India - Pesticides and Health Meeting

8th to 10th October 2002

Indian Social Institute, Bangalore

Collectively organised by

Community Health Cell, Bangalore
Community for Resource Education, Hyderabad
Corpwatch India
Greenpeace India
Institute for Cultural Research and Action (ICRA), Bangalore
Paryavaran Suraksha Samithi, Gujarath
Pesticide Action Network- Asia Pacific
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"A healthy farm culture can be based only upon familiarity and can grow only among a people soundly established upon the land; it nourishes and safeguards human intelligence of the earth that no amount of technology can satisfactorily replace. The growth of such a culture was once a strong possibility in the farm communities of this country. We now have only the sad remnants of those communities. If we allow another generation to pass without doing what is necessary to enhance and embolden the possibility now perishing with them, we will lose it altogether. And then we will not only invoke calamity - we will deserve it."

Wendell Berry

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What follows is a collection of papers on the topic "pesticides and health". India is world leader in pesticide contamination matching with some of the most contaminated countries. But for us who lived in the richness of biodiversity and culture the introduction of the registered poisons - the product of corporate indulgence on natural systems for private profits is relatively a strange thing. So it took time to understand and respond and sadly we have paid heavy toll by impairing our communities.

The village communities and the public interest groups always used to respond to criminal acts against the collective good. The shift from peasant sciences of survival and wisdom to the information and market driven world, and we are once again lost. Current attempts by the individuals and groups are to rediscover where we are and then position ourselves in the struggle for our survival. Many communities are putting efforts to network and join together. The struggle for justice in Bhopal is declaration by all of us that we will not surrender and in the 18th year with more meaning and purpose we continue.
We hope that this meeting on pesticides will be one of such steps to understand where we are in terms of dis-information and illusions and in which direction we have our future and also to reinvent the hope of being in a poison free world of ours.

We have only poisons to lose.

Jayakumar C.

October 8, 2002.

Pesticides - Killers in Our Midst

by Dr. Marion Moses

Introduction

Pesticides are toxic chemicals delib erately added to our environment. They are poisons by design whose purpose is to kill or harm living things. They can kill or harm human beings as well.

Many of the pesticides being used in farms, orchards, plantations and rural rice fields around the world are highly toxic. Farmers and agricultural workers are heavily exposed to pesticides known to damage the brain and nervous system or that cause cancers, birth defects, miscarriages and stillbirths. Many of the pesticides they are exposed to are banned or severely restricted in other countries. ***(See Box: Banned Pesticides are Still Traded).***

A farmer in North Sumatra, Indonesia, spraying while walking through pesticide sprayed fields, with no protective clothing. Photo: PAN North Sumatra.

In rural Asia for example, the use of pesticides has permeated even the remotest village. The availability of highly toxic pesticides, lack of information and knowledge of their hazards, aggressive marketing by the industry as well as poverty, illiteracy, and lack of health facilities ensure that pesticides are a major cause of poisoning in rural farming communities. Impacts on the health of women and children are of a particular concern.

The severity and extent of the problems described by women working in rural farms and plantations in Asia are shocking. Pesticide exposure is a likely source of many of the health problems documented by groups like PAN Asia and the Pacific. Unlike other parts of the world, women in Asia have more direct and heavier exposure to pesticides than their sisters in other regions.

The majority of workers who apply pesticides in plantations, in countries like Malaysia for example, are women. They also mix pesticides and pour them into the spray containers which is an even more serious health risk since they are handling the concentrated products. Often, the women do not even know the names or hazards of the pesticides they are mixing and applying. They receive no education or training in how to use them properly or how to protect themselves and their children. Even if they were provided full protective equipment and clothing appropriate for pesticides they are working with, they would still be at risk from heat stress and even death from heat stroke. This is especially true since they do not have enough water (or sometimes not any water) to drink

Banned Pesticides are Still Traded

Though several highly hazardous pesticides are banned in many countries, they are still being produced and exported, and finding their way to many other countries. Many of these are amongst the "dirty dozen" pesticides—in reality 18 pesticides including chlordane, parathion and lindane that were the subject of a decade long campaign by the Pesticide Action Network.

During 1995 and 1996 for example, the U.S. exported highly toxic pesticides including chlordane, heptachlor, parathion and lindane, to countries which had banned them. Chlordane was exported to Brazil, Singapore and the Netherlands; heptachlor to Brazil and the Netherlands; aldicarb (an "extremely hazardous" pesticide) to Argentina and paraquat (another highly toxic pesticide) to the Dominican Republic. Other hazardous chemicals exported include pentachlorophenol to Thailand and EDB to Belgium.

According to Greenpeace, India, which has emerged as a major centre of pesticide production in Asia (the other being China) exports hazardous pesticides including aldrin, chlordane, heptachlor, DDT and BHC to several countries, "including countries where their use has long since been banned".

"Reports indicate that clandestine manufacturing of several POP (persistent organic pollutant) pesticides may be contributing to illegal exports to Bangladesh and Nepal. As far as many Bangladeshi and Nepali activists are concerned, India is to South Asia what the U.S is to the world—a "toxic imperialist".

For instance, during 1997 India exported DDT to Bangladesh, Japan, Nepal, New Zealand, Sri Lanka, Switzerland and United Arab Emirates, and aldrin to 20 other countries including Australia, the Netherlands and the U.S. "However, officials from the Netherlands and Australia report that their records do not reflect these findings."

Despite aldrin's registration being withdrawn in 1996, Greenpeace's research found that aldrin formulations were being sold in shops in New Delhi. A shopkeeper said several manufacturers continued to supply aldrin as "this is the best for killing termites..., it is poisonous only if you drink it".

Source: "Toxic Legacies; Poisoned Futures –Persistent Organic Pollutants in Asia", by Von Hernandez and Nityanand Jayaraman, Greenpeace International, Amsterdam, 1998; and Global Pesticide Campaigner, Volume 9, Number 1, PAN North America, April 1999.

in order to flush out these toxins. Many women are working with pesticides that are so dangerous they cannot be used safely under any conditions of agricultural practice. Even those who do not spray are exposed to pesticides through agricultural activities involving contact with heavily sprayed crops.

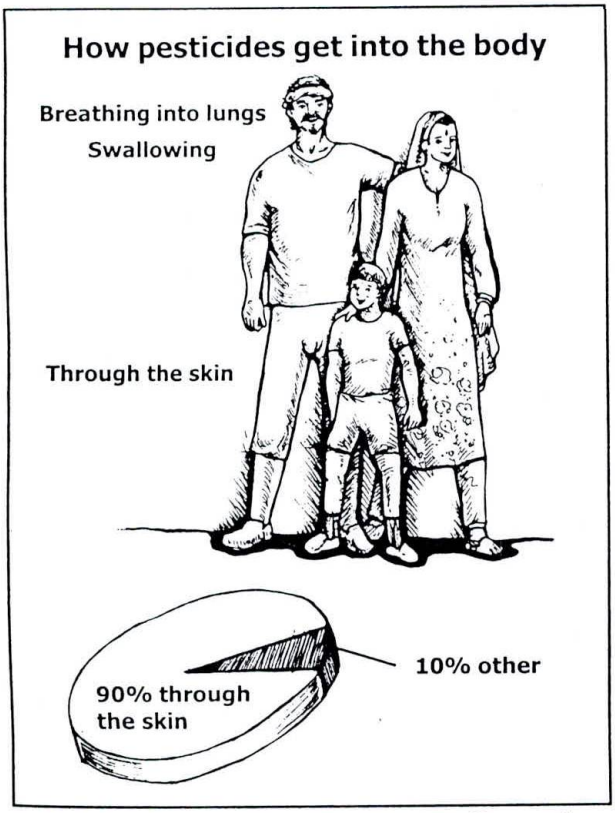


Illustration by Allan Woong, based on illustrations in 'Harvest of Sorrow- Farm Workers and Pesticides', Part I, by Dr. Marion Moses.

The purpose of this article is to briefly summarize the human health effects of pesticides. The discussion is in two parts:

1. The three major factors contributing to the impact of pesticides on human beings.

2. The three major ways that pesticides affect human health.

A special effort has been made to highlight particular concerns women and children face from exposure.

Factors Contributing to the Impact of Pesticides on Human Beings

There are three major factors in the impact of pesticides on human beings - how hazardous or poisonous they are, how they get into the body, and how long they stay there.

