A Generation in Jeopardy

How pesticides are undermining our children's health & intelligence

PESTICIDE ACTION NETWORK NORTH AMERICA

Pesticide Action Network North America

Pesticide Action Network North America (PAN North America) works to replace the use of hazardous pesticides with ecologically sound and socially just alternatives. As one of five PAN Regional Centers worldwide, we link local and international consumer, labor, health, environment and agriculture groups into an international citizens' action network. This network challenges the global proliferation of pesticides, defends basic rights to health and environmental quality, and works to ensure the transition to a just and viable society.

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A Generation in Jeopardy

Executive Summary

Children today are sicker than they were a generation ago. From childhood cancers to autism, birth defects and asthma, a wide range of childhood diseases and disorders are on the rise. Our assessment of the latest science leaves little room for doubt: pesticides are one key driver of this sobering trend.

As the recent President's Cancer Panel reports, we have been "grossly underestimating" the contribution of environmental contamination to disease, and the policies meant to protect us have fallen far short. Nearly 20 years ago, scientists at the National Research Council called for swift action to protect young and growing bodies from pesticides.[1] Yet today, U.S. children continue to be exposed to pesticides that are known to be harmful in places they live, learn and play.

This report reviews dozens of recent studies that examine the impact of pesticides on children's health. Our analysis reveals the following:

- **Compelling evidence now links pesticide exposures with harms to the structure and functioning of the brain and nervous system.** Neurotoxic pesticides are clearly implicated as contributors to the rising rates of attention deficit/hyperactivity disorder, autism, widespread declines in IQ and other measures of cognitive function.
- **Pesticide exposure contributes to a number of increasingly common health outcomes for children, including cancer, birth defects and early puberty.** Evidence of links to certain childhood cancers is particularly strong.
- **Emerging science suggests that pesticides may be important contributors to the current epidemic of childhood asthma, obesity and diabetes.**
- **Extremely low levels of pesticide exposure can cause significant health harms,** particularly during pregnancy and early childhood.

Children's developing bodies are particularly vulnerable to the health harms of pesticides.

Prioritizing children's health requires real change

As a nation, we value the wellbeing of our children. In addition to our natural urge to protect what we love, we know that at a societal level their successful development is key to a vibrant, secure future. Poll after poll shows more than 80 percent of Americans consider healthy children a top priority. We must line up our practice and policies with these values.

Many communities across the country have stepped up to create local or state policies to protect children from pesticide exposure. From pesticide-free schools, parks and playgrounds to protective buffer zones in agricultural areas, locally-driven actions are leading the way to healthier childhood environments.

But to ensure protection of all children from the harms of pesticides, we must dramatically reduce the use of these chemicals nationwide. An estimated 1.1 billion

pounds of pesticides are used in the U.S. every year, with more than 20,000 products on the market. This volume of use is undermining the health of the next generation and, as the science demonstrates, derailing development of our children's potential.

Scientists have understood for decades that children are particularly vulnerable to the harms of pesticide exposure. Quickly growing bodies take in more of everything; they eat, breathe and drink more, pound for pound, than adults. As physiological systems undergo rapid changes from the womb through adolescence, interference from pesticides and industrial chemicals—even at very low levels—can derail the process in ways that lead to significant health harms.

Reducing overall pesticide use would not only limit children's exposure during their most vulnerable years, it would also lower pesticide levels in the bodies of men and women of childbearing age—protecting current and future generations in one fell swoop. Those pesticides most harmful to children should be first on the list.

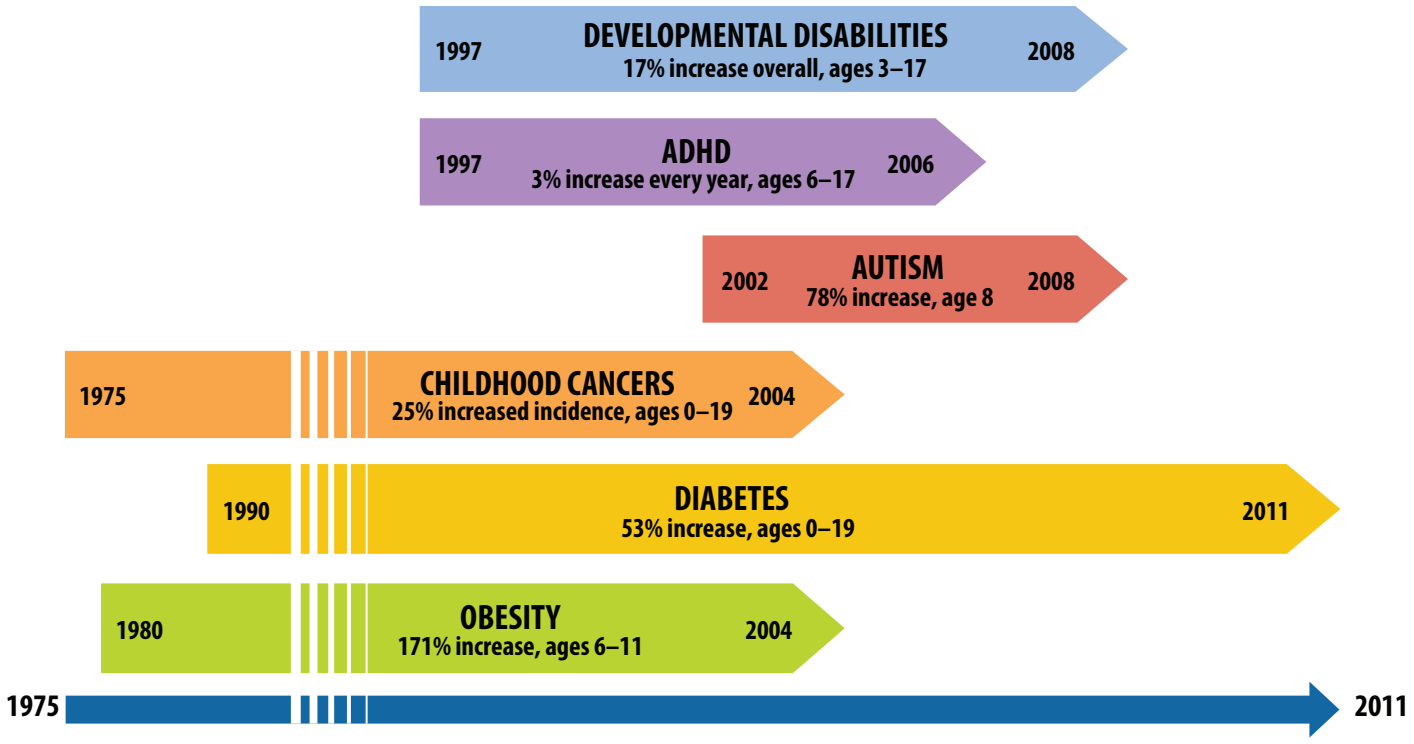
While we must each do what we can with food choices and decisions about home pest control, we cannot accomplish this goal at an individual household level. Policy change is required.

Effective policies urgently needed

To protect children from the health harms of pesticides, policymakers need much more effective tools. We believe change is most urgently needed in the way decisions are made about these three questions:

- Which pesticides are used in agriculture?
- Which pesticides are used in places children live, learn and play?
- How are farmers supported as they reduce reliance on pesticides?

Figure 1: Children's Health Harms on the Rise, 1975–2011*



Statistics show steady increases in many childhood diseases and disorders over the past 30 years. Those highlighted here are just some of the health harms on the rise.
Sources: see endnotes 4, 13,24, 52 and 94.

* With the exception of cancer, all other data are prevalence data, i.e., representing the U.S. population or based on data at several sites within the U.S. Prevalence is total number of cases in a population at a given time, while incidence is a measure of the number of new cases per year. The autism data are from 14 sites in the Autism and Developmental Disabilities Monitoring Network and are not considered fully representative of the U.S. population. The 1990 diabetes data are for type 1 only (type 2 being extremely rare among children at that time), while 2011 data include both type 1 and 2. Prevalence of type 2 diabetes among children is difficult to determine for various reasons, including difficulty of diagnosis.

We recommend the following policy changes in each of these arenas:

1. Prevent the pesticide industry from selling agricultural products that can harm children's health

- *Take swift action on existing pesticides:* If studies find a pesticide to be a neurodevelopmental or reproductive toxicant, endocrine disruptor or human carcinogen—*and* it has been measured in humans, in schools or homes, or as residues on food or in drinking water—EPA should target the pesticide for rapid phaseout, triggering USDA resources to assist rapid farmer transitions to safer pest control methods.

- *Block harmful new pesticides:* EPA should not approve any new pesticide that scientific studies suggest is a neurodevelopmental or reproductive toxicant, endocrine disruptor or human carcinogen—including short-term "conditional" registrations.

- *Prevent harmful low-level exposures:* EPA should act on existing evidence that exposures to endocrine disrupting pesticides pose a particular danger to developing children; the long-delayed endocrine disruptor screening program (EDSP) should be swiftly implemented.

2. Protect children where they live, learn & play

- *Kid-safe homes, daycares & schools:* EPA should withdraw approval of existing pesticide products and not approve new pesticides for use in homes, daycare centers or schools when scientific evidence indicates the chemicals are possible neurodevelopment or reproductive toxicants, endocrine disruptors or human carcinogens.

- *Safer parks & playgrounds:* State and local officials should enact policies requiring that all public playgrounds, playing fields and parks be managed without using pesticides that studies show are harmful to children's health.

Table 1: Pesticides & Childhood Health Harms

		Childhood Health Harms*					
		Brain & nervous system impacts	Childhood cancers	Birth defects	Reproductive & developmental harms	Metabolic effects (e.g., obesity, diabetes)	Immune disorders, asthma
Pesticides	**Herbicides** 442 million lbs † *e.g., atrazine, glyphosate, 2,4-D*	✓	✓	✓	✓		✓
	Insecticides 65 million lbs *e.g., chlorpyrifos, malathion, permethrin*	✓	✓		✓	✓	✓
	Fungicides 44 million lbs *e.g., mancozeb, chlorothalonil*	✓	✓	✓	✓		✓
	Fumigants 108 million lbs *e.g., metam sodium, methyl bromide, chloropicrin*	✓	✓		✓		

Researchers have linked exposure to various pesticides with a range of childhood health harms. A ✓ indicates that links to the health harm are particularly well supported by scientific evidence.

* See Appendix A and www.pesticideinfo.org

† 2007 use estimates, refers to "active ingredient." From *Pesticide Industry Sales & Usage, 2006 and 2007 Market Estimates,* U.S. EPA, Washington, DC, Feb 2011. See www.epa.gov/opp00001/pestsales/07pestsales/market_estimates2007.pdf. Table 3.4.

3. Invest in farmers stepping off the pesticide treadmill

- *Corral resources for farmers:* Federal and state officials should mobilize and coordinate existing resources to help farmers adopt well-known, effective pest management strategies that reduce reliance on pesticides.

- *Increase investment in innovative farming:* Congress should authorize significant funding for programs supporting farmers' adoption of sustainable practices that reduce use of harmful pesticides.

- *Set use reduction goals:* EPA and USDA should set specific and aggressive national pesticide use reduction goals, focusing first on pesticides that studies show to be harmful to children. To track progress toward this goal, farmers should work with applicators and pest control advisors to report their pesticide use to a nationally searchable database.

- *Source for children's health:* Food distributors should require that their suppliers limit use of pesticides that harm children's health.

These proposals are all common-sense measures in the face of clear evidence that our children's wellbeing is at risk. It's time to muster the political will to prioritize the health of our children, grandchildren and future generations.

Even at very low levels, pesticide exposure can derail development and undermine the ability to learn.

1 Brainpower at Risk

New studies find pesticides can compromise intelligence

Knowledge of environmental causes of neurodevelopmental disorders is critically important because they are potentially preventable. — Dr. Philip Landrigan

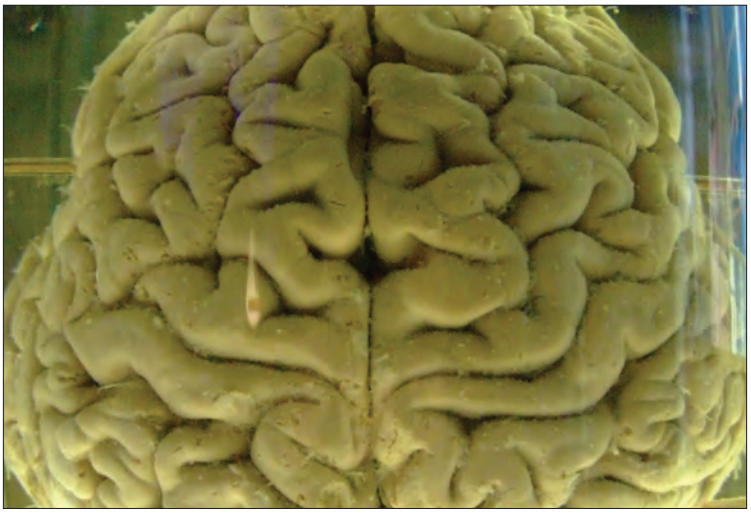
The process of establishing the architecture of the human brain begins in the womb and continues into early adulthood. During this long window of development, many complex processes take place, involving tens of billions of nerve cells making trillions of connections. Cells migrate from one section of the brain to another, and nerve tracts are laid as the final structure of the brain is created.

Many of the processes that occur during brain development are vulnerable to disruption from pesticides. Exposure to neurotoxic pesticides during critical moments of fetal development, even at very low levels, has been shown to fundamentally alter brain architecture.[2] Pesticides that disrupt the hormone system—and particular those affecting the functioning of the thyroid, which plays a key role in brain development—can cause lasting damage. The impacts of exposures are often irreversible because unlike other organs, the brain cannot repair damaged cells (see sidebar).

Children whose brain infrastructure or nervous system fails to develop normally may be disabled for the rest of their lives. Developmental disabilities include autism spectrum disorders, attention deficit disorders, hearing loss, intellectual impairment and vision loss. People with developmental disabilities are often challenged by everyday life activities such as language, mobility, learning and independent living. Reduced cognitive abilities can also lead to behavioral problems, from aggression and social alienation to increased risk of drug abuse.[3]

A "Silent Pandemic"

Some 15 percent of all U.S. children have one or more developmental disabilities—representing a 17 percent increase in the past decade. For some disorders, the numbers are rising even more rapidly.[4] Overall, researchers estimate that between



Pesticides can interfere with brain function in several ways, from altering architecture during fetal development to interfering with neurostransmitter control. Gaetan Lee

Mechanisms of Harm

Misfiring neurons & altered brain architecture

Pesticides can interfere with brain function and development in several ways; we describe three of the most common and best understood mechanisms of harm here:

Neurotransmitter control: Organophosphate pesticides can block the normal functioning of acetylcholinesterase, an enzyme that degrades—and thus controls—a neurotransmitter called acetylcholine. When the functioning of the enzyme is blocked, acetylcholine is not degraded and neurons continue firing instead of shutting down after they've accomplished their mission. This can cause serious problems in the normal functioning of the nervous system.

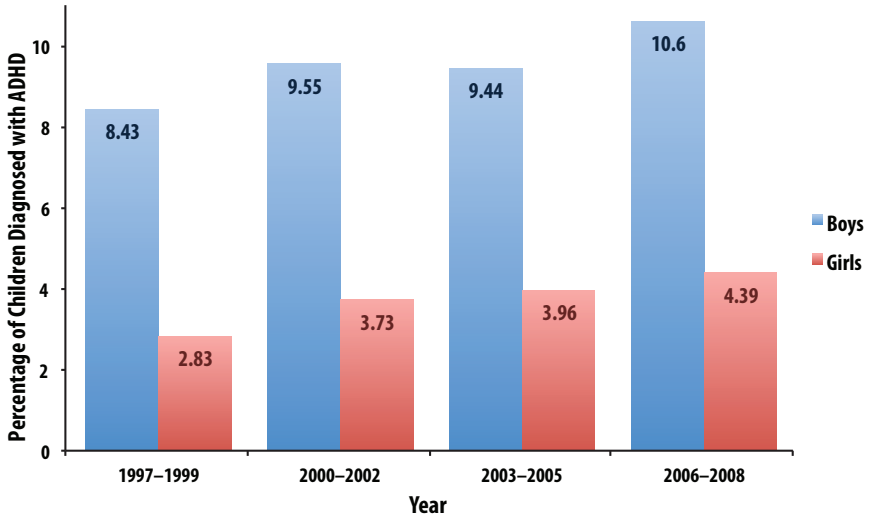
Developing brain cells: To date, EPA assessments have relied on acetylcholinesterase levels as a marker of organophosphate exposure risk, yet studies now show adverse effects can occur at much lower doses than those that block acetylcholinesterase. For example, chlorpyrifos has been shown to interfere with neural cell replication, differentiation and survival. As the brain structure is developing—particularly at key stages *in utero*—chlorpyrifos can disrupt the process in ways that permanently alter the architecture of the brain.*

Sodium flow into nerve cells: Pyrethroid insecticides act on neurons by perturbing voltage-sensitive sodium channels. These sodium "gates" are what allow sodium to flow into a nerve cell, controlling how a neuron fires and transmits signals along a nerve. Pyrethroids cause these gates to open and close more slowly, changing how the nerve cell normally responds—either inducing repetitive firing or causing the nerve cell not to fire at all.†

* Rauh, V. A., F. P. Perera, M. K. Horton, R. M. Whyatt, R. Bansal, X. Hao, et al. "Brain Anomalies in Children Exposed Prenatally to a Common Organophosphate Pesticide." *Proceedings of the National Academy of Sciences.* May 2012 109 (20): 7871-6. See http://www.pnas.org/cgi/doi/10.1073/pnas.1203396109.

† Shafer T.J., D.A. Meyer and K.M. Crofton. "Developmental neurotoxicity of pyrethroid insecticides: critical review and future research needs." *Environ Health Persp.* Feb 2005 113(2):123-36. See http://www.ncbi.nlm.nih.gov/pubmed/15687048.

Figure 2: ADHD Prevalence among Children Ages 3 to 17, from 1997–2008



The number of children diagnosed with ADHD increased an average of 3 percent every year from 1997 to 2008. Boys are much more likely to be affected. Source: C. Boyle et al., "Trends in the Prevalence of Developmental Disabilities in U.S. Children, 1997– 2008."

