked to neurodevelopmental effects in animals, including deficits in motor, speed, and learning ability. Organophosphate exposure during a critical time window can cause cellular deficiencies in the brain and result in behavioral abnormalities (Eskenazi, 1999).

Effects on Adults

Similarly, organophosphate and carbamate pesticides have been found to cause deleterious effects on human development. Acute pesticide exposure has been documented to contribute to motor, verbal, and visual impairment in adults; chronic exposure effects include decreased reaction times, increased anxiety, and weakness. A likely mechanism of these effects is the inhibition of the enzyme acetyl cholinesterase (AchE). If this enzyme is inactivated, then the substance acetylcholine can accumulate at neural junctions and suppress normal neurotransmission to the body's organs. Visible symptoms might include disturbances in locomotion and balance (Eskenazi, 1999).

Chlorpyrifos®, the most widely used organophosphate pesticide, is considered suspect for inhibiting DNA synthesis in the brain and, therefore, significantly impairing neurodevelopment (Eskenazi, 1999). Furthermore, chromosome translocations in which one segment of a chromosome is rearranged with another segment of the same or another chromosome has resulted from human exposure to Ethylenebis dithiocarbamate (EBDC) fungicides (Steenland, 1997). This type of mutation can hinder normal human development and lead to birth defects.
Pyrethroids have been linked to the disruption of the immune system (Muller-Mohnssen, 1999). Damage to the immune system can predispose someone to infections that would otherwise be neutralized by normal immune system components. Health studies have found increases in pesticide exposure to be linked to increases in disease. Loevinsohn’s (1987) study in the Philippines found that adult male farmers exposed to pesticides had a higher mortality rate than adult males living in surrounding areas. Allazov (1994) found that people who lived in a pesticide application zone in Uzbekistan suffered a significantly higher rate of kidney disease (7.9%) than non-exposed residents (1.6%). Those that lived near a pesticide storage facility suffered a 12.5% rate of disease. Organophosphates inhibit esterases, which are enzymes required for the proper functioning of the nonspecific immune response. Phagocytic activity of immune cells and T-cell count has found to be reduced as a result of pesticide exposure. Phagocytes and T-cells are essential in destroying pathogens foreign to the human body, including bacteria and chemical toxins. B-cells, which are essential in producing antibodies, can be damaged from pesticide exposure.

Effects on Children

Although there have been fewer studies focusing on children than mammals and adult humans, children may even be at a higher risk for developmental problems because their bodies are constantly maturing (Repetto, 1996). A major concern with pesticide exposure is the occurrence of birth defects. Garry (2002) found that children of male pesticide applicators in the Red River Valley of Minnesota suffered from more birth defects than other children. In the general U.S. population, about 3.7% of children are born with a birth defect. In contrast, 10.1% of children in the Red River Valley were
born with a birth defect. Garry (2002) also found evidence that congenital cataracts are linked to paternal exposure of dioxin and that miscarriages may be associated to environmental toxins such as pesticides.

The inhibition of DNA synthesis can be a severe problem for children. Although adults are susceptible to this inhibition, adult bodies produce serum-binding proteins that can protect cells from the inhibitory effect. Fetuses and newborns do not have this protective mechanism, and thus have a greater chance of experiencing neurotoxicity.

A research study of Yaqui children indicated that pesticides have a significant effect on the neurodevelopment of children. The study showed that children who lived in the valley and had greater pesticide contact suffered a higher deficit in “...stamina, coordination, recall, and ability to draw a person...” compared to children living further away from pesticides in the foothills (Eskenazi, 1999, 3). Elizabeth Guillette, the principal investigator, suggested that perhaps brain dysfunction could explain the “…breakdown between visual sensory input and neuromuscular output…” (Guillette, 1998, 7).

Other developmental disabilities including hyperactivity, attention and learning deficits, and lower IQ scores have been linked to dioxin exposure (National Environmental Trust, 2000). The recent increase in ADD and ADHD cases could be related to the prevalence of toxicants in the environment.