1. How Hazardous or Poisonous a Pesticide Is

The U.S. Environmental Protection Agency (EPA) and the World Health Organization (WHO) classify each pesticide into one of four categories; depending on how much it takes for the pesticide to kill a laboratory rat or mouse. The less it takes to kill the animal the more toxic it is. The most dangerous pesticides are in EPA Category I, and WHO Category IA and IB. These categories do

Table 1
Highly Toxic Pesticides
EPA Category I - WHO Category IA and IB
LD_{50} or MLD[1] (in milligrams/kilogram of body weight in rats)

Pesticide (brand name)	Use[2]	LD_{50} MLD[1]	Pesticide (brand name)	Use[2]	LD_{50} MLD[1]
Acrolein (Magnacide H)	H	29	Isofenphos (Oftanol)	I	20
Aldicarb (Temik)	I	1	Isolane	I	11
Azinphos-ethyl (Gusathion A,)	I	12	Mephosfolan (Cytrolane)	I	8.9
Azinphos-methyl (Guthion, Gusathion)	I	4	Mecarbam (Afos)	I	36
Bomyl	I	31	MEMA (Organic Mercury Compound)	Fn	25
Calcium cyanide	Fm	10	MEMC (Organic Mercury Compound)	Fn	22
Carbofuran (Furadan)	I	15-26	Methamidophos (Monitor, Tamaron)	I	20
Chloethocarb (Lance)	I	35.4	Methidathion (Supracide)	I	44
Chlormephos (Dotan)	I	7	Methiocarb (Mesurol)	I	20
Cycloheximide	Fn/PGR	2	Methomyl (Lannate, Nudrin)	I	17
Demeton (Systox)	I	2.5-6	Methyl parathion (Folidol-M)	I	20
Demeton methyl (Metasystox)	I	30	Mevinphos (Phosdrin)	I	3
Dieldrin	I	37	Mexacarabate (Zectran)	I	24
Dimefox (Hanane)	I	5	Monocrotophos (Azodrin, Nuvacron)	I	8-23
Dinitro-ortho-cresol (DNOC)	Fn/H/I	20	Omethoate (Folimat)	I	25
Dintirophenol (DNP)	I/Fn	30	Oxamyl (Vydate)	I	5.4
Dinoseb (DNBP)	H	40	Oxydemeton methyl (Metasystox-R)	I	30
Dioxathion (Delnav)	I	45	Oxydifulfuton (DiSyston S)	I	3.5
Disulfuton (DiSyston)	I	4	Parathion (Ethyl parathion, Folidol)	I	2
Endrin	I	7-15	Phorate (Thimet)	I	2-4
Ethion	I	21	Prothoate	I	8
Fenamiphos (Nemacur)	N	5	Schradan	I	9
Fensulfothion (Danasit)	I	5	Sodium arsenite (Pamol)	Fn/H/I	10
Fluenethyl (Lambrol)	I	3-8	Sodium cyanide	Fm	6.4
Fonofos (Dyfonate)	I	8-17	Sulfotepp (Bladafume)	I	10
Formetanate HCl (Carzol)	I	20	Terbuphos (Counter)	I	1.3
Fumitoxin (Phostoxin)	Fm	0.3	Thallium sulfate	R	16
Isazofos (Triumph)	I	40	Zinc phosphide	Fm	45.7

1. Lethal Dose 50, Median Lethal Dose - the lower this number the more toxic the pesticide.
2. I = insecticide, Fm = fumigant, Fn = fungicide, H = herbicide, N = nematicide. R = rodenticide, PGR = plant growth regulator.
Compiled by Dr. Marion Moses, Pesticide Education Center, San Francisco CA., 1999.

not include long term effects. ***(See Table 1 for a list of the most dangerous pesticides).***

2. How Pesticides Get Into the Body

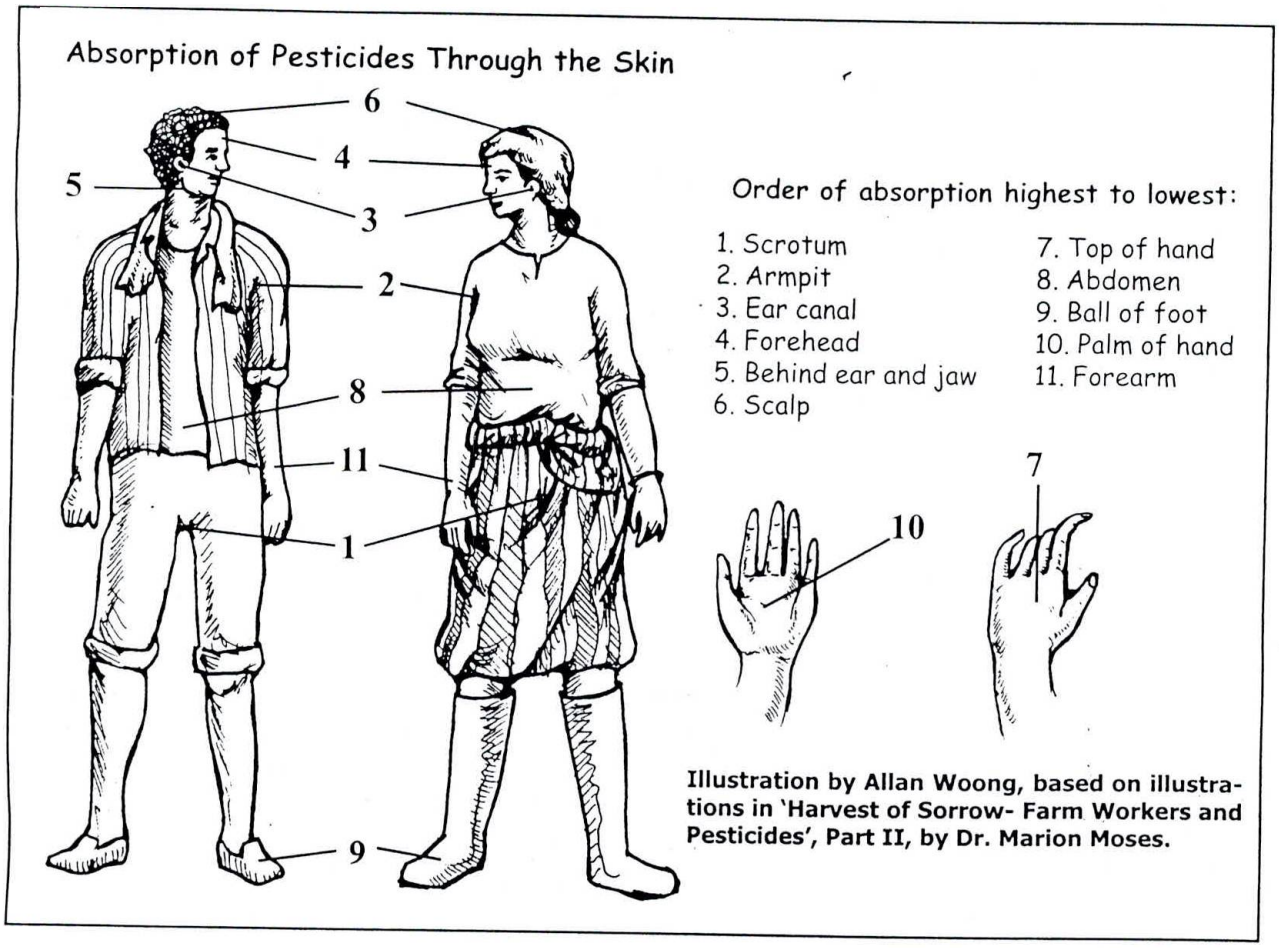
There are four ways that pesticides get into the body - by breathing them in, by swallowing them, through the skin and through the eyes in cases of splashes or spills. Most workers think that breathing in the vapors is the major way that pesticides get into the body. This is not so. The major route of pesticide absorption into the body is through the skin. Some parts of the skin however absorb pesticides more easily than others. The genital area is an area of high absorption, as is the face and neck, followed by the back of the hand, and the armpits and lower forearm. If the skin is damp or wet, or if there is a cut or rash or even minor irritation of the skin, pesticides will go through the skin faster and in larger amounts.

Children will absorb more pesticides than an adult at the same level of exposure. This is because they have a lot more skin surface for their size than adults, and also take in more breaths per minute. ***(See Box: Infants and Children Face Greater Risks! on page 14).***

Women have thinner skin than men and may likewise absorb more under similar levels of exposure. If a woman is pregnant, once pesticides get into the blood stream they can cross the placenta and affect the developing foetus.

3. How Long Pesticides Stay in the Body

A lot of the older pesticides such as DDT, dieldrin, lindane, heptachlor, and chlordane break



down very slowly. Children do not handle toxic chemicals in their bodies as well as adults. This is because their liver enzymes and their immune systems are less mature. Women also may have less efficient detoxifying mechanisms, especially during pregnancy and lactation.

The DDT type of pesticides are also known as persistent organic pollutants, meaning that they are persistent in the environment and resist breakdown by natural processes for long periods of time. Because they are fat-soluble and resist breakdown, these chemicals are stored in fatty tissues and can stay in the body for many years. Since women have a higher percentage of body fat than men, they store more pesticides in their body. Human breast milk is also high in fat and pesticides have been found in human milk in several countries.