400,000 and 600,000 of the four million U.S. children born each year are affected by a neurodevelopmental disorder.[5]

Public health experts from Harvard and Mt. Sinai Hospital have called the damage that chemicals are causing children's developing minds a "silent pandemic,"[6] and scientists now point to a combination of genetic and environmental factors to explain this rapid rise of developmental, learning and behavioral disabilities.[7]

Some children, for example, may have a genetic susceptibility to attention deficit/hyperactivity disorder (ADHD) or autism, but it may only develop if the child is exposed to a triggering chemical during a certain period of development. Other children may be genetically programmed to produce less of a common detoxifying enzyme, rendering their brain and nervous system more susceptible to lasting harm when they are exposed to neurotoxic pesticides (see sidebar, p. 25).[8]

Genetic mutations that occur in parents (both men and women) in response to chemical exposures over the course of their lifetime can also, according to recent research, raise the risk of neurodevelopmental disorders for their children.[9, 10]

The National Academy of Sciences now estimates that about one third of all neurobehavioral disorders (such as autism and ADHD) are caused either directly by pesticides and other chemicals or by interaction between environmental exposures and genetics.[11] Some experts say this estimate is likely to be low, as the health profession is just beginning to fully recognize the contributions of environmental factors to disease formation.*

Whatever the mechanism of harm, recent studies leave little doubt that exposures to pesticides during fetal development, infancy and childhood may contribute significantly to decline in the cognitive abilities of our children. A recent comprehensive review of the science on health effects of pesticides by the Ontario College of Family Physicians found exposure to pesticides in the womb to be "consistently associated with measurable deficits in child neurodevelopment."[12]

We look here at three areas where the evidence is particularly strong: ADHD, autism and falling IQs. A few of the key studies are highlighted below, and more detailed descriptions—along with additional studies—are provided in Appendix A.

ADHD rates continue to rise

ADHD is quite clearly on the rise, and though changes in diagnosis play a role, this cannot fully explain the trend. The number of children diagnosed with ADHD increased an average of three percent every year from 1997 to 2006, and an average 5.5 percent per year from 2003 to 2007 (see Figure 2).[13, †]

The Centers for Disease Control and Prevention (CDC) estimates that ADHD now affects three to seven percent of all school children in the U.S.; one independent study puts the figure at 14 percent.[14] Boys are much more likely to be diagnosed with ADHD, although the American Psychological Association notes that girls are more likely to suffer from the "attention deficit" part of the disorder, and their symptoms are often overlooked.[15]

A variety of brain functions are compromised in children exhibiting ADHD. Learning is often impaired, and those with the disorder may exhibit impulsive behavior and hyperactivity, and lack the ability to sustain attention.

As with other neurodevelopmental disorders, the social impacts can be immense. Parents report that children with ADHD have almost three times as many problems interacting with peers as children without. Diagnosed children are almost 10 times as likely to have difficulties that interfere with friendships, including experiencing exclusion from peer groups.[16]

The Science

Researchers estimate that from 20 to 40 percent of ADHD cases are caused by something other than genetics.[17] Studies have found links to a variety of environmental contaminants, including exposure to organophosphate and pyrethroid insecticides during pregnancy and throughout childhood.

* See for example the 2010 President's Cancer Panel report "Reducing Environmental Cancer Risk: What we can do now" http://deainfo.nci.nih.gov/advisory/pcp/annualReports/index.htm.

† The CDC outlines diagnostic criteria here: http://www.cdc.gov/ncbddd/adhd/diagnosis.html, specifying that children must display at least six characteristic behaviors within six months, and that some symptoms must be present before the age of seven. CDC explains shifts in diagnostic criteria here: http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5810a1.htm.

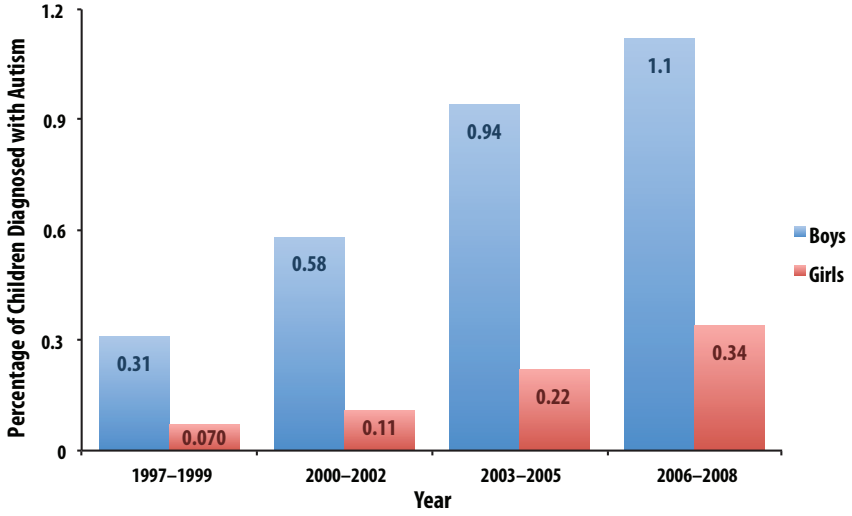
- Children with higher levels of organophosphate breakdown products in their urine were more likely to have ADHD. Researchers found that 94 percent of the 1000+ children tested by CDC had detectable levels of these metabolites, and those with levels above the median were twice as likely to be diagnosed with ADHD as those with no metabolites found.[18]
- Organophosphate metabolites at levels commonly found in the bodies of U.S. children are linked to increased likelihood of ADHD. Every 10-fold increase in levels of organophosphate metabolites in the urine of children aged eight to 15 years was associated with a 55 to 72 percent increased likelihood of the disorder.[19]
- Prenatal organophosphate exposure has been linked to attention problems. Each ten-fold increase in a pregnant mother's urinary concentration of organophosphate metabolites led to a five-fold increased risk that her child would be diagnosed with ADHD by age five.[20]
- Children with low birth-weight are more likely to have ADHD,[21] and there is considerable evidence linking reduced birth-weight with prenatal exposure to organophosphate pesticides.[22]
- Mouse pups were hyperactive after being exposed to the pyrethroid insecticides pyrethrin or cypermethrin, and adult mice injected with permethrin or deltamethrin had long-term elevation of the dopamine transporter, a marker that has been linked to ADHD.[23]

Table 2:
Chemicals Contributing to Autism

- Lead
- Methylmercury
- Polychlorinated biphenyls
- **Organophosphate pesticides**
- **Organochlorine pesticides**
- **Endocrine disruptors**
- Automotive exhaust
- Polycyclic aromatic hydrocarbons
- Brominated flame retardants
- Perfluorinated compounds

This list from public health experts includes both commonly used organophosphate pesticides and long lasting organochlorine pesticides, as well as other chemicals commonly found in consumer products. Source: Landrigan, et al., 2012

Figure 3: Autism Prevalence among Children Ages 3 to 17, from 1997–2008



Rates of autism have risen dramatically in the past decade. While overall prevalence is higher among boys, the rate of increase is higher among girls. Source: C. Boyle et al, "Trends in the Prevalence of Developmental Disabilities in U.S. Children, 1997–2008."

Autism rates jump 250% in one decade

The autism spectrum includes classic autism, Asperger's Syndrome and atypical autism. Incidence rates have risen rapidly in recent years; in its 2012 report, CDC estimated—based on 2008 data on eight-year-olds from 14 states—that 1.1 percent of U.S. children, or one in every 88, are now on the autism spectrum. Boys are more likely to have the disorder, with one in 54 affected.

Data from the National Health Interview Surveys reveal a dramatic rate of increase. Between 1997 and 2008, autism prevalence among boys ages three to 17 years increased 261%. Prevalence among girls, while much lower than boys overall, rose even more quickly, showing an increase of more than 385% over the same period (see Figure 3).[24]

In California, the number of children with autism who are enrolled in statewide programs rose from 3,864 in 1987 to 11,995 in 1998, an increase of more than 210 percent in 11 years.[25] Other states saw similar rates of increase between 2002 and 2006.[26] Though shifts in diagnosis account for some of this dramatic rise, public health experts have determined that diagnostic changes do not fully explain the trend.

Researchers believe autism spectrum disorders reflect changes in brain structure occurring during critical windows of development in the womb. These shifts in brain architecture may be caused by genetics, environmental insults such as chemical exposure, or an interaction between the two.[27, 28]

In 2012, a group of researchers led by Dr. Philip Landrigan of Mt. Sinai Medical Center released a list of ten types of chemicals most likely to be linked to the development of autism (see Table 2), and laid out an urgent strategy for research into the role of these contaminants and how children can be better protected from them. The list includes both commonly used organophosphate pesticides and longlasting organochlorine

pesticides, as well as other chemicals commonly found in consumer products.[29]

The Science

Studies examining the links between pesticide exposure and autism suggest prenatal exposures are particularly damaging.

- One study in California's Central Valley found that when mothers were exposed early in pregnancy to the organochlorine pesticides endosulfan and dicofol, the risk of autism among their children increased sharply. Children whose mothers lived within 500 feet of fields being sprayed were six times more likely to be on the autism spectrum.[30]
- Mothers in California's central coast region who had higher levels of organophosphate metabolites in their urine during pregnancy were much more likely to have children with pervasive developmental disorder—which can include or be an indicator of autism. The risk more than doubled each time metabolite concentrations went up by a factor of 10.[31]
- A study in New York City found that infants most exposed to chlorpyrifos *in utero* were significantly more likely to have pervasive developmental disorders—including autism—by the time they were three years old.[32]
- A trio of U.S. studies examined links between environmental exposures among parents (including, but not limited to, pesticides) and incidence of autism among their children.[33] Among other findings, the scientists reported that older fathers are more likely to transmit tiny, spontaneous gene mutations—that occur over a lifetime in response to environmental stressors—to their offspring, that in turn increase the risk of autism. Recent research in Iceland confirmed these findings.[34]
- Minnesota researchers explored the interaction of exposure to organophosphate pesticides, gene expression and dietary factors as potential contributors to autism.[35] Among other things, they found that mineral deficiencies linked to high fructose corn syrup consumption* make developing minds more susceptible to the neurotoxic effects of pesticides.

These various recent studies show how complex the path to our current autism epidemic has been. But evidence suggests that pesticide exposure—particularly during pregnancy—is implicated in a number of ways.

Derailed brain development means falling IQs

The societal implications of reduced cognitive abilities across an entire generation are nothing short of staggering and have been a concern among public health specialists since the IQ effects of lead exposure became clear in the 1970s. As Dr. Ted Schettler observed back in 2000:

> A loss of five points in IQ is of minimal significance in a person with an average IQ. However a shift of five IQ points in the average IQ of a population of 260 million increases the number of functionally disabled by over 50 percent (from 6.0 to 9.4 million), and decreases the number of gifted by over 50 percent (from 6.0 to 2.6 million).[36]

Twelve years later, Dr. David Bellinger echoed this observation. He pointed out that cognitive effects, often dismissed as "clinically unimportant" at the individual level, become very significant across a whole society in terms of declining intellectual capacity, lost economic productivity and increased costs for education and health care.

Bellinger reviewed published data linking organophosphates and cognitive effects, and concluded that overall, exposure to organophosphate insecticides may be responsible for lowering U.S. children's IQ level† by 17 million points—not much less than the 23 million point loss attributed to lead poisoning.[37]

Bellinger argues that because the potential impacts of organophosphates are so widespread and significant to society, "a risk assessment that focuses solely on individual risk, and fails to consider the problem in a public health context" is misleading and will not lead policymakers to sound and protective decisions.

The Science

Pesticide exposure during pregnancy can have dramatic effects on cognitive development. From a wide range of animal research to studies tracking the intellectual development of children over time, the evidence points squarely at prenatal pesticide exposures as significantly harming the development and functioning of the brain. These harms can then lead to both lower IQ levels and neurodevelopmental delays.

- A particularly compelling study used Magnetic Resonance Imaging (MRI) technology to observe the developing brains of infants who had been exposed to chlorpyrifos during pregnancy. Researchers observed significant structural changes, including abnormal areas of thinning and enlargement. Areas of the brain related to attention, language, reward systems, emotions and control were affected.[38]
- Three cohort studies‡ released in 2011 document cognitive impairment caused by exposure to organophosphates in the womb.§ The first study found that higher metabolite levels in a mothers' urine late in pregnancy increased the likelihood of reduced cognitive development in their children.[39] The second study linked prenatal exposure to a seven-point reduction in IQ by age seven.[40] The third study found that even very low levels of chlorpyrifos residues in cord blood resulted in lower IQ and reduced working memory.[41]
- Pregnant mothers exposed to chlorpyrifos through household use (before this use was withdrawn)¶ had infants with lower birth weight and reduced head circumference, both indicators of impaired cognitive ability later in childhood.[42]

* High fructose corn syrup is found in a wide range of processed foods and beverages.

† The accuracy of Intelligence Quotient (IQ) testing to measure intellectual capacity has long been a source of contention, but IQ is currently the best index for measuring cognitive abilities across a population.

‡ See sidebar in Appendix A for a description of the various types of scientific studies highlighted in this report.

§ See this editorial in *Environmental Health Perspectives* for a discussion of the importance of these three studies: "Strength in Numbers: Three Separate Studies Link in Utero Organophosphate Pesticide Exposure and Cognitive Development," available online at: http://ehp03.niehs.nih.gov/article/fetchArticle.action?articleURI=info%3Adoi%2F10.1289%2Fehp.1104137

¶ Chlorpyrifos was withdrawn from home use in 2001, but remains widely used in agricultural settings where farm, farmworker and rural community mothers and children still face exposure. Children also continue to be exposed from residue on fruits and vegetables.

- Exposure to the organophosphate pesticides diazinon and parathion during early childhood may reduce cognitive function, according to results from animal studies. Low-dose exposures caused changes in the developing brains of rats known to correspond to reduced ability to learn.[43] Other animal studies indicate that *in utero* and neonatal exposure to organophosphates increases the risk of developmental delays.[44]
- Children at three months of age who were most highly exposed to the pyrethroid pesticide synergist piperonyl butoxide,* as assessed by personal air monitors, scored 3.9 points lower on the Bayley Mental Developmental Index. These scores are predictive of school readiness, and the authors described their results as modest, yet "worrisome."[45]
- Prenatal exposure to the DDT† breakdown product DDE is also associated with neurodevelopmental delays in children, especially the "psychomotor" skills linking movement or muscular activity with mental processes.[46] And exposure *in utero* to DDT itself has been associated with reduced cognitive functioning, memory and verbal skills among preschoolers.[47]

Strong emerging evidence links childhood pesticide exposure to other, adult-onset neurological effects such as Parkinson's and Alzheimer's diseases; these studies are not examined here.[48]

The combined, society-wide impact of the various syndromes, disorders and deficits resulting from damage to children's brains and nervous systems early in life is immense. Health professionals and educators across the country have indicated concern that our current policies don't adequately protect our children as their nervous systems develop.[49] Something must be done to address this gap, as the results of such exposures have profound consequences for individuals, families and society as a whole.

Exposure of a developing fetus, infant or child to neurotoxic pesticides can lead to greater risk of learning disabilities and significant drops in IQ.

* Piperonyl butoxide, or PBO, is commonly included in formulations of pyrethroid pesticide products to increase the potency of the active ingredient.

† Agricultural uses of DDT were banned in the U.S. in 1972, but because of its persistence, DDT and its breakdown products continue to appear in human blood samples. DDT use continues in some countries for malaria control programs.

2 Cancer, Birth Defects & Early Puberty

Latest science links many childhood health harms to pesticide exposure

If we are going to live so intimately with these chemicals—eating and drinking them, taking them into the very marrow of our bones—we had better know something about their nature and their power. —Rachel Carson

Our children face a range of health challenges that were not encountered by past generations. Public health experts are concerned, and are increasingly focusing on the contributing role of environmental factors such as pesticides and other chemicals.

The President's Cancer Panel's 2010 report, for example, concluded that the role environmental contaminants play in contributing to cancer has been "grossly underestimated" and called for urgent action to reduce the current widespread exposure to carcinogens. The Panel's chair, Dr. LaSalle Leffall, urged preventative measures to protect public health—even in the face of some uncertainty.*

> The increasing number of known or suspected environmental carcinogens compels us to action, even though we may currently lack irrefutable proof of harm.[50]

Meanwhile, evidence continues to mount linking chemical exposure to a range of children's health harms. Below we present a summary of some of the growing body of recent findings on pesticides and childhood cancer, birth defects and early puberty. More detailed descriptions and additional studies are included in Appendix A.

Some childhood cancers linked to pesticides

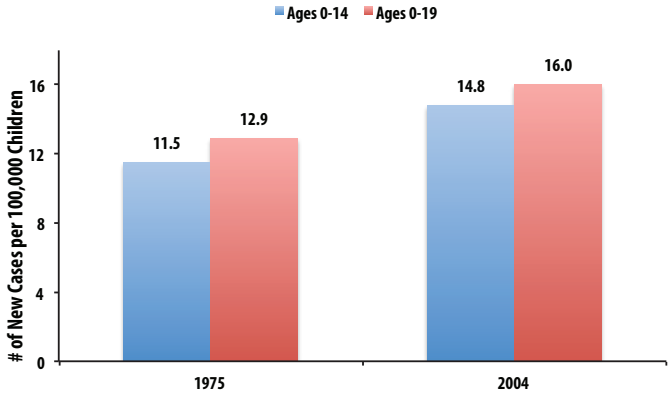
Cancer is the second most common cause of death among U.S. children one to 14 years old.† Over the past 30 years, the number of children diagnosed with all forms of invasive cancer has increased 29 percent, from 11.5 cases to 14.8 cases per 100,000 children per year (see Figure 4).[51]

There are many types of childhood cancer, and incidence rates vary widely. Leukemia and childhood brain cancers are now the most common cancers among children, with rates for these two cancers rising 40 to 50 percent since 1975: leukemia from 3.3 to 4.9 per 100,000 children, and brain cancers from 2.3 to 3.2 (see Table 3).[52]

Survival rates have also risen. Improved cancer treatments have led to dramatic increases in survival of all types of childhood cancer, particularly leukemia (from 50 percent survival in 1975 to more than 80 percent in 2004) and non-Hodgkins lymphoma (from 43 to 87 percent survival over the same time period.) For all types of childhood cancers, African-American children have a lower survival rate than do white children (73 vs. 81 percent).[53]

For some cancers, genetics is a powerful predictor. But as outlined by the President's Cancer Panel, cancers can have multiple and often interacting causes. In some cases genetic factors make an individual more susceptible, and exposure to environmental carcinogens may trigger cancer development.

Figure 4: Incidence of Cancer among Children, 1975 & 2004



Over the past 30 years, the number of children diagnosed with all forms of cancer has increased from 11.5 to 14.8 cases per 100,000 children per year.
Source: SEER, 2004

Table 3: Top 5 Childhood Cancers

- Leukemia
- Brain and other nervous system tumors
- Neuroblastoma
- Wilms' tumor
- Lymphoma

The types of cancers that occur most often in children are different from those seen in adults.
Source: American Cancer Society

The Science

A large number of recent studies link pesticide exposure to childhood leukemia, brain tumors and neuroblastoma. Some evidence suggests pesticide exposure may also be associated with other types of children's cancer, such as non-Hodgkin's lymphoma, Wilms' tumor and Ewing's sarcoma. Many studies

* This call for action in the face of some uncertainty is an example of the "Precautionary Principle," an approach to decision making that has been adopted by many local governments in the U.S. and in countries around the world. For a definition and more information, see the Science and Environmental Health Network's FAQ: http://www.sehn.org/ppfaqs.html

† Lethal accidents are the most common cause of death.

find *in utero* exposure during key windows of fetal development or parental exposure before conception to be particularly important.