The following chart is a summary of results from peer-reviewed articles. Several of these reports reach similar conclusions, thereby strengthening the evidence of the developmental health risks of pesticides.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Title</th>
<th>Sample/Method</th>
<th>Outcomes</th>
<th>Limitations</th>
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<th>Study Authors</th>
<th>Study Title</th>
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<th>Sample Size</th>
<th>Outcomes</th>
<th>Key Findings</th>
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<tbody>
<tr>
<td>1. Eskenazi, Bradman, Castorina (1999). Environmental Health Perspectives</td>
<td>Exposures of children to organophosphate pesticides and their potential adverse health effects</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Chlorpyrifos exposure is increasing among the general population; Birth defects including neural tube associated w/pesticide exposure; Organophosphate and carbamate exposure affects nervous sys.</td>
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<tr>
<td>2. Garry, Harkins, Erickson, Long-Simpson, Holland, Burroughs (2002). Environmental Health Perspectives</td>
<td>Birth defects, season of conception, and sex of children born to pesticide applicators living in the Red River Valley of Minnesota, USA</td>
<td>695 fungicide applicator families and 1,532 children from Red River Valley, MN; Pesticide use survey and detailed health information from families</td>
<td>70 children from 536 couples had one or more birth defects; Ratio of male births to female births lower than usual</td>
<td>Possible seasonal variance in birth defect occurrence</td>
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<td>3. Guillette, Aquilar, Soto, Garcia (1998). Environmental Health Perspectives</td>
<td>An anthropological approach to the evaluation of preschool children exposed to pesticides in Mexico</td>
<td>Yaqui children (4-5 yrs old) from the foothills and valley of Sonora, Mexico; Questionairre for mothers, interviews, standard cognitive and developmental tests</td>
<td>Yaqui valley children more exposed than foothills children; Valley children worse motor, visual, and social skills</td>
<td>N/A</td>
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<td>4. Hauser, Altshul, Chen, Ryan, Overstreet, Schiff, Christiani. (2002). Environmental Health</td>
<td>Environmental organochlorines and semen quality: Results of a pilot study</td>
<td>Semen samples from 29 subjects from Massachusetts General Hospital Andrology Laboratory; Measured sperm</td>
<td>Association suggested between abnormal sperm motility, concentration and morphology to higher</td>
<td>N/A</td>
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<tr>
<td>Perspectives</td>
<td>Concentration and sperm motility as well as presence of serum PCB or p,p'-DDE</td>
<td>Concentration of PCBs and p,p'-DDE</td>
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<td>*5. Kilburn, Thornton. (1995). <em>Environmental Health Perspectives.</em></td>
<td><em>Protracted neurotoxicity from chlordane sprayed to kill termites.</em></td>
<td>216 adults exposed to chlordane and 174 nonexposed adults; Respiratory, neurobehavioral, and rheumatic symptoms identified</td>
<td>Chlordane exposure linked to impairment of neurophysiological and psychological functions; Balance, reaction times, and verbal recall significantly impaired in exposed adults</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>6. Muller-Mohnsens. (1999). <em>Toxicology Letters.</em></td>
<td>Chronic sequelae and irreversible injuries following acute pyrethroid intoxication</td>
<td>144 adults exposed to pyrethroid; Acute and chronic symptoms assessed</td>
<td>Immune deficiency evident</td>
<td>N/A</td>
<td>N/A</td>
</tr>
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<td>*7. Qiao, Seidler, Slotkin. (2001). <em>Environmental Health Perspectives.</em></td>
<td>Developmental neurotoxicity of chlorpyrifos modeled <em>in Vitro:</em> Comparatitive effects of metabolites and other cholinesterase inhibitors on DNA synthesis in PC12 and C6 cells</td>
<td>Neurontotypic PC12 cells and gliotypic C6 cells; Compared effects of chlorpyrifos and metabolites</td>
<td>Chlorpyri-fos inhibited DNA synthesis in both cell lines, but more significant-ly on C6 cells; Addition of serum proteins protected cells which shows that fetuses and newborns are at higher risk than adults (b/c don’t possess as many serum proteins)</td>
<td>Use of transformed cell lines are less responsive to neurotoxins like chlorpyri-fos; Short time span of study, if done over longer period of time, more sensitive results</td>
<td>N/A</td>
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<td>Page</td>
<td>Authors</td>
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<td>8.</td>
<td>Rier, Martin, Bowman, Dmowski, Becker. (1993). <em>Fundamental and Applied Toxicology</em></td>
<td>Endometriosis in Rhesus monkeys (Macaca mulatta) following chronic exposure to 2,3,7,8-Tetrachlorodibenzo-p-dioxin.</td>
<td>24 female rhesus monkeys; Dioxin given in monkey feed (low-dose group and high-dose group) and control group with no dioxin exposure</td>
<td>Moderate to severe endometri-osis among dioxin exposed monkeys; Dioxin is immunosuppressive and inhibits T-cell function</td>
<td>N/A</td>
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<tr>
<td>9.</td>
<td>Steenland, Jenkins, Ames, O’Malley, Chrislip, Russo, (1994). <em>American Journal of Public Health</em></td>
<td>Chronic neurologic sequelae to organophosphate pesticide poisoning</td>
<td>128 men poisoned by organophosphate pesticides from California and 90 nonexposed men; Neurological physical test, nerve conduction tests, vibrotactile sensitivity tests, neurobehavioral tests, and postural sway test</td>
<td>Poisoned group performed significantly worse than non-exposed men on visual attention and mood scales; also worse vibrotactile sensitivity</td>
<td>Low response rate; low proportion of variation; small sample size</td>
</tr>
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<td>10.</td>
<td>Tielemans, van Kooji, te Velde, Burdorff, Heederik, (1999). <em>The Lancet</em></td>
<td>Pesticide exposure and decreased fertilization rates in vitro</td>
<td>836 couples seeking in vitro fertilization; Questionnaire on lifestyle and occupation; Further job-specific details collected</td>
<td>Couples with male partners exposed to pesticides at work had significantly lower rates of fertilization</td>
<td>Because so many chemicals involved, impossible to narrow down to one, specific chemical</td>
</tr>
<tr>
<td>11.</td>
<td>Vartiainen, Jaakkola, Saarikoski, Tuomisto. (1998). <em>Environmental Health Perspectives</em></td>
<td>Birth weight and sex of children and the correlation to the body burden of PCDDs/PCDFs and PCBs of</td>
<td>167 random breast milk samples from urban and rural areas in Finland; Detected traces of polychlorinated dibenzo-p-dioxins (PCDDs),</td>
<td>Possible correlation of presence of congeners in milk with decreased weight</td>
<td>N/A</td>
</tr>
</tbody>
</table>
the mother polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs) by mass spectrometry