Most of the pesticides in use today do not stay in the body for more than two or three days. They are eliminated from the body through the urine. This is why it is very important for workers exposed to pesticides to drink lots of water. Women especially must drink lots of water since their renal function compared to men, is slightly less efficient, especially during pregnancy.

The Major Ways Pesticides Affect Human Health

Pesticides affect human health in three major ways – causing immediate health effects, causing long term effects, and worsening pre-existing conditions.

1. Immediate Effects

Reactions to pesticides that occur within a very short time after exposure are called acute effects. They can appear within minutes or hours, sometimes days of exposure. The most common acute effects are irritation of the eyes, nose and throat, such as tearing, stinging, burning and coughs. Skin rashes and itching are also common. Nose bleeds are less common. These local effects are due to direct contact with the pesticide.

Some pesticides can cause allergic dermatitis. Plants such as poison oak, poison ivy and many others that workers are exposed to can also cause allergic dermatitis. It may be difficult to find out whether it is a pesticide or not without doing special skin tests. Pesticides reported to cause allergic dermatitis include anilizine, benomyl, captan, chlorothalonil, dazomet, dichlorvos, malathion, maneb, naled, and PCNB.

After pesticides go through the skin they get into the blood stream and go throughout the body.

Table 2
Pesticide Chemicals Classified by US EPA as Known, Probable or Possible Human Carcinogens

Group A - Known Human Carcinogens

Arsenic, inorganic
Chromium VI
Ethylene Oxide Group I

Group B1 - Probable Human Carcinogens
(with limited human evidence)

Acrylonitrile
Cadmium
Creosote
Ethylene Oxide
Formaldehyde

Group B2 - Probable Human Carcinogens*
(with sufficient evidence in animals and inadequate or no evidence in humans)

Acetochlor
Acifluorfen, sodium salt
Amitrole
Cacodylic Acid
Captafol
Captan
Chlordimeform
Chloroaniline
Cyproconazole
Daminozide (Alar)
1,2-Dichloropropene (Telone)
1,1-Dimethyl hydrazine (UDMH)
Dipropyl isocinchomeronate (MGK 326)
Fenoxycarb
Folpet
Furmecyclox
Haloxyfop-methyl
Lactofen
Mancozeb
Maneb
Metam Sodium
Orthophenylphenol
Oxythioquinox
Procymidone
Pronamide
Propargite
Propoxur (Baygon)
Propylene Oxide
Terrazole
Thiodicarb
Triphenyltin hydroxide

Group B2 - Probable Human Carcinogens**
(with sufficient evidence in animals and inadequate or no evidence in humans)

Acetaldehyde
Aramite
Azobenzene
Bis(chloroethyl) ether
Carbon Tetrachloride
Chlordane
Chloroform
1,2-Dibromo-3-chloropropane (DBCP)
Dibromoethane, 1,2 (EDB) -ethylene dibromide]
Dichloro diphenyl trichloroethane (DDT)
1,2 - Dichloroethane
Dicloromethane
Dieldrin
Di(2-ethylhexyl)phthalate
Epichlorohydrin
Ethylene thiourea
Heptachlor
Heptachlor epoxide
Hexachlorobenzene
Hexachlorocyclohexane, tech.
Lindane
Methylene chloride (see dichloromethane)
Mirex
Pentachlorophenol
Perchloroethylene
Polychlorinated biphenyls (contaminants
Propiolactone
Toxaphene
Trichloroethylene
Trichlorophenol 2,4,6

Group C - Possible Human Carcinogens

Amitraz
Asulam
Atrazine
Benomyl
Bifenthrin
Bromacil
Bromoxynil
Calcium Cyanamide
Carbaryl
Clofentezine
Cyanazine
Cypermethrin
Dacthal
Dichlobenil
Dichlorvos (DDVP)
Diclofop-methyl
Dicofol
Difenoconazole
Dimethenamid (SAN 682H)
Dimethipin (Harvade)
Dimethoate
Dinoseb
Ethalfluralin
Ethofenprox
Fenbuconazole
Fipronil
Fluometuron
Fomesafen
Hexaconazole
Hexythiazox (Savey)
Hydramethylnon (Amdro)
Hydrogen cyanamide
Imazalil
Isoxaben
Linuron
2-Mercapto benzothiazole
Methidathion
Methyl 2-benzimidazole carbamate (MBC)
Metolachlor
MolinateNitrofen
Norflurazon
N-Octyl bicycloheptene dicarboximide (MGK-264)
Oryzalin
Oxadiazon
Oxadixyl
Oxyfluorfen
Paradichlorobenzene
Parathion
Pendimethalin
Pentachloronitrobenzene
Permethrin
Phosmet
Phosphamidon
Piperonyl butoxide
Prochloraz
Prodiamine
Propazine
Propiconazole
4-Pyridazine carboxylic acid, 2-(4-chlorophenyl)-3-ethyl-2,5-dihydro-5-oxo-,potassium salt (MON 21200)-post FQPA
Pyrithiobac-sodium
Simazine
Tebuconazole
Terbutryn
2-(Thiocyanomethylthio) benzothiazole (TCMB)
Triadimefon
Triadimenol
Triallate
Tribenuron methyl
Tridiphane
Trifluralin
Triflusulfuron-methyl
Uniconazole
Vinclozolin

** Classified by the Office of Pesticide Programs ** Not Classified by the Office of Pesticide Programs*
Source: U.S. Environmental Protection Agency. Pesticidal Chemicals Classified as Known, Probable or Possible Human Carcinogens. Office of Pesticide Programs. Washington, D.C. 1998.
Compiled by Dr. Marion Moses, Pesticide Education Center, San Francisco CA., 1999.

Once pesticides get into the system they can cause poisoning. Signs and symptoms of systemic poisoning include headaches, dizziness, nausea, vomiting, cramping, breathing difficulties and blurred vision. If the poisoning is severe and proper treatment is not available, death can occur. Most serious poisonings and deaths from pesticides occur in developing countries.

2. Delayed Effects

Pesticides can cause delayed or long-term effects which occur months or years after exposure. These are called chronic effects. They can result from low levels of exposure over a long period of time. They can occur even if there has never been any apparent health problems during the time of exposure to pesticides. The three major chronic effects from pesticides are cancer, neurological damage and adverse effects on the reproductive system.

CANCER: Many pesticides are known or suspected to cause cancer in laboratory animals. The U.S. EPA classifies pesticides into groups of known, probable, or possible causes of cancer in humans. (Table 2 lists the pesticides in these different categories).

There is now a large body of evidence that pesticide exposure is a risk factor for cancer in humans, especially children. Studies done in the United States, several European countries, Brazil, and China show that children whose parents are occupationally exposed to pesticides or whose parents use pesticides in and around the home are more likely to get leukemia, brain cancer, non-Hodgkin lymphoma, soft tissue sarcoma, and Wilm's tumour. There are many studies done throughout the world on farmers, pesticide sprayers and factory workers exposed to pesticides that link cancer in adults to pesticide exposures. The kinds of cancer that have been found include: non-Hodgkin lymphoma, brain cancer, leukemia, soft tissue sarcoma, pancreatic, testicular and prostate cancer among others.

NEUROLOGICAL EFFECTS: There is abundant evidence from laboratory animals that pesticides can cause permanent damage to the brain and nervous system. Low levels of exposure to neu-

rotoxic pesticides to the developing brain can potentially affect brain development in complex and subtle ways that are difficult to observe and measure.

Such potential effects include effects on memory, judgement and intelligence as well as personality, moods and behaviour. There are human studies that show permanent effects on the brain and nervous system years after apparent complete recovery from pesticide poisoning. There are many individual reports of permanent changes in behavior and personality in workers and others seriously poisoned by pesticides.

There are very few studies of highly susceptible groups such as pregnant women and children. Recent data show that endocrine disruptor pesticides can affect hormone levels at critical periods of development of the brain at very low levels of exposure that were previously thought to be not harmful. (See Table 3 for a list of pesticides which are endocrine disruptors).