- Home insecticide use during pregnancy can increase risk of childhood leukemia, according to a review of 15 studies over the past two decades. Timing of exposure appears to be particularly important.[54]
- The risk of a child developing acute lymphocytic leukemia—the most common type of childhood leukemia—is higher when the mother is exposed to home insecticides during pregnancy. Risk increased with the frequency of the mother's exposure; the highest risk was associated with use of household insecticides more than five times over the course of gestation.[55]
- Mothers who have a particular genetic variant of an enzyme involved with the metabolic processing of wastes and toxins (including carcinogens)* are more likely to have a child with leukemia when they use pesticide products during pregnancy.[56]
- Several case-control studies link exposure to herbicides and household insecticides during pregnancy to an increased risk of childhood brain cancer.[57]
- Higher risk of neuroblastoma, the most common cancer among infants, was observed in children whose parents reported garden and home pesticide use.[58] An older case-control study of U.S. and Canadian children indicated increased risk of neuroblastoma among children whose fathers were landscapers and groundskeepers.[59]
- In a national case-control study in Australia, increased risk of Ewing's sarcoma tumors among children was linked to occupational exposures of mothers and fathers who worked on farms around the time of conception.[60]
- Children who lived in areas of high agricultural activity in the U.S from birth to age 15 experienced significantly increased risk of childhood cancers.[61] And a study in Norway of agricultural census data found that of 323,359 children under 14, those who grew up on a farm—combined with a high level of pesticides purchased by the family—were nearly twice as likely have brain tumors.[62]

A number of studies—not reviewed here—explore potential links between prenatal or childhood pesticide exposures and incidence of cancers later in life. For example, according to the President's Cancer Panel, girls who were exposed to DDT before they reach puberty are five times more likely to develop breast cancer in middle age.[63]

In general, the association between pesticide exposures and childhood cancer outcomes may be underestimated, as data are somewhat limited and studies focus on certain cancers more than others. In addition, common methodological problems—such as occupational exposures being identified only through self-reporting or job title, considerations of other routes of exposure, small sample sizes, and relying on recall to estimate exposures—may contribute to skewed findings.[64]

* The CYP1A1 gene codes for the expression and activity level of an enzyme that helps clear the body of potentially harmful compounds.

Birth defects rise with seasonal or occupational exposures

Birth defects are the leading cause of infant mortality in the U.S., accounting for 19 percent of the 29,138 infant deaths in 2007. And the overall incidence of birth defects is rising.[65] According to CDC data, about one in every 33 babies born today has some kind of birth defect.[66] Birth defects can affect almost any part of the body; some are mild and impact appearance only, others affect the functioning of organs and can be life threatening, although overall survival rates have increased significantly since 1979.[67]

Incidence trends vary by specific birth defect. Cleft lip/palate is the most common birth defect reported, and incidence has declined slightly over the last decade. Rates of Down Syndrome, gastroschisis (an abdominal wall defect resulting in protrusion of the intestines) and anencephaly (absence of portions of the brain, skull and scalp) have all increased since 1999.[68]

Like many children's health outcomes, a combination of genetic and environmental factors is often at play. CDC's research on environmental factors has focused primarily on smoking, alcohol intake, obesity and diabetes.[69] Other scientists, however, have examined the role of parental exposure to pesticides and other chemicals before conception, and of mothers' exposure to environmental contaminants during pregnancy (see sidebar, p. 17).

The Science

Parents exposed to pesticides occupationally, from exposures in their community or by in-home pesticide use may increase the risk of birth defects in their newborn. Studies indicate that exposure of both mothers and fathers, particularly during the period of conception, can influence birth defect outcomes. Several studies in agricultural areas have correlated conception during peak pesticide spray season with increased birth defect risk.

A mother's exposure during pregnancy can also play a key role, with specific timing once again emerging as a critically important variable.

Children whose mothers were exposed to herbicides and household insecticides during pregnancy have an increased risk of developing brain cancer.

Farmworker Families & Pesticides

As a community organizer and health educator in North Carolina, Ana Duncan Pardo works with many communities directly affected by pesticides.

When we spoke with Ana about her experience working with farmworkers, she described a particular instance—when she was setting up for a presentation to farmworker parents—that awoke her to the health harms faced by many of these families:

> Within five minutes I had noted multiple cleft palates and several children with apparent Down Syndrome…. It was shocking and disturbing to walk into a room with a group of parents and children that easily represented three to four times the national average for birth defects.

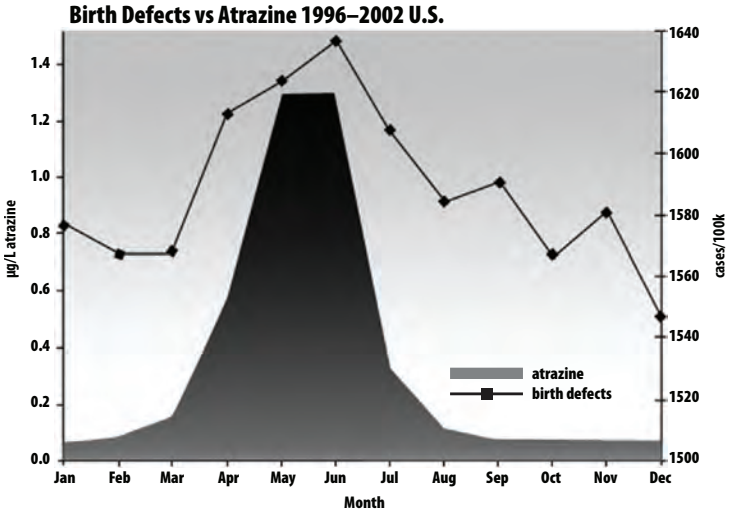
Farmworkers and their families face unique risks, as the harmful chemicals applied in the field follow workers home on their skin, shoes and clothing, and may also drift into their homes from the nearby fields. And, like all families, the food they eat every day may contain pesticide residues.

Ana Duncan Pardo is the farmworker organizer & communications coordinator for Toxic Free North Carolina, and a member of PAN's board.

- A multi-year, national review of USGS water data and CDC birth defect records found a strong seasonal association between birth defects and the presence of the herbicide atrazine in surface water. Infants conceived between April and July, when elevated concentrations of the herbicide are found, have a significantly higher birth defect risk (see Figure 5).[70]
- In Washington state, a seasonal analysis of the risk of the abdominal wall defect gastroschisis showed prevalence peaking when conception occurred between March and May. The birth defect occurred most frequently among infants whose mothers lived within 50 kilometers of a site with high surface water concentration of atrazine.[71]
- Male pesticide applicators in Minnesota had a significantly higher number of children with birth defects, in a study examining 4,935 births to pesticide applicator fathers over three years. The birth defects were more common among boy offspring than girls.[72] Egyptian fathers exposed to pesticides at work also had a greater risk of having children with congenital malformations.[73]
- Increased risk of boys' urogenital malformations such as hypospadia, micropenis and cryptorchidism* has been linked in many studies to prenatal exposure to environmental contaminants. One recent meta-analysis of studies from seven countries (Canada, Denmark, Italy, Netherlands, Norway, Spain and the U.S.) indicated a 36 percent increased risk of hypospadia when mothers were exposed to pesticides at work, and a 19 percent increased risk with fathers' occupational exposure to pesticides.[74]

* Hypospadia is a defect in which the urethral opening develops in the wrong location along the shaft of the penis. Micropenis is a defect where boys have severely reduced penile size, and cryptorchidism is a defect where the testes descend improperly, or not at all.

Figure 5: Atrazine Seasonal Exposure & Birth Defects



Seasonal exposure to pesticides during pregnancy has been linked to increased risk of birth defects. Source: Winchester, P.D., J. Huskins and J. Ying. "Agrichemicals in surface water and birth defects in the United States." Acta Paediatrica. 2009 98: 664–669.

- The risk of having a child with neural tube defects, which are birth defects of the brain and spinal cord, has also been linked to pesticide exposure. Studies indicate a higher risk of this birth defect if insecticide bombs or foggers are used in the home during the period of conception. Risk is also higher if women live within a quarter mile of a cultivated field where pesticides are sprayed.[75]
- Mothers exposed to pesticides at work during a particular period of pregnancy have a significantly greater risk of having a child with anencephaly (a rare defect involving absence of a large part of the brain and skull).[76] A meta-analysis of studies examining fathers' exposure to Agent Orange (containing the herbicides 2,4-D and 2,4,5-T) found the risk of having offspring with spina bifida, a "split spine" defect caused by incomplete formation of the neural tube, was twice as high among those fathers who were exposed.[77] †

Many epidemiological studies over the years have found no association between pesticide exposure and birth defects. It must be considered, however, that these studies may not have taken timing of exposure into account, a variable that is proving to be a critical factor in birth defect outcomes. And as with cancer studies, results may be skewed by use of inappropriate surrogates for pesticide exposure (e.g. job title) or inaccurate subject recall.

Changes in puberty timing linked to low-level exposures

Young girls in the U.S. are moving from childhood to adolescence at an ever-younger age. Changes in the timing of sexual development over the past two decades have been so widespread that the age of "normal" puberty onset has been redefined by health professionals.[78]

† Agent Orange was widely used as a defoliant during the Vietnam War and was often contaminated with dioxins which have also been linked to birth defects. One of the herbicide ingredients, 2,4-D, is still in use in the U.S., and a proposal is currently under consideration for a genetically engineered variety of corn designed to allow increased 2,4-D application.

Dr. Herman-Giddens and her colleagues first documented this acceleration in 1996, in a study finding that the number of girls having some sign of puberty onset before the age of eight was "substantially higher" than previously found.[79]

These initial findings of early puberty were corroborated in 2010 by researchers who found that by age seven, 10 percent of white girls, 23 percent of black non-Hispanic girls, and 15 percent of Hispanic girls had begun the process of breast development, also known as thelarche.[80] Some changes in pubertal development in boys have also been documented.

Changes in puberty timing are concerning for several reasons. For both boys and girls, self-esteem and body image issues can sometimes lead to self-destructive behaviors and poor performance in school. Additionally for girls, both early puberty and obesity (a contributing factor for early puberty) have been linked to health impacts later in life, increasing the risk for breast cancer and later reproductive health issues such as polycystic ovary syndrome.[81, 82]

These changes cannot be fully explained by ethnic, geographic, or socioeconomic factors, and thus a growing body of research has turned to examining the role of endocrine-disrupting chemicals in accelerating puberty in children.[83]

The Science

Although the number of studies is relatively small, researchers have found some associations between pesticide exposure—either during fetal development or early childhood—and effects on puberty.

Most studies focus on *in utero* exposures to pesticides with endocrine-disrupting effects that can interfere with the healthy development of the reproductive system—particularly if exposure occurs at certain times in the process (see sidebar).[84] The majority of studies focus on precocious puberty in girls, but a few studies have also found links between pesticide exposure and changes in the timing of puberty among boys.

Much of the research to date examines impacts of long-lasting organochlorine pesticides. Some of these are chemicals that have already been banned in the U.S. (e.g., DDT, hexachlorobenzene); others are in the process of being phased out (e.g., lindane, endosulfan); but all are still present in our food supply, environment, and in our bodies.[85, *] Though few studies have yet examined the connections, pesticides currently in use are also implicated in some studies.

- Prenatal exposure to the herbicide atrazine was linked to delayed pubertal development in both male and female rats in a recently released animal study.[86]
- Danish greenhouse workers exposed to a range of pesticides during pregnancy were more likely to have daughters showing breast development from 6–11 years old.[87] Increased likelihood of early puberty in girls in Jerusalem was found to coincide with seasons of intensified pesticide usage.[88]
- Daughters in Michigan were more likely to reach puberty at a younger age if their mothers had higher blood levels of the DDT breakdown product, DDE. Participants in this study included women who regularly consumed fish from the Great Lakes, which for years have been heavily contaminated with industrial pollutants such as PCBs and DDT.[89]
- Higher blood levels of hexachlorobenzene and DDE were associated with early puberty among Flemish boys.[90] Two recent studies of boys in India and Russia linked exposure to the pesticide endosulfan and the industrial by-product dioxin to delayed puberty among boys.[91]
- The pyrethroid insecticide esfenvalerate[†] has shown endocrine-disrupting effects related to puberty timing in female rats. Rats exposed to low levels (half of EPA's "no observable effect" level) for seven days showed significant delays in onset of puberty.[92]

As evidence mounts that developmental exposures to pesticides can have an effect on puberty timing, additional studies are now focusing on such endocrine-disrupting effects of pesticides currently in use.

Mechanisms of Harm

Endocrine disruption = development derailed

The term "endocrine" refers to systems in the body that are controlled by hormones, such as brain development, growth, reproduction and puberty. Hormones are chemicals synthesized in the body that bind to receptors to trigger actions at the cellular level resulting in physiological changes. Once their job is done they are released and free to act again.

Some pesticides act as "endocrine disruptors" that mimic hormones and can interfere with systems normally controlled by hormonal action. If such disruption occurs at times during development known as "windows of vulnerability,"—such as when the reproductive system is coalescing, brain or nervous systems are developing, immune system is forming or puberty is getting underway—the process can be derailed in significant ways, sometimes with life-long effects.

Because hormones themselves act at extremely low levels, biological processes controlled by hormones are tremendously sensitive. This means there often is no "threshold" or "safe" dose when it comes to endocrine disrupting compounds.[*]

* Zoeller, R.T., T. R. Brown, L. L. Doan, A. C. Gore, N. E. Skakkebaek, A. M. Sotp et al. "Endocrine-Disrupting Chemicals and Public Health Protection: A Statement of Principles from The Endocrine Society." *Endocrinology* June 2012. See http://endo.endojournals.org/content/early/2012/06/21/en.2012-1422.abstract.

Vandenberg, L., T. Colborn, T. Hayes, J. Heindel, D. Jacobs, D.H. Lee, et al. "Hormones and Endocrine-Disrupting Chemicals: Low-Dose Effects and Nonmonotonic Responses." *Endocrine Reviews.* March 2012 33(3): 378-455.

* CDC sampling from 1999–2000, for example, found DDT's breakdown product in blood samples of 99 percent of U.S. population. See http://www.cdc.gov/exposurereport/.

† Esfenvalerate is listed for Tier 1 screening under EPA's Endocrine Disruptor Screening Program. See http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2009-0634-0001.

3 Emerging Science
Obesity, diabetes & asthma

Chemicals that disrupt hormone messages have the power to rob us of rich possibilities that have been the legacy of our species and, indeed, the essence of our humanity. —Theo Colburn

Many of the health challenges facing children today have strong genetic and/or behavioral components. The rise in childhood obesity, for example, in part reflects the increasingly sedentary habits of many U.S. children.* But it's becoming increasingly clear that personal lifestyle choices do not tell the whole story.

The speed and scope of the society-wide rise in childhood health problems suggest a complex interaction of genetic, behavioral and environmental variables. Researchers are beginning to tease apart these interactions to more fully understand how exposure to environmental contaminants are involved.

We examine here the rapidly emerging science exploring how pesticides may contribute to the recent rise in childhood obesity, diabetes and asthma. Additional studies are included and described in Appendix A.

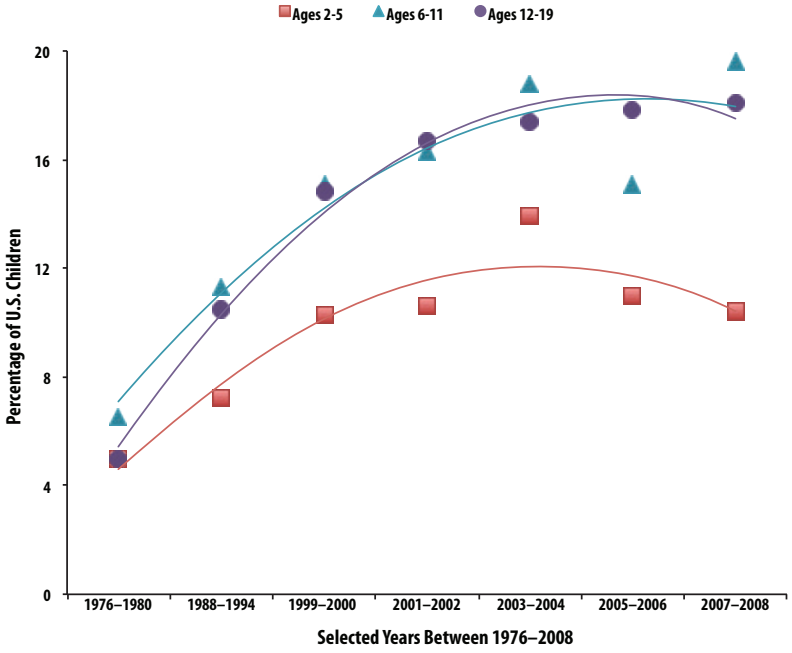
Childhood obesity, diabetes & disrupted metabolism

The recent dramatic rise in childhood obesity in the U.S. has the focused attention of health specialists and the public. The number of clinically obese children has more than tripled in the past 30 years, with obese children ages six to 11 jumping from seven percent of the total in 1980 to nearly 20 percent in 2008. The percentage of obese adolescents (12–19 years old) increased from five to 18 percent over the same period (see Figure 6).[93, †]

Obesity is closely linked to childhood diabetes, which is also on the rise. According to the National Institutes of Health, about 215,000 Americans under the age of 20 had diabetes in 2010—up from roughly 123,000 in 1990.[94, 95]

In addition to increasing related health risks, both obesity and diabetes can have a negative effect on quality of life in terms of ability to engage in physical activities, societal acceptance and self-image.

Figure 6: Prevalence of Obesity among Children Ages 2 to 19 between 1976–2008



Prevalence of obese U.S. children ages 6 -11 jumped from 7 percent in 1980 to 20 percent in 2008, while the percentage of obese adolescents increased from 5 to 18 percent. Source: Center for Disease Control, "Prevalence of Obesity Among Children and Adolescents: United States, Trends 1963-1965 Through 2007-2008."