Non-Governmental/Governmental Organization Reports

Because 70% of developmental defects have no known cause, researchers have been linking environmental chemical factors, such as pesticides, to developmental health concerns (CEC, 2000). The United States Environmental Protection Agency uses a four-step process for health risk assessment of pesticides. The steps include Hazard Identification, Dose-Response Assessment, Exposure Assessment, and Risk Characterization. The first step, Hazard Identification, is performed in order to characterize the type of human health effect a certain pesticide can have. The second step is Dose-Response Assessment which involves determining a safe human dose level based on animal tests. Exposure Assessment is the third step and this could involve the following routes of exposure: inhalation, dermal exposure, and oral exposure. The final step is Risk Characterization and this involves assessing the overall risk of the pesticide based on the previous three steps (EPA, 1999). Children are often not taken into account in these standard risk assessment methods and therefore are not protected by developed policies and laws. Inclusive methodologies that can account for the increased susceptibility of children to pesticides need to be developed (CEC, 2000).

Pesticides are routinely applied in schools and rarely are the parents of the schoolchildren informed. An Integrated Pest Management program is recommended for schools. IPM strategy calls for alternative and preventive measures for pest control and
provides parents with prior written notification to necessary pesticide application (CHEJ, 2001).
Works Cited


Center for Health, Environment and Justice. (March 2001). *Poisoned schools, invisible threats, visible actions*. Falls Church, VA: Child Proofing Our Communities Campaign.


A pesticide is a substance or mixture of substances intended for destroying, repelling, or mitigating any pest. The ingredients of a pesticide are generally classified as "active" or "inert". There are approximately 890 active pesticide ingredients in over 20,000 pesticide products. The chart below identifies the major classes of inert and active ingredients. The chart highlights the classes of insecticides and summarizes key uses, biological mechanisms, and acute and chronic symptoms. Symptoms range from headache and nausea to organ failure and death. The pesticides linked to childhood health effects on the "Definitions" page can be found below.

Abstract

Until recently, children have been overlooked often in research and legislation concerning pesticides. This paper, based on an extensive literature review, examines emerging evidence regarding the health effects of pesticides on children.

A comprehensive bibliography, including peer-reviewed research articles, government documents, non-governmental organization documents, books, and other sources was developed (http://utyeah.utk.edu). Research methods used to study routes of exposure and the certainty and uncertainty of health effects of pesticides - with a focus on children's well-being - are discussed.