Pesticide exposure can increase the risk of Parkinson's disease, especially in younger people. Pesticides may also be implicated in amyotrophic lateral sclerosis (ALS, Lou Gehrig's Disease) and other neurological diseases. The percentage of people poisoned by pesticides who develop changes in brain

Table 3
Pesticides that are Endocrine Disruptors

Alachlor	Fenchlorfos
Aldicarb	Fenitrothion
Aldrin	Fenvalerate
Amitrole	Fipronil
Atrazine	Flucythrinate
Benomyl	Heptachlor
Bifenthrin	Hexachlorobenzene
Bromoxynil	Hexachlorocyclohexane oxynil
Cadmium	Lindane
Carbaryl	Malathion
Carbofuran	Mancozeb
Chlordane	Maneb
Chlordecone (Kepone)	Mercury
Chlorpyrifos	Methomyl
lambda-Cyhalothrin	Methoxychlor
Cypermethrin	Methyl parathion
2,4-D	Metiram
DBCP	Mirex
DDE	Nabam
DDT	Nitrophen (TOK)
Deltamethrin	Ortho-phenyphenol
Dichlorvos (DDVP)	Parathion
Dicofol	Pentachlorobenzene
Dieldrin	Permethrin
Dienochlor	Picloram
Dimethoate	Pyrethrins
Dinitrophenol	Simazine
Dinoseb	2,4,5-T
Endosulfan (thiodan)	Toxaphene
Endrin	Tributyltin
Esfenvalerate	Trifluralin
Ethafluralin	Triphenyltin
	Vinclozolin
	Zineb

Source: Based on data found in U.S. EPA (Environmental Protection Agency) Fact Sheets, RED (Registration Eligibility Documents), and CalEPA (California Environmental Protection Agency) Toxicology Summaries of selected pesticides. Compiled by Dr. Marion Moses, Pesticide Education Center, San Francisco CA., 1999.

DBCP and the Banana Workers

In 1997, four chemical corporations that produced dibromochloropropane or DBCP—Amvac, Dow, Occidental and Shell—reached an out-of-court settlement of over US$45 million dollars with thousands of banana workers from 11 countries. More than 6,000 of the claimants were Philippine farmers who worked in the banana plantations in Mindanao. The rest of the claimants came from Costa Rica, Honduras, Guatemala, El Salvador, Nicaragua and Ivory Coast.

The workers' lawsuits had demanded compensation for permanent sterility linked to DBCP exposure while they were working on the banana plantations. DBCP, an extremely toxic nematicide with severe acute and chronic health effects, is one of the 'Dirty Dozen' targeted by the Pesticide Action Network (PAN) for elimination. The first known human sterility cases linked to DBCP were identified in California in 1977. The companies knew that the product caused male sterility in rats as early as in the 1960's, but concealed this information. U.S. exports of DBCP never the less continued after the California cases came to light; after the fumigant was banned in the U.S. in 1979.

In the Philippines, DBCP was used in the 1970s and 80s. Tests conducted showed that the farmers were not adequately warned, or were not warned at all of the harmful effects of DBCP. Aside from sterility, the affected farmers also complained of impotence and cancers.

According to lawyers representing the banana workers made sterile by DBCP use in the 1970s and 80s, the vast majority of the 26,000 claimants had accepted the deal with the chemical companies. However, organizations representing male victims state that no amount of money could compensate for the suffering caused by the indiscriminate use of DBCP on banana plantations for 15 years. Not surprisingly, some had not welcomed the offer. The payments the individual workers would receive after deducting costs would be minimal. Although legal action is still continuing against banana multinational companies such as Chiquita, Dole, Del Monte and Standard Fruit, there are fears that these companies will also settle out of court for lesser amounts.

Source: Global Pesticide Campaigner, Vol. 8 No. 1, March 1998; and Philippine Daily Inquirer, June 25, 1997.

Infants and Children Face Greater Risks!

Infants and children face greater risk from pesticides and other environmental toxins because they have greater exposure, and less ability to get rid of toxic chemicals from their bodies.

Greater Exposure: Infants and children absorb more into their bodies than adults. The major reasons for this are:

1). They have much more skin surface for their size.
2). They take in more breaths per minute.
3). They eat and drink much more for their weight.
4). They are much more likely to come in contract with contaminated surfaces and objects.

The "job" of children is to explore. Their crawling, toddling, play and other activities put them in direct contact with contaminated soil, floors, furniture, toys, and carpets. They put everything in their mouths. They often wear less clothing therefore have more exposed skin surface. Children living on farms or near agricultural areas risk even greater exposures from drift and contamination of air, soil, food, and water by chemical pesticides.

Less Ability to Get Rid of Chemicals: Once pesticides get into the bodies of Infants and children, they are more vulnerable to toxic effects. The major reasons for this are:

1). Infants and children have less mature mechanisms in their body to break down chemicals into less harmful substances.
2). Infants and children have less mature mechanisms in their bodies to get rid of toxic chemicals from their bodies.
3). Infants and children have less mature immune systems to protect them from toxic chemicals.
4). Infants and children are growing and developing and at a rapid rate putting many body cells and tissues at risk – especially the brain and nervous system, and the blood and immune system.

This puts children at greater risk of cancer and other chronic diseases.

Brain Cancer: Studies done in the United States, Canada, France and Norway show that children whose parents are farmers or who live on farms have a three to seven fold increased risk for brain cancer. Two United States studies found that pesticide use in the home increased the risk of brain cancer in children six to eleven fold.

Leukemia: Studies done in the United States, Canada, and China show that children whose parents work with pesticides on farms have a two to eleven fold increased risk for leukemia. Studies done in the United States and German found that pesticide use in the home increased the risk of leukemia in three to nine-fold. Other studies also found children to be at increased risk for non-Hodgkin lymphoma, Wilm's tumor, and soft tissue sarcoma.

Source: Dr. Marion Moses, Cancer in Children and Exposure to Pesticides, Summary of Selected Studies, Pesticide Education Center, San Francisco CA. May 5, 1999.

function is however not known.

REPRODUCTIVE EFFECTS: Many widely used pesticides are known to cause birth defects, sterility and foetal death in laboratory animals (see Table 4). Occupational exposure to the pesticide DBCP (dibromochloropropane) is a proven cause of sterility in human males. ***(See Box: DBCP and the Banana Workers).***

Human studies have found increases in spontaneous abortion, stillbirth, infertility, and birth defects in exposed workers. The highest risk is in women who work and live on farms or in agricultural areas or who have come into direct contact with pesticides during pregnancy.

Studies often do not find an increase in birth defects associated with pesticide exposure. This may be due to direct toxicity to the embryo and foetus while still in the womb, leading to an early spontaneous abortion.

3. Effects on Existing Conditions

People with asthma and allergies, especially children can react to low levels of pesticides that do not affect those without them. The pesticides most likely to percipitate or aggravate asthma are the pyrethrins and pyrethroid classes of pesticides, and the organophosphates and methyl carbamates. However, any pesticide or inert ingredient can still be a potential problem. The only effective treatment is to avoid exposure to the pesticide.

Pesticides can also cause irregular heart rhythms, and people with heart disease may have a worsening of their condition when exposed.

Pesticide exposure can also weaken the immune system. The most susceptible to such effects are children, pregnant women, those with chronic medical illnesses, and cancer survivors.

Countering the Toxic Legacy

Pesticides are used in ways that maximize opportunities for human exposure and environmental contamination. Most regulations are not strong enough to protect workers from the adverse health effects of pesticides, especially women and children. Many workers are poisoned even when all rules and regulations have been followed.

Just because a pesticide is used according to label directions, it does not mean that potential harmful effects are not occurring. The effects may not show up until many years later. There is often a false sense of security if there is no apparent immediate illness or acute effects.

One of the most important concerns not addressed by current pesticide laws and regulations is the effect of multiple exposure. All workers are exposed to many different pesticides in the course of their working life. The combination of low level exposures to many different pesticides add up to a large toxic burden, especially for the embryo and foetus developing inside the womb. The possible synergistic effects of these combined and mixed exposures have not been studied. The laws that regulate pesticides do not require these kinds of tests to be done. The younger the individual the greater the risk of adverse effects from toxic exposures.

Some pesticides are so toxic that they cannot be used safely under any conditions of agricultural practice. Once we release these toxic chemicals we cannot take them back. The only way to eliminate the health risks from toxic pesticides is to eliminate the exposures; beginning with the most highly toxic pesticides and those that cause cancers and birth defects.

Future generations will no doubt look back on the twentieth century use of toxic pesticides in food production as one of the more bizarre practices of their ancestors. The public health community must work together with workers and their advocates to promote safer alternatives to toxic pesticides that do not threaten the health of people and the environment.