The Science

So much new science exists around the links between obesity and environmental contaminants that a new term, "obesogen" (like carcinogen) has emerged in the literature.‡ Findings increasingly suggest that exposures to pesticides and other chemicals play a role by altering developmental programming in ways that raise the likelihood of obesity and related metabolic effects such as diabetes.[96]

In 2002, Baillie-Hamilton reviewed data suggesting that the obesity epidemic coincided with the marked increase in usage of industrial chemicals, including pesticides, over the past 40 years (see Figure 7). The author suggested that pesticides and other industrial chemicals potentially cause weight gain by affecting the hormones that control weight, altering sensitivity

* CDC points to estimates that U.S. children spend an average 4.5 hours a day watching television and 7.5 hours using entertainment media (TV, computers, video games, cell phones and movies) as a contributing factor to childhood obesity. See http://www.cdc.gov/obesity/childhood/problem.html

† See CDC's "History of State Obesity Prevalence" showing trends in adult obesity by state from 2000-2010, at the bottom of this page: http://www.cdc.gov/obesity/data/adult.html

‡ See Wendy Holtcamp's review article, "Obesogens: An Environmental Link to Obesity" *(Environmental Health Perspectives,* Feb. 2012) for an overview of the current literature. Available online at http://ehp03.niehs.nih.gov/article/info%3Adoi%2F10.1289%2Fehp.120-a62#r13.

to neurotransmitters, or altering the activity of the sympathetic nervous system.[97]

In the 10 years since this review, many studies have linked exposure to endocrine-disrupting chemicals with increased incidence of obesity and diabetes.[98] The National Institutes of Health is offering grants to study "the role of environmental chemical exposures in the development of obesity, type 2 diabetes and metabolic syndrome,"[99] and the National Children's Study, an ongoing 21-year prospective study of 100,000 U.S. children, is now exploring the hypothesis that prenatal exposures to endocrine disruptors are linked to obesity.[100]

- In one animal study, rats exposed to low-level doses of the organophosphate pesticide chlorpyrifos early in life developed metabolic dysfunction resembling pre-diabetes.[101]
- In Denmark, children exposed prenatally to pesticides through their mothers' work in greenhouses had significantly higher BMI (body mass index) scores than greenhouse worker mothers who were not occupationally exposed, with highly exposed children also having larger skin folds and higher body fat percentages.[102]
- Exposure to the pesticide lindane* during childhood has been linked with increased abdominal fat, increased waist circumference, higher BMI and fat mass percentage in adults.[103]
- Organochlorine pesticide exposure† can be a predictor of developing type 2 diabetes later in life, particularly among obese individuals. Serum concentrations of organochlorines were strongly associated with type 2 diabetes, and the association was stronger among obese persons than non-obese persons.[104]
- Obese children are more likely to have higher concentrations of 2,5-DCP in their urine, a metabolite of the pesticide found in mothballs (p-dichlorobenzene). This correlation was observed in data from the National Health and Nutrition Examination Survey (NHANES).[105]

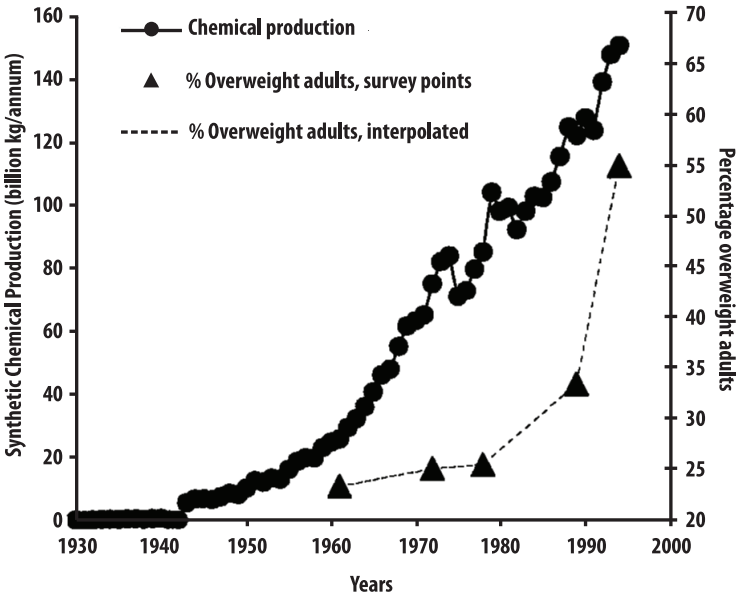
A number of specific genes have been identified as contributing to obesity, with several thought to specifically contribute to obesity in children. Such genes may play a role in regulating metabolic hormones.[106]

Scientists are now investigating the role of environmental factors (such as exposure to pesticides) in influencing the expression of such genes. Such "epigenetic" changes can include the expression of genes that are typically "silent," or inactivation of a gene that is normally active. Researchers are finding that some of these changes can be passed from one generation to the next (see sidebar).[107]

* Lindane, an organochlorine insecticide, is slated for global phaseout under the Stockholm Convention on Persistent Organic Pollutants. Agricultural uses were phased out in the U.S. in 2006; pharmaceutical uses (lice shampoos and scabies treatments) were phased out in California in 2001, but are still allowed in other states.

† Most organochlorine pesticides are now banned in the U.S., and many have been targeted for international phaseout under the Stockholm Convention. Rapid implementation of this treaty will reduce further exposure to these long lasting chemicals that continue to travel the globe on air and water currents.

Figure 7. Chemical Production & the Percentage of Overweight Adults in the U.S.



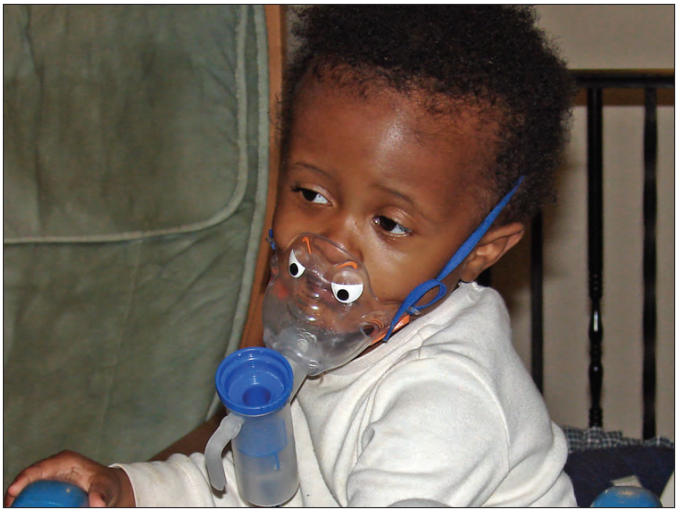
Researchers note that the obesity epidemic coincides with the increase in use of industrial chemicals, including pesticides, over the past 40 years. Source: Baillie-Hamilton, P.F. "Chemical toxins: a hypothesis to explain the global obesity epidemic." *J Altern Complement Med.* 2002 8: 185–192.

Mechanisms of Harm

Changing gene signals

Many environmental pollutants can strip or add chemical tags to DNA, locking the expression of genes on or off and changing how they function. These changes are called "epigenetic tags," and have been linked to various health effects including early puberty, disrupted ovarian function, death of sperm-forming cells and changes in metabolic rate.

Recent studies suggest that some chemicals can even override the genetic "reset button" that usually protects a developing fetus from such changes being passed from one generation to the next.



Today, more than seven million children have asthma, up from just over two million 30 years ago.

Asthma epidemic affects more than seven million children

Asthma is a chronic disease of the pulmonary system that causes wheezing, breathlessness, chest tightness and coughing. The number of U.S. children with asthma today is much higher than it was 30 years ago, rising from 2.1 million in 1980 to 7.1 million in 2009.[108] Today, it is the most common chronic childhood disease in the U.S. (see Figure 8).

Asthma is the leading cause of hospital admission among urban children, with over 200,000 hospitalizations every year. Asthma is also the top cause of days lost from school, with more than 10.1 million school days missed every year.[109] Missed school days in turn negatively impact academic performance, such that children with severe asthma symptoms are more likely to suffer academically than children with milder symptoms.[110]

Asthma disproportionately affects people of color. Data from 2009 show that roughly one in six (17 percent) non-Hispanic black children had asthma in 2009, the highest rate among any racial/ethnic group. Overall, boys are more likely than girls to suffer from asthma (11.3 vs 7.9 percent) from birth through adolescence. As adults, women are more likely to be asthmatic than men.[111,*]

The Science

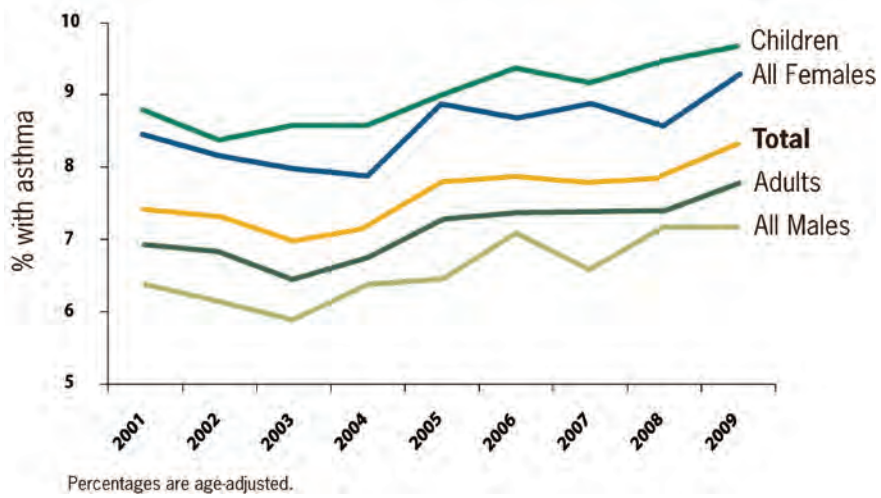
Many studies have explored the relative importance of common "respiratory irritants" in the home environment to triggering the onset of asthma, including cockroaches, dust mites, molds and air pollutants. Many pesticides are considered respiratory irritants,[†] and studies suggest that pesticide exposures may play a role in triggering asthma attacks, exacerbating symptoms, or heightening the overall risk of developing asthma.[112]

Pesticides may also play a role in increasing asthma incidence by affecting the body's immune system, triggering either hypersensitivity or suppression of the body's immune response. Allergic responses, for example, are a hypersensitivity of the immune system to an allergen in the environment.[113]

Numerous studies have documented the association of pesticides and asthma incidence for adults, and more recent studies have examined potential links to both asthma incidence and triggering or exacerbation of wheezing episodes among children.

- In a study of over 4,000 children from 12 southern California communities, exposure to pesticides in the first year of life significantly increased the risk of being diagnosed with asthma by age five.[114]
- A cross-sectional study of 3,291 Lebanese school children found a potential association between childhood asthma and parental occupational exposure to a range of current use pesticides.[115]
- In Spain, children diagnosed with asthma at age six had higher levels of cord serum DDE at birth than children without asthma. And in a study of 343 German children aged 7–10 years who had the DDT breakdown product

Figure 8: Asthma Prevelence by Age and Sex in U.S., 2001–2009



Source Centers for Disease Control and Prevention, Vital Signs: Asthma in the U.S. See http://www.cdc.gov/VitalSigns/Asthma/index.html, viewed May 2012.

* In May 2012, the President's Task Force on Environmental Health and Safety Risks to Children released the *Coordinated Federal Action Plan to Reduce Racial and Ethnic Asthma Disparities.* The effort lays out a plan to address this crucial public health challenge during the next three to five years. See http://www.epa.gov/asthma/childrenstaskforce.

† See the *Recognition and Management of Pesticide Poisonings* page of EPA's National Pesticide Information Center site: http://npic.orst.edu/health/child.html

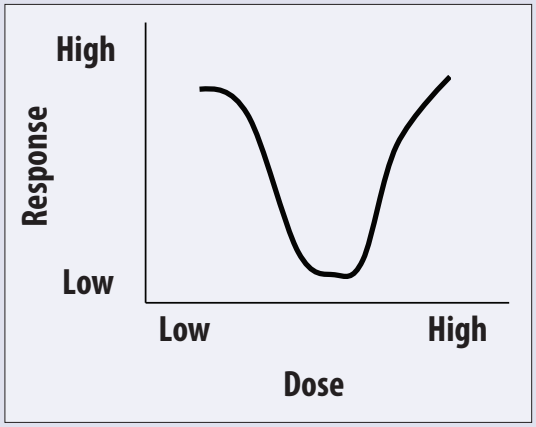
Rethinking "Safe"

Why the dose does not make the poison

Traditional toxicology relied for years on the mantra "the dose makes the poison." We now know that this statement is, in many cases, simply inaccurate. It assumes that the level of harm always increases as the level of exposure goes up (i.e., that every "dose response curve" follows a linear pattern). Assuming a higher dose is always more dangerous, policymakers often base regulations on a level below which no health risks is expected—a "safe" threshold. The reality, as scientists now understand, is quite different.

For some pesticides, the linkage between exposure and effect actually follows a ∪-shaped curve. In this scenario, a very low dose elicits a high level of "response" or health harm. At a higher dose that is along the bottom of the ∪, this same chemical elicits little or no response. Then at the highest doses, the effects increase again. For other pesticides, an inverted ∪–shaped curve can occur, where intermediate doses cause the greatest response, and testing at high doses can completely miss the effect.

Given these complex dose-response patterns, picking a threshold dose—below which exposure can always be considered "safe"—is simply not possible. Throw into the mix the dramatic differences in how sensitive individuals may be to chemical exposures, plus the vulnerabilities of children at particular times during development, and it quickly becomes clear that it is much more than the "dose" that determines how much harm a pesticide will cause.*



* Vandenberg, L., T. Colborn, T. Hayes, J. Heindel, D. Jacobs, D.H. Lee, et al. "Hormones and Endocrine-Disrupting Chemicals: Low-Dose Effects and Nonmonotonic Responses." *Endocrine Reviews*. March 2012 33(3): 378-455.

DDE present in their blood, the risk of having asthma was significantly higher.[116, *]

- Childhood exposure to organophosphate, carbamate and pyrethroid insecticides may trigger or exacerbate asthma symptoms among children by promoting bronchial constriction.[117]

Recognizing the rising prevalence of asthma among U.S. children, Dr. David Schwartz recently called on fellow researchers to focus more attention on the potential links between exposure to air pollutants and environmental contaminants like pesticides and childhood asthma.[118]

* These measurements were taken from blood serum and were thought to represent early life or prenatal exposures, but the actual route of exposure was not known.

4 Critical Junctures

Children exposed just as they are most vulnerable

Children cannot make choices about their environment; it is up to adults to make the right decisions to ensure that they are protected. — Dr. Lynn R. Goldman

Environments we would like to consider "safe" often bring children into contact with pesticides and other chemicals that have been linked to health harms. Many chemicals pass across the placenta into the womb, where they become part of the first environment of a developing fetus. In the months after birth, infants begin to explore their new world, often testing new sights and smells by touching and bringing objects to their mouths. When harmful chemicals are present, they are often taken in.

The environments of toddlers and school-age children expand to include daycare centers, classrooms, playing fields and parks, all of which may offer risk of pesticide exposure. Residues on and in food—from breastmilk to the highchair to the school lunch tray—are also an important source of pesticides throughout childhood.

Many pesticides can pass across the placenta into the womb, where they become part of the first environment of a developing fetus.

Physiological systems undergo rapid development at various stages of childhood, in finely tuned processes often triggered and orchestrated by hormones. During this same period, children take in more food, water and air than adults pound-for-pound, and their biological systems are less able to process harmful contaminants than adults.

In short, the multiple pathways of pesticide exposure mean that in a given day, a child may absorb a wide range of potentially harmful chemicals just as their young bodies are at their most vulnerable.

Fetal pesticide exposures can have life-long effects

Exposure to pesticides has been clearly documented during one of a human organism's most vulnerable stages: fetal development.

Pesticides that have accumulated for years in an expectant mother's body—stored in blood and fatty tissues—can be mobilized during pregnancy and cross the placental barrier. A mother's exposures to pesticides during pregnancy add to this chemical mixture in the womb.[119]

Many studies have documented the pesticide load newborns bring with them into the world. Researchers in New York documented pesticides and their breakdown products in umbilical cord blood of more than 80 percent of newborn infants tested.[120] One 2001 study found metabolites of organophosphate pesticides in 100 percent of the cord blood samples taken.[121] A pilot study of amniotic fluid also found organophosphate metabolites, providing further evidence of fetal exposure.[122]

Pesticide residues from the food mothers eat during pregnancy have also been found in infants. A recent Canadian study showed that when pregnant women consumed soybeans, corn and potatoes that had been genetically modified for use with particular herbicides, metabolites of one of the herbicides showed up in cord blood of 100 percent of their babies.[123] *

Fetal development is almost entirely controlled by the expectant mother's hormones, acting at very low levels to trigger and control growth of the various systems of the body. Some chemicals—including many pesticides—mimic hormones and so interfere with natural developmental processes. This disruption of hormone function can lead to irreversible life-long effects including birth defects or learning disabilities in childhood, or adult onset cancer or infertility later in life (see sidebar, p. 17).[124]

Pesticide exposures common at home, daycare & school

Pesticides tend to be especially persistent in the indoor environment where sunlight, rain, soil microorganisms and high temperatures cannot degrade them, which means longer windows of exposure.

At home & in daycare facilities

Infants and toddlers have busy hands that often reach their mouths, and they commonly play on or near the floor—so

* The women in the study were in urban environments, and had no contact with the herbicides beyond residues on or in their food.

Children as Farmworkers

Some children are exposed to pesticides as they work in agricultural fields. Specific rules vary from state to state, but federal law allows children under 12 to do field work outside of school hours on farms where their parents are employed.*

Age restrictions for hazardous work such as applying pesticides are more lenient in the agriculture sector, and age restrictions simply do not apply for children working on farms owned or operated by a parent or guardian.

Documenting the exact number of child workers in U.S. agriculture is difficult, and estimates vary widely. A Human Rights Watch report published in 2000 put the number somewhere between 300,000 and 800,000.† The nonprofit group Toxic Free North Carolina recently documented the experience and voices of young farmworkers facing pesticide exposure in the field; the stories can be viewed at www.panna.org/youngfarmworkers.

* U.S. Dept. of Labor. "Child Labor Requirements in Agricultural Occupations Under the Fair Labor Standards Act." June 2007. See http://www.dol.gov/whd/regs/compliance/childlabor102.htm.

† Human Rights Watch. *Fingers to the Bone: United States Failure to Protect Child Farmworkers.* Washington: Human Rights Watch, 2000.

National Center for Farmworker Health. *Child Labor.* Buda, Texas. 2009. See www.ncfh.org/docs/fs-Child%20Labor.pdf

Davis, S. and J.B. Leonard, *The Ones the Law Forgot: Children Working in Agriculture,* Farmworker Justice, Washington DC. 2000.

when pesticides are used in homes or daycare facilities, exposure is a near certainty. Inhaling spray droplets, vapors or pesticide-contaminated dust from indoor use of pesticide products is one of the primary routes of exposure for many U.S. children. Pesticides used to control ticks and fleas on pets are another important source of children's exposure.[125]

One Massachusetts study found residues of DDT in house dust many decades after use of the chemical had been discontinued.[126] Even pesticides that are relatively short-lived in the environment are more persistent indoors; one study found the semi-volatile insecticide chlorpyrifos to be longer lasting than expected in closed apartments, detectable for more than two weeks on rugs, furniture, soft toys and pillows.[127] Pesticide vapors often settle after application indoors, so levels tend to be highest in the infant breathing zone.[128]

Exposure from home lawns and gardens or outdoor play areas at daycare centers can also be significant. Children often roll and play on lawns and sit or lie on bare soil, and toddlers are known to put dirt directly into their mouths.[129] If pesticides have been used in these areas, the likelihood of ingestion or inhalation is high.