The three health problems of endocrine disruption, developmental disabilities, and childhood cancers are reviewed. Studies regarding these three health conditions in children, adults, and animals were analyzed in the current research. The rationale of the precautionary principle, especially taking into consideration children's increased susceptibility, is explained. Practice, policy, and research interventions are suggested for individuals concerned with children's well-being in homes and child-serving facilities.

Sadaf Shaukat, The University of Tennessee Department of Microbiology
Undergraduate Honors Thesis

Mary E. Rogge, Ph.D. Associate Professor, The University of Tennessee College of Social Work
Thesis Advisor
Definitions

Pest: Any living thing that exists where it is not wanted

Endocrine Disruption: The process of a chemical substance mimicking or interfering with the hormone system of an organism. Health effects include early onset of puberty, decline of sperm count, and underdevelopment of sexual organs.

*Precocious puberty in young girls from Belgium linked to exposure to organochlorine (p,p'-DDE) pesticides (Krstevska-Konstantinova, et. al, 2001)

*Dieldrin is an endocrine disruptor and can travel across the placenta and accumulate in the fetus; It is also transferred to the child via breast milk (Jorgenson, 2001)

Developmental Disability: A broad spectrum of defects including physical, mental, genetic, and reproductive disabilities. Health effects include birth defects, genetic mutations, and neurodevelopmental problems.

*Chlorpyrifos, now banned by the EPA, is still found on children's toys and in children's urine samples. Chlorpyrifos associated with decreased DNA synthesis in the brain and to attention deficit hyperactivity disorder. (ADHD; Schettler, et. al, 2000)

*Dioxin exposure has been linked to lower IQ scores, hyperactivity, and learning disorders. (National Environmental Trust, 2000)

Cancer: A potentially fatal condition brought about by the unregulated proliferation of cells. Childhood cancers include acute lymphoblastic leukemia and non-Hodgkin's lymphoma.

*Some organochlorine pesticides are associated with non-Hodgkins lymphoma. (Safi, 2002)

*Exposure to indoor household pesticides at an early age has been linked to an elevated risk of childhood leukemia (Ma, X. et. al, 2002)

Precautionary Principle: Risk assessment standards that build on, yet contrast with, traditional scientific risk assessment methods (e.g., risk averaging methods that estimate adult effects), by estimating effects on the most vulnerable children.

*The 1996 U.S. Food Quality Protection Act mandates pesticide residue safety standards that use a tenfold margin (10x greater than adult standards) of safety for infants and children.
Methods

This project was based on a comprehensive literature review of peer-reviewed articles, government reports, non-governmental organization reports, books, and other sources. An existing body of literature from the University of Tennessee Youth Environment and Health (UT YEAH) Research Team was used and supplemented. Additional updated literature was obtained from database searches, mainly utilizing the “Web of Science” database through the University of Tennessee-Knoxville Hodges Library system. The keywords “children” and “pesticides,” “cancer,” “endocrine disruption,” “developmental disabilities,” and associated keywords were used.

The resulting 35-page bibliography, was further organized by the three major health effects of endocrine disruption, developmental disabilities, and childhood cancer. Comparative charts, including the methods, outcomes, and limitations of the peer-reviewed articles, were created. Each section of the paper was developed using these charts and other selections from the subdivided bibliography.
Implications

Practice: Individuals concerned with children's well-being in homes, schools, child care, and other child serving facilities can:

- Assess with children and their caretakers what pesticides or other chemicals are in their homes, neighborhoods, and facilities where they spend time.
- Arrange age- and constituent-appropriate educational venues (e.g., workshops, brown-bags, other training) that involve interdisciplinary experts and identify quality Internet and other resources.
- Document suspected pesticide and other chemical exposures among children; seek out and promote appropriate medical assessment and treatment.
- Promote the use of tools such as integrated pest management (IPM) and the U.S.E.P.A's Indoor Air Quality "Tools for Schools" resource kit (see http://utyeah.utk.edu).

Policy: Parents, age-appropriate children and youths, social service and health care providers, educators, public health officials, environmental scientists and others can:

- Collaborate and advocate, through community and professional groups, for child-serving facilities, local-national legislative bodies, and other policy-makers to use the precautionary principle to supplement state-of-the-art information about how to reduce and balance the risk of children's exposure to pests, pesticides, and other chemicals.