Table 4
Pesticides That Are Teratogenic (cause Structural Birth Defects) in Laboratory Animals

Acrolein	Fenarimol
Abarmectin	Fenoxaprop ethyl
Bacquacil	Fluazifop-butyl
Bitertanol	Folpet
Benazolin-ethyl	Hexachlorobenzene
Benomyl	Kinoprene
Bentazon	Maleic hydrazide
Bromoxynil	Mancozeb
Cacodylic acid	Methyl parathion
Captafol	Methoprene
Captan	Mirex
Carbaryl (Sevin)	Fenamiphos (Nemacur)
Chloramben	Nitrofen (TOK)
Chlordimeform	Ortho-phenylphenol
Chlorpropham	Paclobutrazol
Copper sulfate	PCNB
Cyanazine	Phosmet
Cycloheximide	Picloram
Cyromazine	Propargite (Omite)
2,4-D	Sodium arsenate
Dichlobenil	Sodium arsenite
Dichlorophene	Sodium omadine
DMF	2,4,5-T
2,4-DP (Dichlorprop)	Terrazole
Dinocap (Karathane)	Triadimefon
Dinoseb	Tributyltin oxide
Diquat	Trichlorfon
Endosulfan	Trifluralin
Endothall	Triphenyltin fluoride
Ethion	Triphenyltin acetate
2-Ethyl 1,3-hexanediol	Triphenyltin hydroxide
Ethylene dichloride	Vinyzene
	Warfarin

Sources: U.S. Environmental Protection Agency. Teratogenic Pesticides (as of June 1988), Office of Pesticide Programs, Washington, D.C. 1998. California Environmental Protection Agency, 'Chemicals Known to the State to Cause Reproductive Toxicity', Office of Environmental Health Hazard Assessment, Sacramento, CA. December 26, 1997. Compiled by Dr. Marion Moses, Pesticide Education Center, San Francisco CA., 1999

Dr. Marion Moses is President of the Pesticide Education Center (PEC) in San Francisco, California. A physician, certified in Public Health and Preventive Medicine (specializing in Environmental and Occupational Medicine), Dr. Moses interest in pesticides began in the 1960s with her work with the United Farm Workers of America, affiliated to the American Federation of Labour-Congress of Industrial Organizations (AFL-CIO), in one of the largest agricultural areas in the world. She has many years experience investigating and documenting pesticide related illnesses in farm workers both short and long term. She has published widely on the adverse health effects of pesticide in humans, and is a consulting editor for the American Journal of Industrial Medicine, and the Archives of Environmental Health.

Endocrine Disruption: New Threats From Old Chemicals

by Dr. Michael Smolen

Through a series of accidental discoveries, researchers stumbled on the fact that some widespread, man-made chemicals, called "endocrine disruptors", can interfere with the body's own hormones and jeopardize health. In the past five years, the scientific investigation of this problem has intensified and provided steadily growing evidence linking these synthetic endocrine-disrupting compounds to impaired health in wildlife and people. The exploration is ongoing and far from complete...

"Chemicals that compromise life – A call to action", World Wildlife Fund.

Reports of disturbing global trends in human health are appearing regularly in government reports, scientific papers, and even the news media. During the past few decades, increases have been recorded in the incidence of prostate, testicular and breast cancers [1], developmental problems such as hypospadias and undescended testicles [2] – forms of genital malformations, and reported global declines in sperm quality and quantity[3,4]. Scientists now have an explanation that could account for many of these problems: *disruptions to the developing endocrine system.*

Discussion of this hypothesis has previously been confined to scientific literature, and only in the last few years has it seeped into the policy and public arenas. This visibility has been greatly increased with the publication of a book, "Our Stolen Future: Are we threatening our fertility, intelligence and survival?", written by Theo Colborn, Dianne Dumanoski, and John Peterson Myers. This book presents the scientific evidence supporting concern for the endocrine-disrupting effects of some man-made chemicals. Written specifically for the general public, it has already sparked much debate.

The Endocrine System and Endocrine Disruption

The endocrine system is the body's chemical "messenger system" of hormones and other special messengers, which help communication be-

What Are Hormones?

Hormones are naturally-occurring chemicals that circulate at very low levels in the blood stream of all vertebrate animals including reptiles, amphibians, fish, birds and mammals. (Vertebrates are animals with a backbone.) In all vertebrate species, hormones act as chemical messengers and as switches, turning on and off bodily systems that control growth, development, learning and behaviour. Hormones start affecting every animal shortly after it begins life as a fertilized egg. Hormones control growth and development prior to birth or hatching, and hormones continue to influence behaviour throughout life. Hormones tell bears when to hibernate, tell salmon when to return to their spawning grounds, and cause women to menstruate every 28 days or so. Hormones profoundly affect the nervous system, the reproductive system, and the immune system. Naturally-occurring hormones are also implicated in some forms of cancer, such as female breast cancer which is widely believed to be linked to a woman's lifetime exposure to estradiol (estrogen), the main female sex hormone.

Source: 'Hormonally Active Agents In The Environment', Ernst Knobil and others, Washington, D.C.: National Academy Press, July 1999. Page 197.

Diagram 1: Important glands, organs, and tissues sending or receiving hormonal messages in the human body

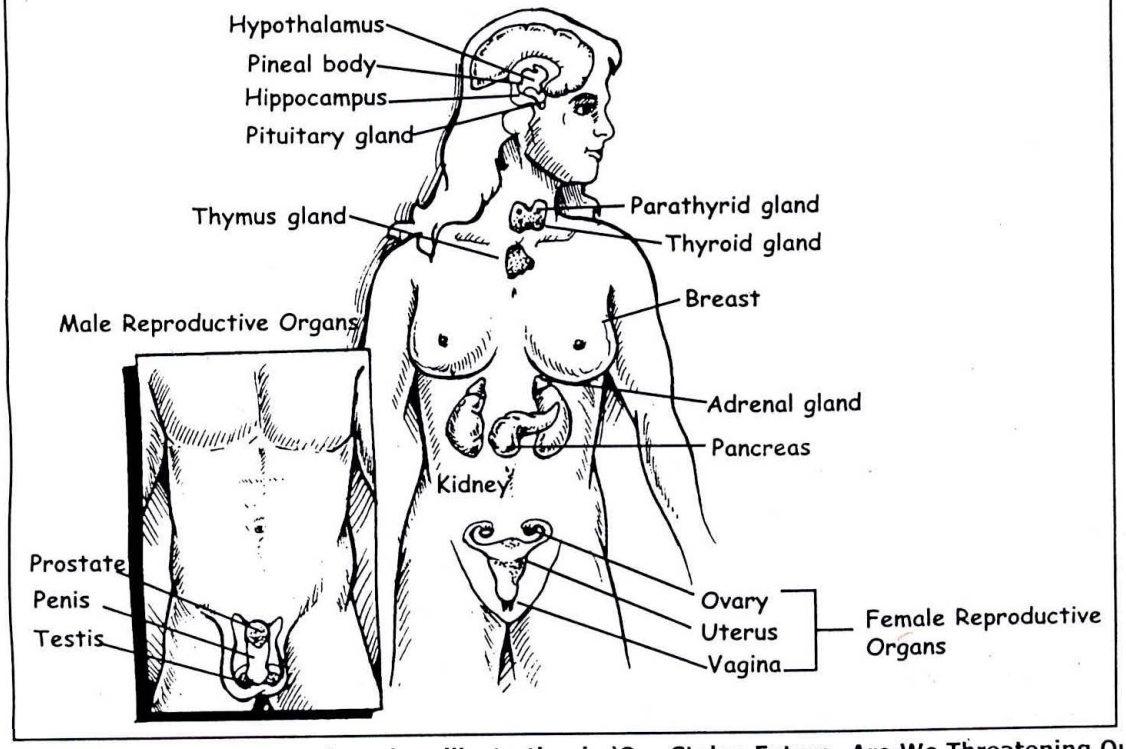


Illustration by Allan Woong based on illustration in 'Our Stolen Future- Are We Threatening Our Fertility, Intelligence, and Survival?-A Scientific Detective Story, Dutton U.S., 1996.

tween the various parts of the body. The system involves a variety of organs, called endocrine glands (the thyroid, thymus, pituitary, adrenal, the testicles, ovaries, etc) that release the hormones to be carried in the bloodstream to specific target sites (cells) in the body. ***(See Box: What are Hormones?)***

Latching on to unique "receptors" at the target site, the hormones signal and govern various processes and functions such as growth and development (including brain development), metabolism, reproduction, immune system, etc. *(See Diagrams 1 and 2).*

Distantly related groups of living things like birds, mammals and humans share almost identical hormone and receptor systems, and similar biological responses. Disruption of this finely balanced endocrine system occurs when biologically active foreign chemicals interfere with the body's messenger system of hormones, and this can lead to developmental, reproductive, behavioural, immunological (i.e. effecting the immune system) and physiological changes.

However, chemicals have always been assessed for safety based only on whether they cause cancer, poison people outright or produce obvious developmental abnormalities[5]. Toxicologists use high doses of chemicals to assess their effects and, when no effects appear, the chemicals are considered safe until proven otherwise. Examples of the effects of foreign chemicals on the endocrine system have always been portrayed as novelties or rarities of nature.