In rural communities, the risk may be compounded by drift from nearby agricultural fields. A study conducted in Washington State found residues of several agricultural pesticides—including chlorpyrifos and ethyl parathion—in outdoor play areas.[130] Air monitoring studies using PAN's Drift Catcher in

Evidence shows that when pesticides are used at home, on pets or in daycare centers, children's exposure is a near certainty.

California and Minnesota have documented a range of agricultural pesticides in backyards and play areas as well.[131, 132]

Rural infants and toddlers also face potential exposure from drift directly into their homes, and from pesticide contamination of water supplies. Water sampling results from Illinois, Nebraska, Iowa and Minnesota detected the common herbicide atrazine at levels above those linked to low birth weight.[133] Young children in farmworker families face additional exposure from residues carried into the home on the bodies and work clothes of working family members.[134]

At school & on playgrounds

Pesticides used in school buildings can settle on desks, books, counters and walls. When children touch contaminated surfaces, they may absorb chemical residues that can remain in the school environment for days. Herbicides used to keep playing fields free of weeds may be picked up on children's hands, bodies, clothes and tennis shoes, or drift into classrooms after application.

According to one recent national review, of the 40 pesticides most commonly used in schools, 28 are probable or possible carcinogens, 26 have been shown to cause reproductive effects, 26 damage the nervous system, and 13 have been linked to birth defects.[135]

In rural areas, pesticides often drift into schoolyards during and after spraying on nearby fields. Community air monitoring studies across the country using the Drift Catcher device have documented pesticides in or near school grounds in agricultural communities,[136] and incidents of pesticide poisonings in schools are not uncommon. For example:

- In Florida, high school students used a Drift Catcher to measure the pesticides endosulfan, diazinon and trifluralin* drifting into the school from nearby cabbage fields.[137]

* Endosulfan is currently being phased out in the U.S., and also globally under the Stockholm Convention on Persistent Organic Pollutants. See http://www.epa.gov/oppsrrd1/reregistration/endosulfan/endosulfan-cancl-fs.html.

- Schoolchildren in Strathmore, CA were exposed to pesticides sprayed in a neighboring field, feeling dizzy and falling sick in November, 2007.[138]
- Seven children were hospitalized and a total of 11 people sickened in Kahuku, Hawaii, in 2007, when fumes from an organophosphate insecticide drifted over the school from a nearby sod farm.[139]

Pesticide use on playing fields has raised concerns among families and environmental health advocates nationwide. The National Coalition for Pesticide-Free Lawns notes that "the common, everyday practices used to maintain our children's playing fields are unintentionally and unnecessarily exposing them to carcinogens, asthmagens, and developmental toxins," and calls for a shift to organic turf management on playing fields across the country.[140]

Pesticide residues, from breastmilk to the school lunch tray

Pesticide residues in food and drink are a key source of constant, low-level exposure to mixtures of pesticides throughout childhood.

Nature's Finest, Compromised
Pesticides in breastmilk

Human breastmilk is without doubt the best source of nutrition for infants, offering the perfect combination of fats, carbohydrates and proteins for developing babies. It also offers protection from infection, increases resistance to chronic disease and contributes to the emotional wellbeing of both infant and mother.

But decades of breastmilk sampling also leaves no doubt that around the world, nature's perfect food for infants is compromised by pesticides and other toxic chemicals. Today there is no corner of the planet where human breastmilk remains pure. The chemicals found in a mother's milk represent a combination of long-lasting pesticides and industrial pollutants that have accumulated over a lifetime (many of which the body tends to store in fatty tissues), and shorter-lived chemicals that a woman is exposed to during pregnancy and breastfeeding.

This chemical burden is transferred to nursing infants just as their bodies are most vulnerable to chemical harms. The good news is that analysis of decades of banked breastmilk in Sweden shows that bans on specific chemicals can result in rapid and dramatic decreases in the levels of some of those compounds in human milk.*

* Norén K., D. Meironyté. "Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20-30 years." *Chemosphere.* May-Jun 2000;40(9-11):1111-23. See http://www.ncbi.nlm.nih.gov/pubmed/10739053.

Natural Resources Defense Council. "Healthy Milk, Healthy Baby: Chemical Pollution and Mother's Milk." See www.nrdc.org/breastmilk.

Children take in more food, water and air than adults pound-for-pound, just as their bodies are less able to process harmful contaminants.

Studies from around the world have documented pesticides in human breastmilk, though experts agree it remains the best source of nutrition for infants (see sidebar). Baby foods and fruit juices consumed by infants and toddlers tend to be highly processed, which can sometimes concentrate pesticide residues existing on the fresh produce.[141] U.S. researchers measuring pesticides in baby foods found low-level residues of many pesticides, including eight known to be toxic to the nervous system, five that disrupt hormones and eight that are potential carcinogens.[142]

Food consumed by school-age children can also contain pesticide residues. Researchers examining the diets of urban children found that 14 percent of the foods sampled contained at least one organophosphate pesticide. In total, 11 different organophosphates and three pyrethroids were found.[143] USDA residue sampling of produce commonly eaten by children—such as carrots, apples and peaches—found metabolites of dozens of different pesticides in each of these foods over the course of their testing (26 found in carrots, 42 in apples and 62 in peaches).*

Pesticides directly measured in children's bodies also tell a story about the importance of dietary exposure. Researchers compared levels of organophosphate metabolites in the urine of children who were eating organic fruit, vegetables and juice with children eating conventionally farmed produce. They found that those with more organic diets had metabolite levels six times lower than those with conventional diets.[144] Other studies show that when families switched to organic fruits and vegetables, metabolites of the insecticides chlorpyrifos and malathion fell quickly to undetectable levels.[145]

The widespread presence of pesticide metabolites in children's bodies,[146] combined with studies showing that changes in these levels are linked to changes in dietary exposure, make a very clear case that pesticide residues in food are a consistent source of children's daily intake of a mixture of pesticides.

* These numbers do not necessarily reflect residues on a single sample. See USDA data at www.whatsonmyfood.org.

Why children are particularly vulnerable

So what do all of these well-documented pesticide exposure pathways mean for children's health?

In their first six months of life, children take in roughly 15 times more water than the average adult per pound of body weight.[147] Children also inhale more air. Up to around age 12, a child's breathing rate is roughly twice that of an adult, which means a child will inhale roughly double the dose of a pesticide in the air from spray drift or household use.[148]

Exposure to pesticides occurs largely through touching, inhaling or ingesting. For each of these routes, children are much more likely to absorb what they come into contact with than adults. The skin of infants and young children, for example, is particularly permeable, and the skin surface area relative to body weight is much greater in children than adults.[149] The lung surface area relative to rate of breathing is also higher among children,[150] and absorption levels in the gastrointestinal tract are also greater (especially for alkaline pesticides), as adult levels of gastric acid are not reached until a child is about two years old.[151]

As noted above, the brain and nervous system are especially vulnerable during fetal development and for the first six months of life. During this period the blood-brain barrier,* which provides the adult nervous system some protection from toxic substances, is not yet fully developed.[152]

Finally, young bodies are less equipped to process and excrete harmful chemicals as the liver and kidneys—the body's primary detoxifying organs—are not yet fully developed. Levels of enzymes that help the body process chemicals are also not yet at full strength (see sidebar). Genetic variations lead to tremendous range in the production of these protective enzymes—with some newborns as much as 164 times more vulnerable to chlorpyrifos than less sensitive adults.[153]

According to researchers, this finding alone means that most, if not all infants and toddlers—as well as a subpopulation of adults—are much more likely to have adverse health effects from organophosphate exposure. Policies that don't account for this variability fail to protect the most vulnerable, leaving many children in harm's way.

Mechanisms of Harm

When enzymes don't detoxify

Enzymes are proteins that catalyze reactions on a molecular level, and there are many that occur naturally in the human body. Without enzymes to catalyze reactions, some of the chemical reactions that make up the normal functioning of our body could take much longer, or not happen at all.

One key human enzyme, known as paraoxonase 1 (or "PON1"), catalyzes the metabolic process that renders organophosphate pesticides and other compounds less harmful to our systems. Researchers say infants have very low levels of this enzyme up to age two, and children don't reach adult PON1 levels until about age seven.* This suggests that children are less protected from harmful contaminants by enzyme activity, and newborns may be especially vulnerable.

There is also tremendous natural variability in the level and effectiveness of the PON1 enzyme, which means some individuals are much more susceptible to health harms of organophosphate pesticides and other contaminants.†

* Huen K., K. Harley, A. Bradman, B. Eskenazi, N. Holland. "Longitudinal changes in PON1 enzymatic activities in Mexican-American mothers and children with different genotypes and haplotypes." *Toxicol Appl Pharmacol.* 2010. 244(2):181-9. See http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2846980/?tool=pubmed

† Holland, N., C. Furlong, M. Bastaki, R. Richter, A. Bradman, K. Huen, et al. "Paraoxonase Polymorphisms, Haplotypes, and Enzyme Activity in Latino Mothers and Newborns." *Environ Health Persp.* July 2006 114 (7): 985–991. See http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1513322/.

The human body undergoes rapid growth and development throughout childhood, with many processes vulnerable to disruption from pesticides and other chemicals.

* The blood-brain barrier is made up of high-density cells that protect the brain from potentially harmful substances circulating in the bloodstream.

5 Case Studies
Communities win protections for children

What we love we must protect. — Sandra Steingraber

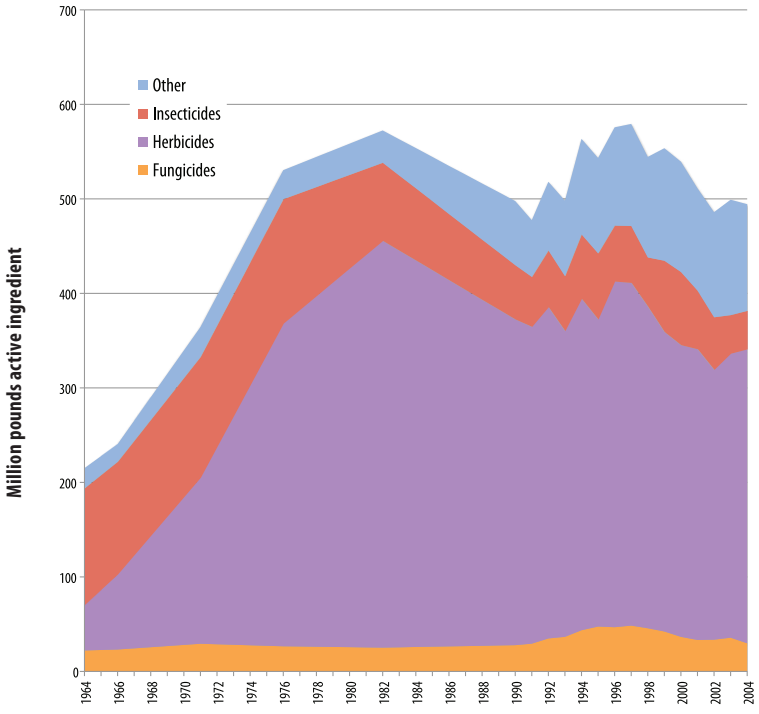
Since the middle of the last century, the overall increase in pesticide use in this country has been steady and dramatic. As documented above, these pesticides are a critical contributor to many of the chronic diseases and disorders now affecting our children.

To address the unique vulnerability of children, concerned communities, public health officials and advocates are beginning to put policies in place at the state and local level that reduce the use of harmful pesticides. In this chapter we provide a brief overview of U.S. pesticide use patterns and trends, and highlight on-the-ground stories of successful efforts to protect children from exposure in their early environments.

Pesticide use now 1.1 billion pounds yearly

Since 1945, use of herbicides, insecticides and other pesticides has grown from less than 200 million to more than 1.1 billion pounds per year, with well over 1,000 chemicals registered and formulated into more than 20,000 pesticide products (see Figure 9). This does not include pesticides used as wood preservatives or specialty biocides (in plastics and paints, for example). If these products are included, the number jumps to more than five billion pounds annually.[154, 155, 156]

Figure 9: Pesticide Use on Major Crops, 1964–2004



Source: "Land and Farm Resources: AREI, 2006 Edition," USDA Economic Research Service

Pesticide use in agriculture

The majority of pesticides are used in agricultural fields, with weed-killing herbicides being the highest by volume. Soil fumigants, which are injected as a gas into soil before planting to kill weeds, insects and fungi, are used at particularly high volumes and have a tendency to drift after application. Use of organophosphate insecticides, which gained widespread use in the 1980s as replacement chemicals for long-lasting organochlorine pesticides (such as DDT, chlordane and aldrin) has gradually declined in recent years.

In part to address growing concerns about organophosphate toxicity, a group of insecticides called pyrethroids were marketed as "safer" and gained widespread use in the 1990s, and use has grown rapidly. According to the American Chemical Society, use of pyrethroids in California (agricultural, structural and landscape maintenance applications) almost tripled from 1992 to 2006.[157] Recent research suggests that pyrethroids may be more harmful to humans than originally believed, acting as developmental neurotoxicants, endocrine disruptors and carcinogens.[158, *]

Another class of pesticides now in widespread and rapidly rising use is neonicotinoids. Most neonicotinoids show much lower toxicity in mammals than insects, but emerging science demonstrates that many may also have neurodevelopmental effects, and some are considered likely carcinogens by EPA.[159] These pesticides are considered 'systemic,' which means they are often applied at the root (as seed coating or drench) and are then taken up through the plant's vascular system. Systemic pesticides on food cannot be washed off.

Neonicotinoid pesticides have been linked with honey bee colony collapse disorder and bee kills, and several products have been banned in European countries for this reason. One neonicotinoid, imidacloprid, is now one of the most widely used insecticides in the world.[160]

* Ten years' worth of adverse-reaction reports (filed by manufacturers) show that pyrethrins and pyrethroids together accounted for more than 26 percent of all fatal, "major," and "moderate" human pesticide poisoning incidents in the U.S. in 2007, up from 15 percent in 1998. See http://apps.cdpr.ca.gov/calpiq/calpiq_input.cfm to see the primary data; for data analysis, see http://www.iwatchnews.org/environment/health-and-safety/perils-new-pesticides.

Pesticide use at home

While 80 percent of all pesticides are applied in agricultural fields, use in homes, gardens, playgrounds, schools, hospitals and other buildings is also significant—and as noted above, such uses pose a particular risk to children's health.

In 2007, an estimated 78 million pounds of pesticides (measured by active ingredient) were applied in homes and gardens across the country, with the herbicides 2,4-D and glyphosate (RoundUp) topping the list.[161] The household pesticide product industry has an estimated annual net worth of $1.4 billion; according to EPA, more than 78 million households—roughly 74 percent of all households in the U.S.—report using pesticides at home (see Table 5).[162]

Many home-use insecticides contain pyrethroids, and the chemicals are used extensively in homes where the potential for exposure to children is very high. Researchers from Emory University and the CDC found that even children fed an exclusively organic diet had pyrethroid metabolites in their systems after their parents had used pyrethroid insecticides in their homes.[163]

Neonicotinoid products are widely used in pet products to control fleas and ticks—another use which poses particularly high exposure risks for children.[164]

Safer pest control at schools & daycare centers

More than 3,000 pesticide products are currently approved for use in schools;[165] yet current national pesticide rules do not address the use of pesticides in and around schools or daycare centers. The federal School Environmental Protection Act (SEPA) was first introduced in November 1999 in an attempt to address this oversight—and it continues to be debated in Congress today.

In the non-profit sector, the national Children's Environmental Health Network (CEHN) moved to fill this gap by creating the Eco-Healthy Child Care (EHCC) program to provide tools that facilities need to create environmentally healthy spaces for children. Today, the program endorses over 1600 "Eco-Healthy" daycare facilities across the country and provides this list to parents online.*

To protect children's health, several states have put policies in place prohibiting the use of pesticides on playing fields and playgrounds.

Meanwhile, several states are moving forward with policies designed to protect children from pesticides in these early environments.

- In 2005 Connecticut lawmakers prohibited use of pesticides on K–8 lawns and playing fields; in 2009, the law was extended to daycare center grounds. Through this policy, schools have successfully implemented organic turf programs in various municipalities.[166]
- New York followed suit in 2010, signing the Child Safe Playing Fields Act into law to ban the cosmetic use of pesticides on playgrounds and sports fields at schools (including high schools) and daycare centers.[167]

Table 4: Pesticide Usage in All Market Sectors, 2007

Pesticide Class	Active Ingredient
Herbicides	531 million lbs
Insecticides	93 million lbs
Fungicides	70 million lbs
Fumigants/Nematicides	133 million lbs
Other	30 million lbs
Total	*857 million lbs*

Herbicides are the most commonly used type of pesticide in the U.S., with 531 million pounds of active ingredient applied in 2007. Source: *Pesticide Industry Sales & Usage, 2006 and 2007 Market Estimates*, U.S. EPA, Washington, DC Feb 2011. See www.epa.gov/opp00001/pestsales/07pestsales/market_estimates2007.pdf.

Table 5: Households Using Pesticides

Pesticide Type	# Households
Insecticides	59 million
Fungicides	14 million
Herbicides	41 million
Repellents	53 million
Disinfectants	59 million
Any pesticides	78 million

According to EPA, more than 78 million households—roughly 74 percent of all households in the U.S.—use pesticides at home. Source: EPA estimates based on the 1992 EPA National Home and Garden Survey and 2000 U.S. Census Bureau population estimates (www.quickfacts.census.gov/qfd/states).

* See http://www.cehn.org/ehcc for more information about this program.

At What Cost?

Economic impacts of health harms

The impact on families of caring for—and sometimes losing—a child in ill health cannot be reflected in monetary terms. Nor can the incalculable costs of lowered IQ, lost opportunities and social alienation that can accompany developmental effects. But actual costs of providing medical care for a child with a chronic condition or illness can be calculated, and according to public health officials, health care costs for childhood diseases are significant. Here are some examples:

ADHD: Researchers estimate annual ADHD health care costs in the U.S. to be between $36 and $52 billion (in 2005 dollars).*

Autism: One analyst at the Harvard School of Public Health estimates that it costs $3.2 million to care for an autistic person over their lifetime.†

Cancer: The total costs per case of childhood cancer—from treatment, to laboratory costs to lost parental wages—is an estimated $623,000 per year.‡ This translates into a society-wide cost of roughly $6.5 billion annually for the 10,400 newly diagnosed cases each year.

Asthma: Families nationwide pay a combined total of $14.7 billion dollars a year on medical care costs of asthma.§, ¶ The combined direct and indirect costs of asthma to the U.S. economy were an estimated $19.7 billion in 2007.**

Society-wide costs also include higher educational costs for public school systems to meet the needs of children with neurodevelopmental disorders, missed school days (and thus less well-educated students) caused by asthma, and the general productivity losses due to time parents and caregivers take off from work to care for an ill child.