Research: Physical, biological, environmental, social and other scientists can promote the increase of research that:

- Targets children's unique vulnerabilities and routes of exposure, with a focus on the most vulnerable children, such as children of color and children living in poverty.
- Addresses current research limitations such as inadequate sampling of potential sources of contamination, multiple chemical effects, and synergistic effects of pesticides and other chemicals.
KIDS AND PESTICIDES BIBLIOGRAPHY:
EXCERPTED CHILDHOOD CANCER, ENDOCRINE DISRUPTION,
AND DEVELOPMENTAL DISABILITIES RESEARCH RESOURCES

AUGUST 2002

CHILDHOOD CANCER

CCI. CHILDHOOD CANCER FULL ARTICLES

CCI. A. Peer-Reviewed Articles
CCI. B. Government Reports
CCI. C. Non-Government Organization Reports
CCI. D. Books/Book Chapters/Monographs
CCI. E. Magazines/Newsletters/Newspapers
CCI. F-CCI.G.: N/A

CCII. CHILDHOOD CANCER ABSTRACTS

CCII.A-CCII.G.: N/A

CCIII. CHILDHOOD CANCER TITLE-ONLY

CCIII.A-CCIII.G.: N/A

ENDOCRINE DISRUPTION

ED1. ENDOCRINE DISRUPTION FULL ARTICLES

ED1. A. Peer-Reviewed Articles
ED1. B. Government Reports
ED1. C. Non-Government Organization Reports
ED1. D. Books/Book Chapters/Monographs
ED1. E. Magazines/Newsletters/Newspapers
ED1. F. Testimony/Presentations
Children's Unique Vulnerabilities

- **Intergenerational:** Grandparents' and parents' exposure to pesticides can affect children through sperm and ova development (grandparent exposure) and placenta and breast milk transfer. (mother exposure)

- **Developmental Stages:** Children during fetal, neonatal, infant, preschool, school, and adolescent stages are more susceptible to harmful chemicals due to immature metabolic systems.

- **Environmental:** Rate of food, water, and air consumption; behavior (e.g. Children live closer to ground and floor where chemicals accumulate, have high hand-to-mouth behavior, have contact with pets and toys that may be contaminated).
EDII. ENDOCRINE DISRUPTION ABSTRACTS

EDII. A. Peer-Reviewed Articles

EDII. B.-EDII. G.: N/A

EDIII. ENDOCRINE DISRUPTION TITLE-ONLY

EDIII. A.-EDIII. G.: N/A

DEVELOPMENTAL DISABILITIES

DDI. DEVELOPMENTAL DISABILITIES FULL ARTICLES

DDI. A. Peer-Reviewed Articles

DDI. B. Government Reports

DDI. C. Non-Government Organization Reports

DDI. D. Books/Book Chapters/Monographs

DDI. E. Magazines/Newsletters/Newspapers

DDI. F. Testimony/Presentations

DDI. G.: N/A

DDII. DEVELOPMENTAL DISABILITIES ABSTRACTS

DDII. A. Peer-Reviewed Articles

DDII. B.-DDII. G.: N/A

DDIII. DEVELOPMENTAL DISABILITIES TITLE-ONLY

DDIII. A.-DDIII. G.: N/A
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CCI. A. Peer-Reviewed Articles


CCI. B. Government Reports


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*Center for Health, Environment and Justice. (March 2001). Poisoned schools, invisible threats, visible actions*. Falls Church, VA: Child Proofing Our Communities Campaign.


**CCI. D. Books/Book Chapters/Monographs**


**CCI. E. Magazines/Newsletters/Newspapers**


**CCI.F-CCI.G.:** N/A
CCII. CHILDHOOD CANCER ABSTRACTS

CCII.A-CCII.G.: N/A

CCIII. CHILDHOOD CANCER TITLE-ONLY

CCIII.A-CCIII.G.: N/A
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**EDI, A. Peer-Reviewed Articles**


**EDI. B. Government Reports**


**EDI. C. Non-Government Organization Reports**


**EDI. D. Books/Book Chapters/Monographs**


**EDI. E. Magazines/Newsletters/Newspapers**


ED1. F. Testimony/Presentations


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EDII. A. Peer-Reviewed Articles


EDIII. ENDOCRINE DISRUPTION TITLE-ONLY

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DDI. D. Books/Book Chapters/Monographs


DDI. E. Magazines/Newsletters/Newspapers


DDI. F. Testimony/Presentations

DDII. DEVELOPMENTAL DISABILITIES ABSTRACTS

DDII. A. Peer-Reviewed Articles


DDIII. DEVELOPMENTAL DISABILITIES TITLE-ONLY

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**Other**


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