Thus when fleas, which were living on rabbits,

Diagram 2: The Lock-and-Key model of hormone-receptor interaction necessary for a hormone to trigger biochemical activity in a cell

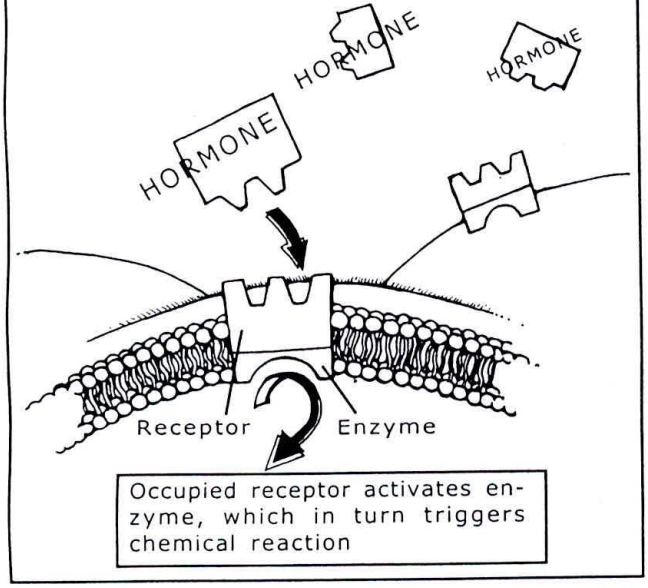


Illustration by Allan Woong based on illustration from 'Generations at Risk: How Environmental Toxicants May Affect Reproductive Health in California', A Report by Physicians for Social Responsibility (L.A. and San Francisco), and The California Public Interest Research Group Charitable Trust, 1999.

DES and Vaginal Cancer

From 1950 – 1971 diethylstilbestrol (DES), a synthetic estrogen with a chemical structure considerably different from naturally-occurring estrogen, was used in an attempt to prevent spontaneous abortions in women. An estimated 5-10 million Americans were exposed to DES during pregnancy (DES mothers) or in the uterus (DES daughters or sons). [1]

No harmful effects of DES exposure were suspected until 1970 when a rare form of vaginal cancer was reported in six young women, ages 14 – 21, who had been exposed to DES in the uterus. [2] Previously, this disease had occurred almost exclusively in older women, but it is now know to be caused in younger women by exposure of the developing foetus to DES. The risk for developing vaginal cancer from birth to age 34 is estimated to be 1 in 1000 to 1 in 10,000 for women exposed in the uterus – accounting for thousands of cases in the U.S. alone.

Later studies demonstrated that DES daughters often have abnormalities of their reproductive organs, reduced fertility, and unfavourable pregnancy outcomes including ectopic pregnancies, miscarriages, and premature birth, as well as immune system disorders. DES sons are more likely to have small and undescended testicles, abnormal semen, and hypospadias.[3] DES mothers have a breast cancer risk about 35 per cent greater than those not exposed.[4] Animal studies in mice and monkeys show that prenatal DES exposure may result in masculinization of parts of the female brain and feminization in males.[5] Several studies in humans suggests similar results.[6]

Some DES daughters and sons are now in their mid-20's. Many do not know that they were exposed in the uterus. Their health status require careful attention. As yet there is no definite evidence for adverse health effects in the offspring of those who themselves were exposed to DES in the uterus (DES grandchildren). However, since many are still young, it is too early to draw final conclusions and the issue is not resolved.

DES is an example of an estrogenic chemical which causes reproductive and developmental abnormalites, immune system malfunction, and cancer in some people exposed as foetuses.

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6. *Reinisch J.M., Zienba-Davis M., Sanders S.A., Hormonal Contributions to Sexually Dimorphic Behavior in Humans, Psychoneuroendocrinology 16(1-3): 213-278, 1991.*

Source: Generations at Risk: How Environmental Toxicants May Affect Reproductive Health in California, A Report by Physicians for Social Responsibility (L.A. and San Francisco), and The California Public Interest Research Group Charitable Trust, 1999.

For more information visit the following websites: http://www.igc.apc.org/psr/index.html or http://www.pirg.org/pirg

were discovered to use the hormones in rabbits to signal their own reproductive cycle, that was an amazing fact of natural history. When DES (diethylstilbestrol), an estrogen-like synthetic molecule given to pregnant women to guard against miscarriages, was found to alter the development of their offspring[6], that was considered an unfortunate side-effect of a drug. ***(See Box: DES and Vaginal Cancer).*** When sheep and cows developed reproductive problems after eating plants rich in plant estrogens, that was a problem in animal husbandry.

But disruptions to the endocrine systems are not isolated or rare events. Today there is concern that animals and people are experiencing disruptions to their endocrine systems, leading to the changes mentioned earlier. ***(See Box: Wildlife Health Effects).*** Although a number of natural chemicals in plants (i.e. phytoestrogens like genistein, daidzein, and coumestrol) can also interfere with the endocrine system in vertebrates, the main concern now is with man-made chemicals which our bodies had never before encountered. And this concern is not limited only to persistent chemicals that build up to high concentrations in the body but also to many short-lived ones which, while they are in the body, can disrupt the endocrine system.

So in 1992, a group of scientists with expertise in varied fields (from anthropology, endocrinology, medicine, immunology reproductive physiology, and histopathology) met to explore the potential for endocrine system disruption in humans and wildlife. They concluded that: "A large number of man-made chemicals that have been released into the environment, as well as a few natural ones, have the potential to disrupt the endocrine system of animals, including humans"[7]. These chemicals include a variety of pesticides and industrial chemicals. Published research convinced the scientists that wildlife populations have been affected, examples of which included:

- "thyroid dysfunction in birds and fish;
- decreased fertility in birds, fish, shellfish, and mammals;

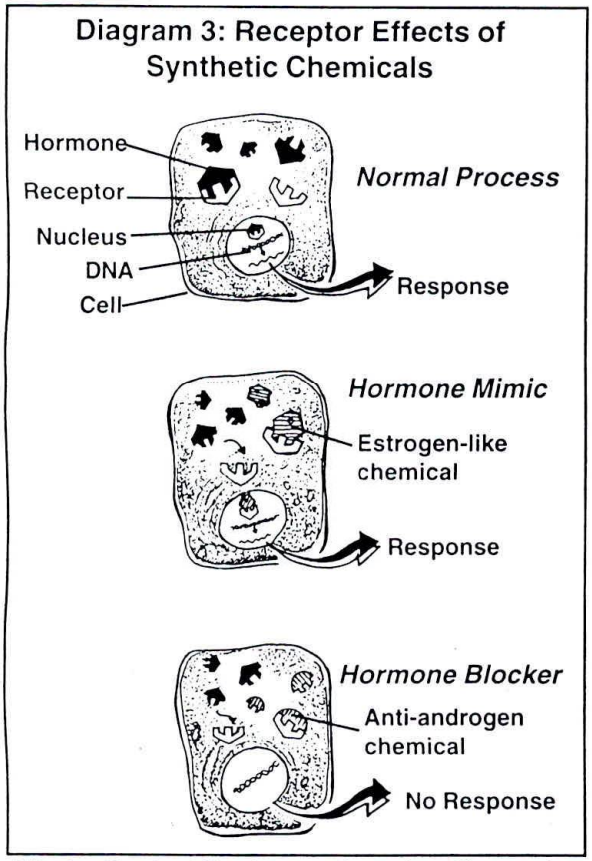


Illustration by Allan Woong based on illustration in 'Our Stolen Future- Are We Threatening Our Fertility, Intelligence, and Survival?-A Scientific Detective Story, Dutton U.S., 1996.

- gross birth deformities in birds, fish, and turtles;
- metabolic abnormalities in birds, fish, and mammals;
- behavioural abnormalities in birds;
- demasculinization and feminization of male fish, birds, and mammals;
- defeminization and masculinization of female fish and birds;
- and compromised immune systems in birds and mammals"[7].