The numbers above do not take into consideration the loss to individuals, families and society as a whole of children not reaching their full physical or intellectual potential. The overall impact of lost creativity, productivity, problem-solving skills and civic engagement, along with higher rates of social alienation and disruption, cannot be overstated.

* Pelham W., E.M. Foster and J.A Robb. "The Economic Impact of Attention Deficit/Hyperactivity Disorder in Children and Adolescents" *Journal of Pediatric Psychology.* 2007. See http://jpepsy.oxfordjournals.org/content/32/6/711.full.pdf+html.

Centers for Disease Control and Prevention. Attention-Deficity/Hyperactivity Disorder (ADHD): Data and Statistics in the United States. See http://www.cdc.gov/ncbddd/adhd/data.html.

† Ganz, Michael "The Costs of Autism," in *Understanding Autism: From Basic Neuroscience to Treatment* (CRC Press, 2006). See http://www.hsph.harvard.edu/news/press-releases/2006-releases/press04252006.html

‡ Landrigan, P. J., C.B. Schechter, J.M. Lipton, M.C. Fahs and J. Schwartz. "Environmental pollutants and disease in American children: estimates of morbidity, mortality, and costs for lead poisoning, asthma, cancer, and developmental disabilities." *Environ. Health Perspect.* 2002; 110, 721–728.

§ EPA, Children's Heath Protection. "Fast Facts on Children's Health." See http://yosemite.epa.gov/ochp/ochpweb.nsf/content/fastfacts.htm. Viewed June 2012.

¶ Centers for Disease Control and Prevention. *Vital Signs: Asthma in the U.S.* See http://www.cdc.gov/VitalSigns/Asthma/index.html. Viewed May 2012.

** EPA, Children's Heath Protection. "Fast Facts on Children's Health." See http://yosemite.epa.gov/ochp/ochpweb.nsf/content/fastfacts.htm. Viewed June 2012.

- Many school districts in California have significantly reduced pesticide use after a 2000 state law required pesticide reporting and provided incentives for adoption of IPM. School districts in Los Angeles, San Francisco, Santa Barbara and Palo Alto have made particular progress.[168]
- In 2001, California legislators passed a law (AB 947) allowing county agricultural commissioners to restrict pesticide spraying near sensitive sites, including schools and daycare facilities. Under this provision, communities in Tulare County won new rules in 2008 requiring a quarter mile buffer zone banning the aerial application of restricted-use pesticides around schools when they are in session or due to be in session within 24 hours, occupied farm labor camps and residential areas.[169] Kern, Stanislaus, Merced and Fresno counties enacted similar rules in subsequent years.

Pesticide-free school lunches

Currently, neither state nor national policies are in place to reduce pesticide residues in school lunches. But many communities across the country are leading the way to provide children with nutritious school lunches including fresh (sometimes locally produced) fruits and vegetables free from pesticides.

- In Washington state, the Olympia School District has implemented an Organic Choices Salad Bar (25 percent of the produce is purchased directly from local farms and 50 percent of the salad bar is organic), and the Orcas Island Farm-to-Cafeteria Program integrates produce from local, organic farmers and a school garden, and hosts student chef competitions.
- In Minnesota, the White Earth Land Recovery Project added a farm-to-school component in the 2007–2008 school year to their Mino-miijim (Good Food) Program to help reach their goal of food sovereignty on the reservation and promote access to fresh, local and organic ingredients.[170]
- Berkeley, California's Edible Schoolyard (ESY) Project began as a one-acre "interactive classroom" providing primarily organic, fresh fruits and vegetables for student's meals at King Middle School. It has grown into an online initiative building and sharing a food curriculum, and it has inspired similar programs across the country.[171]

Many of these programs are part of the National Farm to School Network (NFSN), which connects K–12 schools across the country with local farms in an attempt to serve healthy meals at school lunch tables while supporting local, often organic, farmers.[172]

Parks & playgrounds without pesticides

Communities across the country are choosing to manage public parks and playgrounds without harmful pesticides. In the Pacific Northwest, 17 cities are phasing out pesticide use with the creation of 85 pesticide-free parks and playgrounds, building momentum for strong policies at the local level despite legislative hurdles (see sidebar on following page).[173]

Farm-to-school programs across the country are providing children with fresh, pesticide-free fruits and vegetables in school cafeterias.

Seattle in particular has emerged as a pioneer of pesticide-free cities, dramatically reducing its pesticide use in parks by an estimated 80 percent since the 1970s. In 1999, they adopted a pesticide reduction strategy for all city departments and designated 14 pesticide-free parks.[174] The program is now expanding to 22 parks and 50 acres distributed throughout the city.[175]

On the other side of the country, New Jersey legislators unanimously voted in 2011 to pass "The Child Safe Playing Field Act" prohibiting pesticide use on all municipal, county and state playgrounds and playing fields, as well as daycare and school grounds.[176]

Many other communities across the country are following this trend. From a pilot program in Lawrence, Kansas to innovative communities throughout Oregon, California and Colorado, cities are creating pesticide-free parks and playgrounds for children to safely enjoy.

The Pre-emption Law Hurdle & Canada's Local Pesticide Bans

As of 2010, 40 states had pre-emption laws specifically prohibiting municipalities from passing local pesticide ordinances that are stricter than state policy. These laws, which are strongly supported by the pesticide industry, limit the ability of city or county governments to ban or restrict pesticide use.

Such pre-emption laws do not exist in Canada. Over the past 20 years, dozens of Canadian cities have used their local authority to outlaw the application of home and garden pesticides for "cosmetic" purposes such as lawn care.

In 1991, the municipal council of Hudson, Canada, enacted the first ban on cosmetic uses. Similar local bans were adopted across the country, and today more than 170 Canadian cities and towns have passed full or partial bans on pesticide use, and the provinces of Quebec, Nova Scotia and Ontario have enacted comprehensive cosmetic pesticide bans. According to Canadian community activists, more than 22 million Canadians (65% of the population) are now protected from exposure to cosmetic pesticides.*

* Pesticide Free B.C. "Pesticide Bylaw Communities Across Canada." See http://www.pesticidefreebc.org/index.php?option=com_content&view=category&layout=blog&id=53&Itemid=72. Viewed July 2012.

6 Investing in a Healthier Future

A solid start for our children must be a national priority

Those who argue that societies cannot afford to make immediate investments in reducing environmental pollution fail to appreciate that there are some forms of harm that cannot be repaired. — Deborah Axelrod, Devra Lee Davis & Lovell A. Jones

As a nation, we value the wellbeing of our children. In addition to our natural urge to protect what we love, we know that at a societal level their success is key to a vibrant, secure future. Poll after poll shows more than 80 percent of Americans consider healthy children a top priority.[177] We must line up our practice and policies with these values.

Our current use of over a billion pounds of pesticides every year puts their wellbeing at risk and, as the science demonstrates, can derail brain and body development and rob them of their full potential.

If there were no other way to control pests, it would be one kind of choice: weighing one set of needed benefits against known and evolving harms. But given the fact that there are many proven ways to control pests without use of harmful chemicals, the choice is quite clear. It is time to have policies in place that better protect our children (see sidebar).

The National Research Council recommended swift action to protect children from pesticides nearly 20 years ago, and it has been 50 years since Rachel Carson sounded the initial alarm about the health harms pesticides can cause. What is standing in the way?

Pesticide industry well served by current policies

Our current system of industrial agriculture and pest control relies on chemical inputs sold by a handful of corporations. These multinational corporations wield tremendous control over the system, from setting research agendas[178] to financing, crop selection and inputs throughout the production and distribution chain.

Not surprisingly, these same corporations also hold significant sway in the policy arena, investing millions of dollars every year to influence voters, lawmakers and regulators at both the state and federal level to protect the market for pesticides.[179]

The result is agriculture, food and pest control systems that serve the interests of these corporations well. It does not, however, serve farmers, who have lost day-to-day control of their operations and are putting themselves and their families in harm's way. Farmworker interests are not served, as workers are continuously exposed to chemicals known to harm human health.

And the health of children across the country is compromised by exposure to pesticides used to control pests in agriculture and where they live, learn and play.

In short, the system is broken.

Prioritizing children's health requires real change

The best way to protect children from the harms of pesticides is to dramatically reduce the volume used nationwide. This would not only limit children's exposure during their most vulnerable years, it would also lower pesticide levels in the bodies of men and women of childbearing age—protecting current and future generations in one fell swoop. Those pesticides most harmful to children should be first on the list.

This is not a small change, and not a recommendation made lightly. Yet the science tells us the problem is serious and urgent, and that viable and safer alternatives are available. If we stay on our current path, our children will not reach their full potential as we continue to compromise their health.

U.S. Pesticide Rules

Overdue for overhaul?

A little over 100 years ago, Congress enacted our first national pesticide law. The 1910 Insecticide Act put labeling guidelines in place to protect farmers from "hucksters" selling ineffective, misbranded or adulterated pesticide products.

To this day, we control pesticides through a system of registration and labeling. The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), passed by Congress in 1947, is our primary national pesticide law. It has been updated several times in the last 65 years as the health and environmental effects of pesticides came into light, most significantly in 1972 and again in 1996.

It remains, however, a system of registration and labeling, and as such has significant shortcomings. Our current pesticide rules:

- Do not allow for quick response to emerging science;
- Do not assess risk based on real-world exposures;
- Rely heavily on corporate safety data that is not peer-reviewed; and
- Do not encourage the safest form of pest control.

In addition, enforcement of any guidelines or restrictions specified on product labels is relegated to state governments that rarely have adequate resources for the job. Overall, our current rules do not provide adequate tools to protect children from the harms of pesticide exposure.

Informed household food choices can help protect families and grow the market for food that is produced without harmful pesticides—encouraging more farmers to make this shift. And reducing household use of pesticides can provide immediate and long lasting benefits to children's health.* But the burden of protecting children from dangerous chemicals cannot rest solely with individual families. Policy change is required.

Recommendations: Effective policies urgently needed

To protect our children from the health harms of pesticides, policymakers must have much more effective tools. We believe such tools are most urgently needed as decisions are made about these three questions:

- Which pesticides are used in agriculture?
- Which pesticides are used in places children live, learn and play?
- How are farmers supported as they reduce reliance on pesticides?

We recommend the following policy changes in these three arenas:

1. Prevent the pesticide industry from selling agricultural products that can harm children's health

Given the wide-ranging susceptibility of children to pesticide exposures, plus the potential impacts on children from extremely low doses of toxic chemicals, the current approach to assessing and controlling risks of agricultural pesticides does not adequately protect our children.

Decisionmakers must have tools to remove an agricultural pesticide from the market quickly or deny a newly proposed pesticide market access when science suggests it can harm children's developing minds or bodies and there is evidence that children are likely to be exposed. Specifically, we recommend that rulemakers should:

- *Take swift action on existing pesticides:* If studies find a pesticide to be a neurodevelopmental or reproductive toxicant, endocrine disruptor or human carcinogen—*and* it has been measured in humans, in schools or homes, or as residues on food or in drinking water—EPA should target the pesticide for rapid phaseout, triggering USDA resources to assist rapid farmer transitions to safer pest control methods.†
- *Block harmful new pesticides:* EPA should not approve any new pesticide that scientific studies suggest is a neurodevelopmental or reproductive toxicant, endocrine disruptor or human carcinogen—including short-term "conditional" registrations.
- *Prevent harmful low-level exposures:* EPA should act on existing evidence that exposures to endocrine disrupting pesticides pose a particular danger to developing children; the long-delayed endocrine disruptor screening program (EDSP) should be swiftly implemented. At the current rate, it will be 2017 before the first set of *only 58 chemicals* are screened.

The best way to protect children from the harms of pesticides is to dramatically reduce the volume used nationwide.

The insecticide chlorpyrifos provides a clear example of the startling flaws in our regulatory system. Over 10 million pounds of the pesticide are still applied in agricultural

When Is There Enough Evidence to Act?

Scientific studies often identify a "link" or "association" between exposure to a particular pesticide and a specific health harm—but individual studies rarely demonstrate definitive causation. Epidemiological studies often lack statistical power, and case control and animal studies may miss key variables such as exposure timing.

A "weight of the evidence" approach recognizes that a body of scientific work will contain conflicting studies, but holds that when a number of well designed, robust studies come to similar conclusions, the findings should be considered valid.*

When such findings involve widespread, significant and irreversible health harms to our children, the bar for taking action should not be high. When credible evidence of harm emerges, a pesticide product should immediately be taken off the market until its manufacturer can prove its safety. Put simply, it is time the burden of proof shifted to the pesticide corporations, rather than regulators—and the public—as it currently stands.

* In addition to choosing non-toxic approaches to pest control (see PAN's Homes, Pets & Gardens online resource at http://www.panna.org/your-health/home-pets-garden), see also the National Pesticide Information Center's page on Pesticides and Children for suggestions on reducing children's exposure in the home: http://npic.orst.edu/health/child.html.

† See, for example, criteria and process for developing the "chemicals of high concern" list in Maine. http://www.maine.gov/dep/safechem/highconcern/chemicals.htm

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Investing in farmers who grow food without relying on chemicals that harm children's health must be a national priority.

fields every year, more than a decade after household uses were withdrawn because of *clear dangers to children's developing brains.*[*] Yet children across the country continue to be exposed—in rural schools and communities, and by eating foods that have been treated with the neurotoxic chemical.

2. Protect children where they live, learn & play

Policymakers need strong tools to protect children from exposure to pesticides where they live, learn and play. Such protections will help keep developing bodies and minds healthy during the years they are most vulnerable to harm from chemical exposures.

We recommend rapid implementation of the following measures:

- *Kid-safe homes, daycares & schools:* EPA should withdraw approval of existing pesticide products and not approve new pesticides for use in homes, daycare centers or schools when scientific evidence indicates the chemicals are possible neurodevelopment or reproductive toxicants, endocrine disruptors or human carcinogens.
- *Safer parks & playgrounds:* State and local officials should enact policies requiring that all public playgrounds, playing fields and parks be managed without using pesticides that studies show are harmful to children's health.
- *Protective buffer zones:* State legislators should establish—or give local governments authority to establish—protective pesticide-free buffer zones around schools, daycare centers and residential neighborhoods in agricultural areas.
- *Healthier school lunches:* Local school districts, state agencies and USDA's Farm-to-school program should provide schools with incentives to procure fresh, local fruits and vegetables that have been grown without pesticides that studies show are harmful to children's health.

3. Invest in farmers stepping off the pesticide treadmill

Investing in farmers who grow food without relying on chemicals that harm children's health must be a national priority. Specifically:

- *Corral resources for farmers:* Federal and state officials should mobilize and coordinate existing resources to help farmers adopt well-known, effective pest management strategies that reduce reliance on pesticides. USDA, EPA and many state agencies and universities have important programs—research, outreach and education—with this stated aim that could be ramped up in complementary ways.
- *Increase investment in innovative farming:* Congress should authorize significant funding for programs supporting farmers' adoption of sustainable practices that reduce use of harmful pesticides. Existing programs receive a small fraction of the funding supplied to programs serving conventional growers.
- *Set use reduction goals:* EPA and USDA should set specific and aggressive national pesticide use reduction goals, focusing first on pesticides studies show to be harmful to children.[†] To track progress toward this goal, farmers should work with applicators and pest control advisors to report their pesticide use to a nationally searchable database.[‡]
- *Source for children's health:* Food distributors should require that their suppliers limit use of pesticides that harm children's health.

Effective agroecological methods exist for production of all major crops—but these approaches are often knowledge-intensive, requiring significant training as well as real changes in farm operation.[§] Growers need direct support to make the shift away from pesticide reliance, including provision of hands-on field training and technical advice from independent experts as well as incentives to invest in agroecological practices.

These proposals are all commonsense measures in the face of clear evidence that our children's wellbeing is at risk. It's time to muster the political will and prioritize the health of our children, grandchildren and future generations.

* Chlorpyrifos was phased out for household use after studies clearly indicated that exposed children had smaller head circumference, a known indicator of reduced cognitive function.

† See Appendix B.

‡ Pesticide use reporting is already in place in California; lessons learned from implementation of this program (established in 1990) should inform and enable rapid adoption of a federal use reporting system.

§ Agroecological practices are based on the application of intricate place-based knowledge of soil/plant/animal interactions designed to prevent or minimize pest problems. Farmers are successfully using such practices in virtually every crop now grown in the U.S.

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165 *Use, Effects and Alternatives to Pesticides in Schools*, Report to the Ranking Minority Member, Committee on Governmental Affairs, U.S. Senate. United States General Accounting Office 1999. See www.gao.gov/new.items/rc00017.pdf.

166 Owens, K. "Schooling of State Pesticide Laws 2010 Update." *Pesticides and You.* Fall 2009 29(3): 9–20.

167 "Child Safe Playing Field Act Signed into Law by New York Governor" Beyond Pesticides Daily News Blog, May 2010. See http://www.beyondpesticides.org/dailynewsblog/?p=3637.

168 *Green Schools Within Reach: Moving Beyond the Healthy Schools Act of 2000.* Californians for Pesticide Reform. See http://pesticidereform.org/article.php?id=385.

169 "Tulare County Residents Win Greater Protection from Dangerous Pesticides: New rules announced for pesticide applications around schools, homes and labor camps," Press Release, Californians for Pesticide Reform. Feb 2008. See www.panna.org/sites/default/files/imported/files/CPR20080220.pdf.

170 White Earth Land Recovery Project, Farm to School Program. See http://nativeharvest.com/node/255 , viewed July 2012.

171 Rauzon, S., M. Wang, N. Studer and P. Crawford. An Evaluation of the School Lunch Initiative. A report by the Dr. Robert C and Veronica Atkins Center for Weight and Health, University of California at Berkeley, Sept 2010. See edibleschoolyard.org/sites/default/files/file/Final%20Report_9-22-10v4_LoRes.pdf.

172 The Olympia School Districts' Organic Choices Program, National Farm to School Network. See http://www.farmtoschool.org/state-programs.php?action=detail&id=8&pid=58, viewed June 2012.

173 *Action Alert: Help protect Ashland Parks, Schools and Waters from Pesticides*. Klamath Siskiyou Wildlands Center. See http://kswild.org/get-involved/ActionAlerts/help-reduce-or-eliminate-pesticides-at-ashland-parks-and-schools, viewed June 2012.

174 *Pesticide-free parks: It's time!* Northwest Coalition for Alternatives to Pesticides, Eugene OR. 2005. See www.pesticide.org/get-the-facts/ncap-publications-and-reports/pesticide-free-parks/pfptime.pdf.

175 "Horticulture: Pesticide reduction." Seattle Parks and Recreation. See http://www.seattle.gov/parks/horticulture/pesticide.htm, viewed June 2012.