Man-made chemicals can interfere with the endocrine system in a number of ways. The hormones or messengers in the body have a complex feedback system, which closely controls their release and persistence in the body. Some man-made chemicals can mimic the natural hormone and activate biological processes (some can even super-activate the processes). Others can merely bind to and block the receptors so that the natural system can no longer be turned on. Yet others may react directly or indirectly with natural hormones or alter natural patterns of hormone synthesis. *(See Diagram 3).*

Problems for the Unborn

Reports of endocrine system disruptions involve development of a baby can thus have pronounced

Wildlife Health Effects

A variety of invertebrates, reptiles, birds, fish and mammals have been adversely affected by Endocrine Disruptors (EDs). The following examples illustrate the diversity of health effects:

- Various types of snails exposed to environmental levels of tributyl-tin, an anti-fouling additive used in marine paint on ships, develop a condition called imposex in which affected female snails have irreversibly superimposed male sex characteristics.
- Hermaphroditic (having both the male and female sex) fish are found in rivers below sewage treatment plants in Great Britian. Vitellogenin, a protein normally synthesized by female fish in response to estrogen, is utilized as a yolk protein to nourish the developing fish. Male fish have vitellogenin levels similar to gravid females in some rivers. Laboratory tests show that nonylphenol, an alkylphenol used in detergents and surfactants and found in effluent, behaves as an estrogen mimic and induces vitellogenin formation and testicular inhibition in male trout. However, it is not entirely clear which chemical or combination of chemicals in the sewage effluent mixture is responsible for the observations in river fish. Some investigators believe that estrogens from the urine of women taking birth control pills also contribute.
- Alligators and red-eared turtles in Lake Apopka in Florida are demasculinized after exposure to a mixture of chemical contaminants including the pesticide, dicofol. There are no normal male turtles in Lake Apopka. All hatchlings have either normal appearing ovaries or are intersex.
- Gulls breeding in the Puget Sound and Great Lakes regions show evidence of eggshell thinning and reproductive tract abnormalities with feminization of male embryos. In some instances, populations have declined and sex-ratios are skewed. These areas are contaminated with mixtures of DDT, PCBs, and polycyclic aromatic hydrocarbons, each of which may cause the observed effects. Birds from these areas and from locations far more remote from industrial activity show elevated tissue levels of contaminants.
- Great Lakes gulls and terns, as well as some western gulls, have, within the past several decades, shown supernormal egg clutches and female-female paring. Gulls in these colonies also show excessive chick mortality, birth defects, and skewed sex ratios, with an excess of females. These effects correlate with levels of persistent organic pollutants like PCBs and DDT.
- Seal populations have markedly declined in portions of the Wadden Sea in the Netherlands. Fish from the area of decline are contaminated with higher levels of PCBs and pesticides than those from other areas. Captive seals fed fish exclusively from the contaminated areas were less able to reproduce and had altered estrogen levels compared to seals fed less contaminated fish over a two year period.

Source: Generations at Risk: How Environmental Toxicants May Affect Reproductive Health in California, A Report by Physicians for Social Responsibility (L.A. and San Francisco), and The California Public Interest Research Group Charitable Trust, 1999.

See also the following websites: http://www.igc.apc.org/psr/index.html or http://www.pirg.org/pirg

Pesticide Exposure May Impair Children's Brain Function

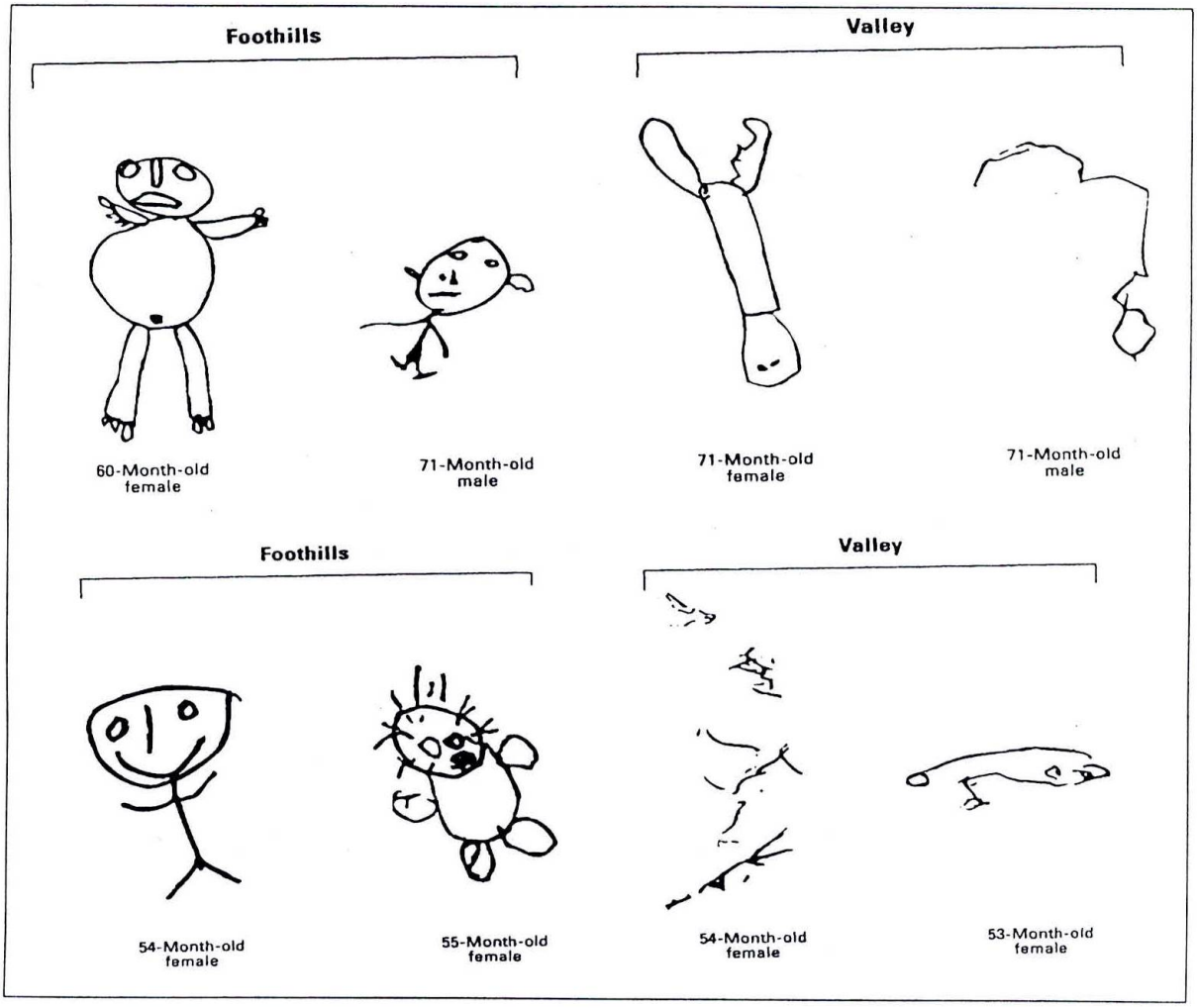
Dramatic deficits in brain function are seen in rural children with long-term exposure to pesticides compared with children not similarly exposed, according to a recent study in "Environmental Health Perspectives." The study compared two groups of four-and-five-year old children in the Yaqui Valley of Sonora, Mexico, who are very similar except in their levels of pesticide exposure. The children share a common genetic and cultural background, eat the same foods and drink the same water. The major difference was in their exposure to pesticides.

Thirty-three of those studied lived in the valley, a farming area where pesticide use was relatively intense. Farmers reported that two crops a year may be planted with up to 45 pesticide applications per crop. Organophosphates, organochlorines and pyrethroids were among the chemicals used. In addition, household insecticides were usually applied each day throughout the year. Contamination of the local population had been documented, with women's breast milk containing concentrations of lindane, heptachlor, benzene hexachloride, aldrin and endrin all above limits established by the UN Food and Agricultural Organization.

The second study group (17 children) lived in the foothills, where most families were involved in ranching and pesticide use was minimal. Foothill residents used traditional methods of intercropping for pest control in gardens and rarely used insecticides indoors. Residents stated that their only exposure to pesticides was annual government spraying of DDT to combat malaria (this programme was also carried out in the valley).

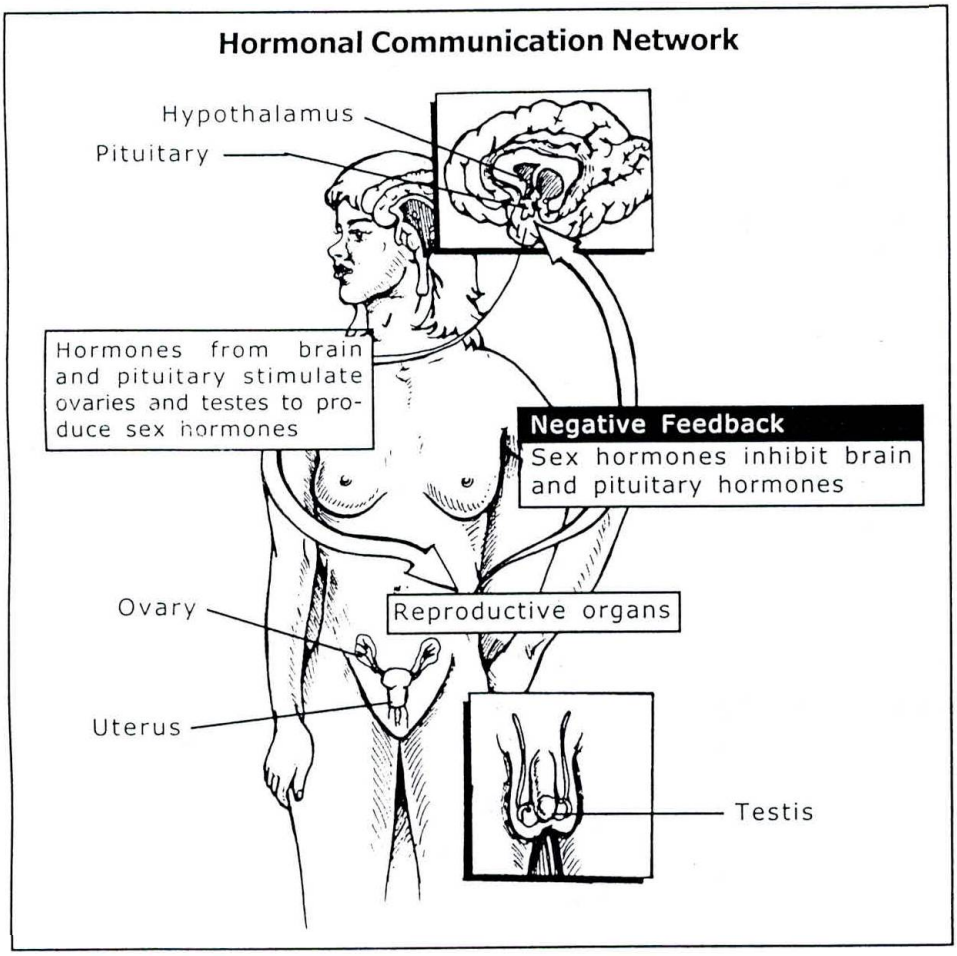
The researchers developed and used a Rapid Assessment Tool to measure the growth and development of these two groups of children. Although the groups were similar in physical growth, a comparison of their functional abilities showed some marked differences.