176 NJ Senate Environment Committee Passes Nation's Strongest Pesticide Bill, Press Release, Clean Water Action, Jan 2011. See http://www.cleanwater.org/press/nj-senate-environment-committee-passes-nation%E2%80%99s-strongest-pesticide-bill.

177 New Polling Data Indicates Overwhelming Public Support for Chemicals Regulation. Safer Chemicals Healthy Families: Resources. Sept 2010. See http://www.saferchemicals.org/resources/opinion-2010.html

178 Philpott, T. "How Your College Is Selling Out to Big Ag," *Mother Jones*, May 2012. See http://www.motherjones.com/tom-philpott/2012/05/how-agribusiness-dominates-public-ag-research

179 Agribusiness. OpenSecrets.org, Center for Responsive Politics. See http://www.opensecrets.org/industries/indus.php?ind=A, viewed August 2012. See also Undue Influence, Pesticide Action Network at http://www.panna.org/issues/pesticides-profit/undue-influence.

Appendix A

More Science: Key study descriptions

Our intention in undertaking this review was not to conduct a comprehensive evaluation of the evidence. The body of scientific literature exploring how pesticides affect children's health is wide, deep and decades long.

Our goal is to provide a snapshot of recent findings, coming fast and furious in the just the past few years, that—taken together—provide compelling reason for concern about the impact of pesticides on our children's health.

In the report itself we highlight a few of the key findings for each health effect, focusing on studies that were particularly compelling, and/or represented other studies we reviewed with similar findings. We simplified descriptions of each study to provide a basic sense of how the research was conducted and what researchers found. Here in Appendix A we provide a bit more detail on some of the key studies described above, as well as additional studies. Study descriptions are organized by health effect, and alphabetically by author within each category.

Brain & nervous system harms (reduced cognitive function, autism, ADHD)

Bouchard M.F., D.C. Bellinger, R.O. Wright and M.G. Weisskopf."Attention-deficit/hyperactivity disorder and urinary metabolites of organophosphate pesticides." *Pediatrics* 2010. 125(6): e1270–e1277.

This study examines the association between urinary concentrations of organophosphate metabolites and ADHD in children eight to 15 years of age. Researchers analyzed cross-sectional data from the National Health and Nutrition Examination Survey for 1139 children representative of the U.S. population. Urinary DMAP metabolite levels (which are an indicator of exposure to OP pesticides), an ADHD assessment, and household surveys were used in the analysis. The data support the hypothesis that organophosphate exposure, at levels common among U.S. children, may contribute to ADHD prevalence.

Eskenazi B., K. Huen, A. Marks, K.G.Harley, A. Bradman, D.B. Barr, et al. "PON1 and Neurodevelopment in Children from the CHAMACOS Study Exposed to Organophosphate Pesticides in Utero." *Environ Health Perspect.* Aug 2010 118: 1775-1781. See http://dx.doi.org/10.1289/ehp.1002234.

The enzyme paraoxonase 1 (PON1) detoxifies metabolites of some organophosphate (OP) pesticides, andPON1 genetic polymorphisms influence enzyme activity and quantity. The study authors investigated whether PON1 genotypes and enzyme activity levels in mothers and their children were linked to neurodevelopmental changes, and whether PON1 levels and genotypes had an effect on the association of *in utero* exposure to OP pesticides (as assessed by maternal urinary concentrations of dialkyl phosphate metabolites, a marker of OP pesticide exposure) and neurodevelopment and behavior. The researchers found that of the 353 two-year-olds assessed, children with a certain variation of PON1 (the $PON1_{-108T}$ allele) scored more poorly on the Mental Development Index and somewhat lower on the Psychomotor Development Index. The authors concluded that while the variations of PON1 were associated with outcomes in child neurobehavioral development, additional research is needed to confirm whether it modifies the relation with *in utero* exposure to OP pesticides.

Pessah I.N., P.J. Lein. "Evidence for environmental susceptibility in autism" in: *Autism,* (Zimmerman AW, ed). Totowa, NJ: Humana Press 2008 409–428.

The authors aim to illustrate how research into the pathophysiology and genetics of autism may inform the identification of environmental susceptibility factors that promote adverse outcomes in brain development. They highlight three examples of gene-environment interactions that are likely to contribute to autism risk, including: (1) pesticides that interfere with the neurotransmitter acetylcholine; (2) pesticides that interfere with γ-aminobutyric acid (GABA) neurotransmission; and (3) persistent organic pollutants that directly

A Study by Any Other Name…

Epidemiological study: A study of distribution or patterns in health trends or characteristics and their causes or influences in specific populations. Includes both case-control and all types of cohort studies.

Case-control study: Compares a "case" group (e.g., U.S. children ages 0–14 with cancer) with a group serving as a control (e.g., cancer-free U.S. children ages 0–14).

Cohort study: Profiles a specific population where shared exposure may be assumed, such as occupational exposure to pesticides among farmworkers.

Prospective cohort study: Follows a group that is slightly different in some respects. (i.e., studying a cohort of pesticide applicators who use varying protective methods while working with pesticides.)

Longitudinal cohort study: Tracks a specific group over time. For example, a UC Berkeley study on the central California coast has followed a specific group of children from conception through adolescence.

Meta-analysis: Pulls together several studies on the same topic and does further statistical analysis on the basic findings.

Review: Examines the "state of the science" and often provides evaluation of conflicting pieces of data. Review authors give their view on what is currently happening in the field.

alter calcium ion (Ca^{2+}) signaling pathways and Ca^{2+}-dependent effectors. If both genetic factors and environmental ones converge to interrupt the same neurotransmitter or signaling systems at critical times during development, adverse effects can be amplified.

Rauh V.A., F.P. Perera, M.K. Horton, R.M. Whyatt, R. Bansal, X. Hao X, et al. "Brain anomalies in children exposed prenatally to a common organophosphate pesticide." *Proc Natl Acad Sci* 2012 109(20):7871-6.

This study investigated associations between prenatal exposure to chlorpyrifos and brain morphology (examining brain structure). With a sample of 40 children—who experienced low prenatal exposure to tobacco smoke and polycyclic aromatic hydrocarbons—20 subjects with high chlorpyrifos exposure were compared to 20 low-exposure subjects. The data revealed a significant association between prenatal exposure to chlorpyrifos, at standard use levels, and structural changes in the developing human brain. High exposure was associated with the enlargement of several areas of the brain and in preliminary analyses, the reversal of sex differences or a lack of expected sex differences.

Shafer, T.J., D.A. Meyer and K.M. Crofton. "Developmental Neurotoxicity of Pyrethroid Insecticides: Critical Review and Future Research Needs." *Environmental Health Perspectives* 113, no. 2 Oct 2004: 123–136 .

A review of pyrethroid insecticides and the data related to potential developmental neurotoxic effects of pyrethroids, with recommendations for improving study design and statistical analyses. The review discusses the various effects on voltage-sensitive sodium channels, which are a primary target of pyrethroids.

Childhood cancers

Carozza S.E., B. Li, K. Elgethun and R. Whitworth."Risk of childhood cancers associated with residence in agriculturally intense areas in the United States." *Environ Health Persp* 2008 116(4): 559–565.

Researchers from the U.S. evaluated whether children under the age of 15 who live in a county associated with greater agriculture production—and hence, exposure to pesticide drift—experienced different risk rates for developing cancer. Using incidence data for U.S. children provided by the North American Association of Central Cancer Registries, researchers were able to compare county-level, sex- and age-specific rates of childhood cancer with agricultural census data containing county acreage, percent cropland, and percent acres for specific crops. The data revealed statistically significant increase in risk for many types of childhood cancers for residents living in those counties with a moderate to high level of agricultural activity. Risk for different cancers varied by type of crop; for example, there was increased risk of non-Hodgkin lymphoma and thyroid cancer associated with residence at diagnosis in counties that produced corn or oats.

Infante-Rivard C, S. Weichenthal. Pesticides and childhood cancer: an update of Zahm and Ward's 1998 review. *J Toxicol Environ Health B Crit Rev* 2007 10(1): 81–99.

Infante-Rivard and Weichenthal reviewed the epidemiological and ecological studies published since the 1998 Zahm and Ward review. The authors found that15 case-control studies, four cohort studies, and two ecological studies have been published since this review, and 15 of these 21 studies reported a statistically significant increase in risk of childhood cancer among children whose parents were experienced occupational pesticide exposure. These studies found that the risk of all childhood cancers increased with the frequency of maternal exposure to herbicides and plant insecticides. Furthermore, maternal and paternal exposure to insecticides and herbicides up to five years before having a child increased risk of all childhood brain tumors, astroglial tumors, non-Hodgkin's lymphoma, primitive neuroectodermal tumors, and other glial tumors. Parental occupation in agriculture is also associated with an increased risk of Ewing's sarcoma. The authors conclude that evidence supports an association between at least some pesticide exposure and childhood cancer.

Kristensen, P., A. Andersen, L.M. Irgens, A.S. Bye and L. Sundheim. "Cancer in Offspring of Parents Engaged in Agricultural Activities in Norway: Incidence and Risk Factors in the Farm Environment." *International Journal of Cancer. Journal International Du Cancer.* Jan 1996 65 (1): 39–50.

A cohort study in Norway of 323,359 children born between 1952–1991 reported that children 0-14 years had a nearly doubled risk for brain tumors and a more than tripled risk for neuroepithelial tumors except for astrocytomas associated with pesticide purchase. These associations were stronger when sub-groups, such as growing up on the farm, were considered. Offspring born April–June showed a clustering of neuroepithelial brain tumors, suggesting that paternal exposure during periods of increased pesticide application, from 0–3 months before conception, may have been a factor.

Meinert, R., J. Schuz, U. Kaletsch and J. Michaelis. "Leukemia and Non-Hodgkins Lymphona in Childhood and Exposure to Pesticides: Results of a Register-based Case-Control Study in Germany." *Am Journal of Epidemiology* 2000. 151 (7): 639-646.

A case-control study conducted in Germany from 1993-1997 found parental occupational exposure to be related to childhood cancer regardless of period of exposure and type of cancer, which the authors point out might be due to different recall of past exposures between parents of cases and parents of controls. Residential insecticide use was associated with childhood lymphoma, both professional exterminator and parental usage were significantly associated with increased risk.

Nielsen S.S., R. McKean-Cowdin, F.M. Farin, E.A. Holly, S. Preston-Martin and B.A. Mueller. "Childhood brain tumors, residential insecticide exposure, and pesticide metabolism genes." *Environ Health Persp* 2009 118(1): 144-149.

Researchers in California and Washington found evidence of increased risk of childhood brain tumors (CBT) associated with certain genetic polymorphisms when kids were exposed to insecticides. Strong interactions between genotype and insecticide exposure during childhood was observed. Among exposed children, CBT risk increased per $PON1_{-108T}$ allele, whereas among children never exposed, CBT was not increased. Nielsen et al. concluded childhood exposure to organophosphorus pesticides coupled with a reduced ability to detoxify these pesticides, may be associated with CBT.

van Wijngaarden E, P.A. Stewart, A.F. Olshan, D.A. Savitz and G.R. Bunin. "Parental occupational exposure to pesticides and childhood brain cancer." *Am J Epidemiol* 2003. 157(11): 989–997.

Researchers from the U.S. evaluated parental exposure to pesticides at home or on the job in relation to the occurrence of brain cancer in children. The sample consisted of children diagnosed with cancer and matching controls from four U.S. states. Interviews were performed with the biological mothers of the subjects to assess the residential and occupational exposure to pesticides in the two years before the child was born. The data revealed a significant risk of astrocytoma associated with residential use and exposure to herbicides. Combining parental exposures to herbicides form both residential and occupational sources, the elevated risk remained significant.

Birth defects

Brender, J.D., M. Felkner, L. Suarez, M.A. Canfield and J.P. Henry. "Maternal Pesticide Exposure and Neural Tube Defects in Mexican Americans." *Annals of Epidemiology*. 2010 20(1): 16–22.

Researchers investigated the relationship between maternal pesticide exposures and neural tube defects (NTDs) in offspring comparing to groups of Mexican American women (184 in case group, 225 for comparison). After adjusting for differences in maternal education levels, smoking, and folate intake during pregnancy, women who reported using pesticides in their homes or yards were twice as likely to have children with NTDs than women not reporting exposures (95% confidence interval [CI], 1.2–3.1) Case-women were also more likely to live within ¼ mile of agricultural fields. As possible sources of pesticide exposure increased, risk of NTDs also increased. Associations were stronger for risk of anencephaly than for spina bifida.

Garry V.F., M.E. Harkins, L.L. Erickson, L.K. Long-Simpson, S.E. Holland and B.L. Burroughs. "Birth defects, season of conception, and sex of children born to pesticide applicators living in the Red River Valley of Minnesota, USA." *Environ Health Persp* 2002. 110(3): 441–449.

A cross-sectional study performed in the Red River Valley of Minnesota examined the reproductive health outcomes in 695 farm families (analyzed data from 1,532 children) from parent-reported birth defects. Researchers determined conceptions in the spring time led to significantly more children born with birth defects, compared to children conceived in any other season. Their data suggests environmental agents present in the spring, like herbicides, have an adverse effect on the birth defect rate. Furthermore, the data revealed an association between fungicide exposure and the determination of child sex—affecting the survival rate of the male fetus (female to male birth ration is 1.25 to 1).

Gaspari L., F. Paris, C. Jandel, N. Kalfa, M. Orsini, J.P. Daures and C. Sultan. "Prenatal environmental risk factors for genital malformations in a population of 1442 french male newborns: a nested case-control study." *Hum Reprod* 2011. 26(11): 3155–3162.

Researchers from France analyzed a physician's examinations and parental interviews for 1442 full-term newborn males in southern France to identify risk factors for male external genital malformations, with a focus on parental occupational exposure to endocrine disrupting chemicals, such as organochlorine pesticides. Infants were examined for cryptochidism, hypospadias, and micropenis, while a questionnaire asked parents about the pregnancy, personal characteristics, lifestyle, and occupational exposure to EDCs. In total, 39 cases of genital malformation were reported (2.70%). A significant relationship was observed between newborn cryptochidism, hypospadias or micropenis and parental occupational exposure to pesticides with the odds of genital malformation increasing 4.41-fold. These data supports the hypothesis that prenatal contamination by pesticides may be a potential risk factor for newborn male external genital malformation.

Rocheleau, C.M, P.A. Romitti and L.K. Dennis. "Pesticides and Hypospadias: a Meta-analysis." *Journal of Pediatric Urology*. Feb 2009 5(1): 17–24.

A meta-analysis of studies done in 7 different countries (Canada, Denmark, Italy, Netherlands, Norway, Spain, US) indicated a 36% increased risk of hypospadia with maternal occupational exposure and a 19% increased risk of hypospadias with paternal occupational exposure.

Winchester PD, Huskins J, Ying J. 2009. Agrichemicals in surface water and birth defects in the United States. *Acta Paediatr* 98(4): 664–669.

Researchers from Indiana and Ohio compared water data from the USGS National Water Quality Assessment (NAWQA)—measuring the levels of nitrates, atrazine, and other pesticides in surface water—and Centers for Disease Control data detailing monthly pregnancy and birth outcome outcomes. The data reveal that between 1996 and 2002 women in the US were significantly more likely to give birth to a child with birth defects if conception had occurred in the months of April through July. NAWQA surface water samples indicate that concentrations of atrazine, nitrates, and other pesticides were also higher in the months of April through July. This correlation was statistically significant, demonstrating elevated concentrations of agrichemicals in surface water coincided with a higher risk of birth defects among live births for children conceived between April and July.

Early puberty

Aksglaede L., K. Sorensen, J.H. Petersen, N.E. Skakkebaek and A. Juul. "Recent decline in age at breast development: the Copenhagen puberty study." *Pediatrics* 2009. 123(5): e932-939.

Researchers from Denmark collected data from 2095 females aged 5.6 to 20 years in two Copenhagen cohorts (1991–1993 and 2006–2008) to examine differences in breast development. Using the most accurate method of palpation, Aksglaede et al. found the onset of puberty—defined as the mean estimated age at the attainment of glandular breast tissue—occurred significantly earlier in the 2006 cohort. The ages at which menarche and pubic hair development occurred also slightly decreased in the 2006 cohort. As a result of these timing changes in early and later markers of puberty, the length of puberty appears to have increased. The authors interpreted these observations as indicative of gonadotropin-independent estrogenic actions at the level of breast development, rather than an earlier activation of the pituitary-gonadal axis. These changes in timing could not be explained by alterations in reproductive hormones and BMI, suggesting other factors involved need to be explored.

Gladen B., N. Ragan and W. Rogan. "Pubertal growth and development and prenatal and lactational exposure to polychlorinated biphenyls and Dichlorodiphenyl Dichloroethene." *Pediatrics* 2000. 136(4): 490-496.

Researchers from the National Institute of Environmental Health Sciences explored the relationship between prenatal and early-life exposure to PCBs and DDE on children. This is one of a very few studies examining environmental contaminants and male puberty onset. Using 594 children from the North Carolina Infant Feeding Study cohort, they found no effect on the ages at which puberty began. However, the height and weight (adjusted for height) of boys at puberty increased with transplacental exposure to DDE.

Massart F., P. Seppia, D. Pardi, S. Lucchesi, C. Meossi, L. Gagliardi et al. "High incidence of central precocious puberty in a bounded geographic area of northwest Tuscany: an estrogen disrupter epidemic?" *Gynecol Endocrinol* 2005. 20(2): 92–98.

Researchers in Italy preformed an analysis of central precocious puberty (CPP) distribution in northwest Tuscany (NWT). The overall incidence rate of sexual precocity is estimated at 10–20 per 100, a rate similar to that found in four of the cities in the NWT sample; however 47 percent of the CPP cases found in NWT were in the Viareggio area, a rate of 161 per 100,000. This area hosts a high density of navy yards and greenhouses—consequently it is at higher risk of chemical estrogen pollution. As this population represented only 13.73 percent of the total population of NWT, living in this area significantly increased the risk of CPP. The definite geographic distribution of CPP in this suggests that environmental involvement/pollution may be a major determinant of CPP development.

Nebesio T and O. Hirsh Pescovitz. " Historical perspectives." *Endocrinologist* 2005. 15(1):44-48.

Nebesio and Pescovitz reviewed reports alleging endocrine disruptors blamed for altering the age of normal puberty, including an examination of studies implicating pesticides and accidental environmental exposures. Studies reviewed include two seminal studies on early puberty in girls: Vasiliu et al.'s (2004) examination of the Michigan anglers cohort daughters and Krzstevska-Konstantinova et al.'s (2001) examination of precocious puberty in native and non-native Belgian girls. Nebesio and Hirsch Pescovitz (2005) also review Boneh et al. (1989), who examined cases of girls with precocious sexual development from Jerusalem over a 10-year time period and found strong evidence for a seasonal increase in incidences of early sex development observed (from April–June). Seasonal pesticide usage was a potential cause, but the reasons for this were unknown.

Steingraber S. 2007. *The falling age of puberty in U.S. girls: what we know, what we need to know.* The Breast Cancer Fund.

In this report Steingraber suggests that pubertal onset and menarche are two sexual maturation processes that appear to be becoming uncoupled, therefore increasing the length of puberty in girls. The author cites environmental contaminants as the cause in light of recent evidence suggesting even minimal exposure to an endocrine disruptor on sex hormones can have a profound consequence in childhood.

Obesity & diabetes

Baillie-Hamilton, P.F. "Chemical toxins: a hypothesis to explain the global obesity epidemic." *J Altern Complement Med* 2002 8(2): 185–192.

Hamilton puts forth a new hypothesis to explain the global obesity epidemic: chemical toxins. Overeating and inactivity do not fully explain the current trend in obesity. Baillie-Hamilton calls for an examination of environmental causes rather than genetic factors. The sympathetic nervous system is perhaps the key weight-controlling system, and is targeted by many of the commonest synthetic chemicals. Numerous widely used synthetic chemicals induce weight gain, including pesticides (specifically organochlorines and organophosphates). They do so by disrupting major weight controlling hormones, altering levels and sensitivity to neurotransmitters, interfering with metabolic processes, and causing widespread damage to body tissues. These interferences change appetite, food efficiency, and the metabolism of fats, proteins, and carbohydrates.

Janesick, A. and B. Blumberg. "Endocrine Disrupting Chemicals and the Developmental Programming of Adipogenesis and Obesity." *Birth Defects Research Part C: Embryo Today*: Reviews 2011. 93, no. 1: 34–50.

This review article explores possible explanations for the variation in individual propensity to gain weight and accrue body mass, even at identical levels of caloric input. The authors review evidence from clinical, epidemiological, and biological studies showing that obesity is largely programmed early in life, including prenatally. They examine the environmental obesogen hypothesis, which holds that "prenatal or early life exposure to certain endocrine disrupting chemicals can predispose exposed individuals to increased fat mass and obesity. Obesogen exposure can alter the epigenome of multipotent stromal stem cells, biasing them toward the adipocyte lineage at the expense of bone." Individuals exposed to obesogens early in life or prenatally might thus experience changes in their stem cell compartment, which in turn influences adipogenic fate

Lee D.H., I.K. Lee, K. Song, M. Steffes, W. Toscano, B.A. Baker and D.R. Jacobs."A strong dose-response relation between serum concentrations of persistent organic pollutants and diabetes: results from the National Health and Examination Survey 1999-2002." *Diabetes Care* 2006 29(7): 1638–1644.

Researchers performed a cross-sectional examination of the association between serum concentrations of six POPs (selected because they were detectable in greater than 80 percent of participants) and diabetes prevalence. After adjustments were made for confounding variables (age, sex, race and ethnicity, poverty income ratio, BMI and waist circumference) diabetes prevalence was strongly positively associated with lipid adjustment serum concentrations of all six POPs tested for in the sample of 2,016 adult participants from the National Health and Nutrition Examination Survey 1999–2002. Furthermore, the association between POPs and diabetes was much stronger among obese subjects compared to lean subjects.

Lee, D.H., M.W. Steffes, A. Sjödin, R.S. Jones, L.L. Needham, D.R. Jacobs. "Low dose organochlorine pesticides and polychlorinated biphenyls predict obesity,

dyslipidemia, and insulin resistance among people free of diabetes." *PLoS One* 2011 6(1): e15977.

In a follow up study to their 2010 study of low-dose persistent organic pollutant (POP) exposure and prediction of type 2 diabetes, Lee et al. conducted a nested case-control study to explore the relationship between serum concentrations of POPs and adiposity, dyslipidemia, and insulin resistance among people confirmed to be diabetes free (assessing study subjects on 5 occasions over 20 years). Researchers concluded that simultaneous exposure to various OC pesticides and PCBs in the general population may contribute to the development of obesity, dyslipidemia, and insulin resistance—common precursors of type 2 diabetes and cardiovascular diseases—among those without diabetes. POPs exposure may also contribute to excess adiposity and other dysmetabolic conditions. Ten POPs were found to predict future higher triglycerides and 14 POPs predicted lower HDL-cholesterol. Among organochorine pesticides, p,p'-DDE most consistently predicted higher BMI, triglycerides and HOMA-IR, as well as a lower HDL-cholesterol at year 20.

Newbold R.R., E. Padilla-Banks, R.J. Snyder, T.M. Phillips and W.M. Jefferson. "Developmental exposure to endocrine disruptors and the obesity epidemic." Reprod Toxicol 2007. 23(3): 290–296.

Research from the US has shown an association between exposure to environmental endocrine disrupting chemicals with the development of obesity. Researchers utilize an animal model of developmental exposure to diethylstilbestrol (DES)—a potent perinatal endocrine disruptor with estrogenic activity—to study the mechanisms involved in programming an organism for obesity. Their data supports the idea that brief exposure early in life to environmental endocrine disrupting chemicals, especially those with estrogenic activity, like DES. These chemicals may contribute to overweight and obesity as well as other obesity-associated diseases (type 2 diabetes and cardiovascular disease). This research complicates the current understanding of obesity and necessitates a consideration of more complex factors, including environmental chemicals.

Asthma

Hernández A.F., T. Parrón and R. Alarcón. "Pesticides and asthma." *Curr Opin Allergy Clin Immunol* 2011 11(2): 90–96.

Hernández et al. performed a review of clinical and epidemiological studies that link exposure to pesticides, asthma attacks, and an increased risk of developing asthma. These authors concluded that while many pesticides are sensitizers or irritants, their potential to sensitize is limited. However, more importantly, pesticides may increase the risk of developing asthma, exacerbate a previous asthmatic condition or even trigger asthma attacks by increasing bronchial hyper-responsiveness.

Salam MT, Y.F. Li, B. Langholz, F.D. Gilliland."Early-life environmental risk factors for asthma: findings from the Children's Health Study." *Environ Health Perspect* 2003 112(6): 760–765.

Researchers from the University of Southern California selected 4,244 subjects from the Children's Health Study conducted in 12 southern California communities to measure the relationship between childhood environmental exposures and asthma risk. Matching those subjects diagnosed with asthma before age five with asthma-free counterparts that acted as controls (matched for age, sex, community of residence, and in utero exposure to maternal smoking), the authors concluded that environmental exposures during the first year of life are associated with an increase in the risk for early-onset persistent asthma, a subtype of asthma associated with long-term morbidity. Compared to never-exposed children, children exposed to herbicides within the first year of life had a 4.6-fold increased risk of asthma and children exposed to pesticides had a 2.4-fold increase in risk—considered together children exposed to any pesticide or herbicide in the first year of life experience a 2.53-fold higher risk of asthma compared to children who were never exposed to either of those.

Salameh P.R., I. Baldim, P. Brochard, C. Raherison, B.A. Saleh and R. Salamon. "Respiratory symptoms in children and exposure to pesticides." *Eur Respir J* 2003 22(3): 507–512.

Public health researchers from Lebanese University in Lebanon and Victor Segalen Bordeaux II University in France conducted a cross-sectional study to evaluate if exposure to pesticides resulted in chronic effects on the respiratory health of Lebanese children. From 19 public schools, 3,291 randomly selected school children—aged five to 16 years—revealed exposure (residential, paraoccupational, and domestic) to pesticides was significantly associated with respiratory disease (1.82-fold higher) and chronic respiratory symptoms such as chronic phlegm, chronic wheezing, and wheezing at any point (the only exception was chronic cough). Twelve percent of the sample reported a chronic respiratory disease and of those, 84 reported a medically confirmed asthma diagnosis (2.6 percent of the sample).

Sunyer J, M. Torrent, R. Garcia-Esteban, N. Ribas-Fitó, D. Carrizo, I. Romieu et al. "Early exposure to Dichlorodiphenyldichloroethylene, breastfeeding and asthma at age six." *Clin Exp Allergy* 2006 36(10): 1236–1241.

Researchers from Spain and the United Kingdom conducted a longitudinal study from a sample of 468 Minorcan children (Balearic Island in the northwest Mediterranean sea with no local pollution sources) to examine the association between prenatal exposure to DDE and other organochlorine compounds and asthma. Asthma was defined as the presence of a wheeze, persistent wheezing, or parental report of doctor-diagnosed asthma at age four. All children were born with quantifiable levels of DDE and PCB compounds. Wheezing at age four was reported for 11.6 percent of all children. Wheezing at four years of age increased with DDE concentration, particularly at the highest quartile, which was also found for persistent wheezing. This association was maintained even after adjusting for potential confounding variables. These results corroborated the association established between DDE and asthma in German school children conducted by Karmaus et al. in 2001.

Appendix B: Top Pesticides Used in Agriculture & at Home

Key
? – Insufficient data
ND – No data available
I – Insecticide
H – Herbicide
F – Fungicide
PGR – Plant growth regulator
FUM – Fumigant

Table B-1: Most Commonly Used Pesticide Active Ingredients - Agriculture Listed by volume of use[1]

Pesticide & use level range (millions of lbs active ingredient)	PAN HHP[2]	Type	High[3] acute toxicity	Carcin-ogen	Acute neuro-toxicant (ChE inhibitor)	Devel. or reprod. toxicant	Endocrine disruptor	Primary crops	Food residues[4]
Glyphosate (180-185)		H				?	?	Hay/pasture, soybeans, corn	ND
Atrazine (73-78)	Y	H		Y		?	suspected	Corn, sugarcane	Spinach, wheat, onions, lettuce, water
Metam-sodium (50-55)	Y	FUM	Y	Y		Y	suspected	Potatoes, carrots, tomatoes, onions, peanuts	ND
Metolachlor, (S) (30-35)	Y	H		possible		?	suspected	Tomatoes, beans, corn, cotton	Oats, celery, water, corn
Acetochlor (28-33)	Y	H		Y		?	suspected	Corn, popcorn	Water
Dichlorpropene (27-32)		FUM	Y	Y		?	?	Strawberries, sweet potatoes, tree nuts	
2,4-D (25-29)	Y	H		possible		?	suspected	Grasses, wheat, citrus fruits, tree nuts	Potatoes, water
Methyl bromide (11-15)	Y	FUM	Y			Y	suspected	Tomatoes, strawberries, almonds, peppers, watermelon, cucumbers	ND
Chloropicrin (9-11)	Y	FUM	Y	?		?	?	Tobacco, tomatoes, strawberries, bell peppers	ND
Pendimethalin (7-9)	Y	H		possible		?	suspected	Soybeans, corn, cotton, peanuts	Carrots, collard greens, kale
Ethephon (7-9)		PGR			Y	?	?	Cotton, walnuts, grapes, tomatoes	ND
Chlorothalonil (7-9)	Y	F	Y	Y		?	?	Tomatoes, watermelons, onions	Cranberries, celery, green beans
Metam Potassium (7-9)		FUM	Y	Y		Y	?	Lettuce, potatoes	ND
Chlorpyrifos (7-9)	Y	I			Y	?	suspected	Tree nuts, apples, alfalfa, broccoli, citrus, grapes, sweet corn	Apples, bell peppers, cranberries, kale, grapes, peaches
Copper Hydroxide (6-8)		F				?	?	Tree nuts, grapes, peaches	ND
Simazine (5-7)	Y	H				Y	suspected	Corn, citrus, grapes, tree nuts	Blueberries, kale, water, oranges
Trifluralin (5-7)	Y	H		possible		?	suspected	Soybeans, cotton, green beans, broccoli, tomatoes	Carrots, spinach, wheat, soybeans, broccoli
Propanil (4-6)	Y	H		possible		?	suspected	Rice, oats, barley, wheat	Wheat
Mancozeb (4-6)	Y	F		Y		Y	suspected	Apples, tomatoes, onions, watermelon	ND
Acephate (2-4)	Y	I		possible	Y	?	suspected	Cotton, tobacco, cranberries, mint	Green beans, bell peppers
Diuron[5] (2-4)	Y	H		Y		Y	suspected	Oranges	Asparagus, oranges, water, potatoes
MCPA (2-4)	Y	H	Y	possible		?	?	Flax, barley, wheat, rice	water
Paraquat (2-4)	Y	H	Y			?	suspected	Corn, soybeans, cotton, apples	ND
Dimethenamid (2-4)	Y	H		possible		?	?	Corn, soybeans, sugarbeets	Soybeans, water

Table B-2: Most Commonly Used Pesticide Active Ingredients – Home & Garden

Listed by volume of use

Pesticide & use level range (millions of lbs active ingredient)	PAN HHP	Type	High acute toxicity	Carcinogen	Acute neurotoxicant (ChE inhibitor)	Devel. or reprod. toxicant	Endocrine disruptor
2,4-D (8-11)	Y	H		possible		?	suspected
Glyphosate (5-8)		H				?	?
Carbaryl (4-6)	Y	I		Y	Y	Y	suspected
Mecoprop-P (MCPP) (4-6)	Y	H		possible		?	?
Pendimethalin (3-5)	Y	H		possible		?	suspected
Pyrethroids[6] (2-4)	Y	I	Y	Y		Y	suspected
Malathion (2-4)	Y	I	Y	possible	Y	Y	suspected
Dicamba (1-3)		H				Y	?
Malathion (2-4)	Y	I	Y	possible	Y	Y	suspected
Trifluralin (1-3)	Y	H		possible		?	suspected
Pelargonic Acid (< 1)		H/F		?		?	?

Notes

1 See Table 3.6 and 3.7 in *Pesticide Industry Sales & Usage, 2006 and 2007 Market Estimates,* U.S. EPA, Washington, DC Feb 2011. See www.epa.gov/opp00001/pestsales/07pestsales/market_estimates2007.pdf. Aldicarb was removed from the list as registration was withdrawn in 2010.

2 PAN International has compiled and published a list of Highly Hazardous Pesticides (HHPs) that are harmful to human health and the environment, and targeted for global reduction and elimination. See www.panna.org/issues/publication/pan-international-list-highly-hazardous-pesticides.

3 PAN's online pesticide database provides an explanation of these categories and additional toxicity, use and regulatory information for these and other pesticides. See www.pesticideinfo.org.

4 Based on USDA's Pesticide Data Program, as listed on www.whatsonmyfood.org.

5 Noted health effects not applicable for products with < 7% diuron, and applied to foliage.

6 Health hazards of specific pyrethroids vary, the effects indicated here represent those with most hazardous potential effects.

Appendix C

Online Resources & Tools

This compilation highlights a number of key online resources available through government agencies and public interest groups. It is not intended to be comprehensive.

Pesticide use data

California pesticide use reporting: *calpip.cdpr.ca.gov*

EPA Pesticide Industry Sales & Usage: *www.epa.gov/opp00001/pestsales*

USDA National Agricultural Statistics Service: *www.nass.usda.gov*

Pesticide health harms

Agency for Toxic Substances & Disease Registry, ToxFAQs: *www.atsdr.cdc.gov/az/c.html*

Collaborative on Health & the Environment, Toxicant & Disease Database: *www.healthandenvironment.org/tddb*

EPA Pesticides & Human Health Issues: *www.epa.gov/opp00001/health/human.htm*

EPA Recognition & Management of Pesticide Poisonings: *npic.orst.edu/rmpp.htm*

Ontario College of Family Physicians, Systematic Review of Pesticide Human Health Effects: *www.ocfp.on.ca/docs/pesticides-paper/pesticides-paper.pdf*

PAN International Highly Hazardous Pesticides: *www.panna.org/issues/publication/pan-international-list-highly-hazardous-pesticides*

PAN's pesticide database: *www.pesticideinfo.org*

Physicians for Social Responsibility, Pesticides & Human Health: A Resource For Health Care Professionals: *www.psr-la.org/resources/reports-training-materials/#Pesticides*

The Endocrine Disruption Exchange (TEDX): *www.endocrinedisruption.com/pesticides.introduction.php*

Pesticides & children's health

Beyond Pesticides, Learning/Developmental Disorders resource page: *www.beyondpesticides.org/health/learningdevelopmental.htm*

Center for Environmental Research & Children's Health: *cerch.org/research-programs/chamacos*

EPA Pesticides & Children: *www.epa.gov/opp00001/health/children.htm*

National Academy of Sciences: *www.nap.edu/catalog.php?record_id=2126*

PAN's Children's health page: *www.panna.org/children*

Pesticide food residues

FDA Total Diet Study: *www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/TotalDietStudy/default.htm*

Whats On My Food? database (also includes health effect data): *www.whatsonmyfood.org*

USDA Pesticide Data Program: *www.ams.usda.gov/AMSv1.0/pdp*

Childhood disease & disorders

American Academy of Pediatrics: *www.aap.org*

CDC Child Health Statistics: *www.cdc.gov/nchs/fastats/children.htm*

Children's environmental health

Children's Environmental Health Network: *www.cehn.org*—A national multidisciplinary organization whose mission is to protect the developing child from environmental health hazards and promote a healthier environment.

Children's Environmental Health Project: *www.cape.ca/children*—A project of the Canadian Association of Physicians for the Environment, CEHP is intended to introduce clinicians (and their patients) to children's environmental health issues. Information on the health effects from environmental exposures is presented in a systems approach.

Healthy Child, Healthy World: *healthychild.org*—Protecting children's health and wellbeing from harmful environmental exposures through education and prevention strategies.

Healthy Kids: *www.healthy-kids.info—Provides resources and programs to help educators, health p*rofessionals, community officials, organizations, policy makers and parents work together to ensure schools are safe for children's healthy development.

Learning & Developmental Disabilities Initiative: *www.healthandenvironment.org/initiatives/learning*—An international partnership fostering collaboration among LDD organizations, researchers, health professionals and environmental health groups to address concerns about the impact environmental pollutants may have on children's neurological health.

Making our Milk Safe (MOMS): *www.safemilk.org*—A national grassroots movement of mothers working to create a healthier, safer environment for children, MOMS engages in education, advocacy and corporate campaigns.

Pediatric Environmental Health Specialty Units: *www.aoec.org/PEHSU.htm*—ATSDR and EPA support this network to provide education for health professionals, public health officials and others about the topic of children's environmental health.

Physicians for Social Responsibility: *www.psr.org/resources/pediatric-toolkit.html*—PSR has developed a pediatric environmental health toolkit that combines easy-to-use reference guides for health providers and user-friendly health education materials on preventing exposures to toxic chemicals and other substances that affect infant and child health. The toolkit is endorsed by the American Academy of Pediatrics.

Safer Chemicals, Healthy Families: *www.saferchemicals.org*—A coalition pressing for reform of national chemicals policy. SCHF represents more than 11 million individuals including parents, health professionals, advocates for people with learning and developmental disabilities, reproductive health advocates, environmentalists and businesses.

The Children's Environmental Health Institute: *cehi.org*—Works to identify, validate and develop solutions to address adverse health effects to children occurring as a consequence of exposure to hazardous environmental substances.

Pesticide Action Network
NORTH AMERICA

Pesticide Action Network North America
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