The valley children showed: less stamina (or physical endurance, measured by making the child jump in place for as long as possible); lower motor or hand-eye co-ordination (ability to catch a large ball from a distance) and even lower fine eye-hand co-ordination (ability to drop a raisin into a bottle cap); and poorer short-term memory.



One of the most striking differences between the two groups was the ability to draw a person. The valley children showed much less ability to draw a person than the foothill children (see drawings); even while looking at a person and drawing, the valley children "continued to draw meaningless circles". Some of the valley mothers later told the researchers about their frustration in trying to teach their children how to draw. The decreased eye-hand co-ordination and ability to draw could indicate impairment of brain function among the pesticide-exposed valley children, say the researchers.

Source: Environmental Health Perspectives, Volume 106 Number 6, June 1998; and Global Pesticide Campaigner, Pesticide Action Network (PAN) North America, September 1998.



A negative feedback loop in hypothalamic-pituitary-gonadal (HPG) hormonal communications tends to keep sex hormons at constant levels. In males, the feedback loop is always negative. In females, it fluctuates between negative and positive. Illustration by Allan Woong based on illustration from 'Generations at Risk: How environmental Toxicants May Affect Reproductive Health in California', A Report by Physicians for Social Responsibility (L.A. and San Francisco), and The California Public Interest Research Group Charitable Trust, 1999.

the more familiar thyroid, estrogen and testosterone hormones as well as less well understood developmental messengers. One such specialized developmental messenger (Mullerian inhibiting substance) is released in the developing male foetus to signal the resorption of the embryonic tissue that would otherwise produce a female reproductive system. All embryos have the potential to become either male or female, and simultaneously develop two separate kinds of tissues, one that will give rise to male and the other to female reproductive systems.

Early in life, a developmental switch is thrown (under the direction of the sex chromosomes) signalling the right set of tissues to develop the appropriate reproductive organs while tissues fated for the opposite sex are signalled to self-destruct. The switch sets in motion specific activities along a number of endocrine pathways, and the resulting chorus of messengers directs the further constuction of the anatomy, physiology and behavioural traits relevant to that sex. Disturbance to these hormonal ebbs and flows confounds development and causes potentially serious problems. For example, crossed messages signalling the development of parts of both sexes can cause "feminization" and "demasculinization" of males or "defeminization" and "masculinization" of females, the offspring acquiring an intermediate or "intersex" design compared to what was to be by genetic inheritance alone. ***(See Box: Edocrine Disruptors and Genital Birth Defects)***

Similarly, disturbance to thyroid, estrogen, testesterone and other harmone systems can cause reduced growth, birth defects, functional abnormalities, altered behaviour, reduced fertility, learning disabilities ***(See Box: Pesticide Exposure May Impair Children's Brain Function)***, lower intelligence and greater susceptibility to diseases.

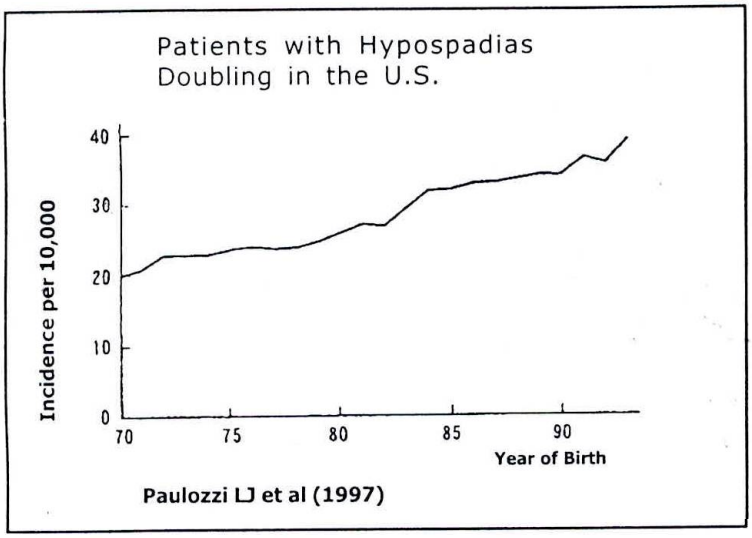
Of all these endocrine-disruption effects, the most serious arise from changes occurring during development. Endocrine disruption can occur in adults, but these typically require higher concentrations of the chemicals, and when these chemicals are removed from the system, the effects may disappear. The threat to the developing foetus is more severe in that the changes caused during this stage cannot be undone later. These effects are typically irreversible and permanent [8]. ***(See Box: Suffer the Little Children...)***

Concern for Women

This is a cause for special concern for women who use, mix or spray pesticides. Chemicals are readily adsorbed into the body and can be easily passed on through the bloodstream to the foetus or to breast tissue from where it can pass into breast milk and to suckling babies. Pesticides are designed to be biologically active in order to kill pests, and many of them have been discovered to affect the developing endocrine, reproductive, neural and immune systems. Exposures to endocrine disruptor chemicals during the critical stages of growth and

Edocrine Disruptors and Genital Birth Defects

In 1997, a group of researchers at the U.S. Centre for Disease Control and Prevention, lead by Len Paulozzi, reported that cases of male genital birth defects (known as hypospadias) among boys in the US was increasing, and had doubled between 1970 and 1993 - from 20 cases per 10,000 births in 1970 to 40 cases in 1993 (see chart below). Rising rates of hypospadias have also been reported from European countries.



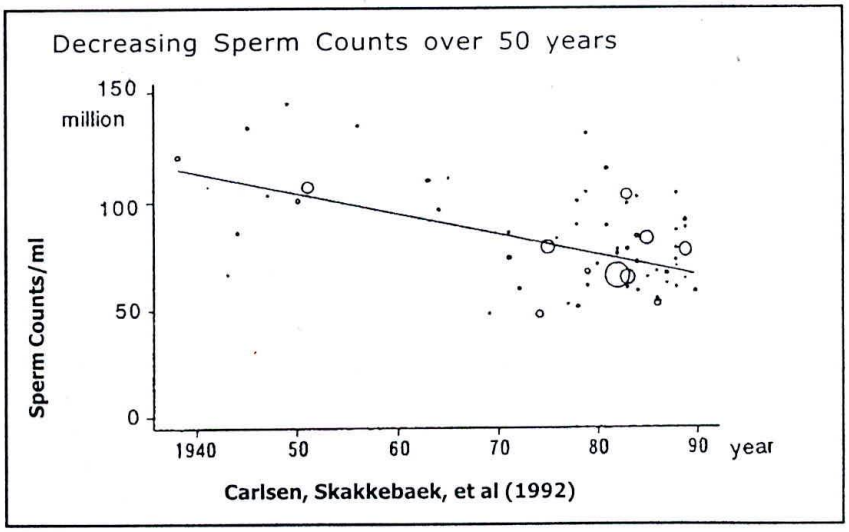
Hypospadias is a genital defect in males where the urinary opening is misplaced on the underside of the penis instead of at the end or, in some cases, located in the scrotum. This condition has been linked to the inadequate release of the male hormone testosterone during a critical period of foetal development - between the 56th and the 80th day after conception when the urogenital tract develops in the foetus. This leads to the "incomplete masculinization" of the male genitals.

"As you block the foetus' own testesterone, the foetus cannot masculinize itself, and you end up getting these various states of feminization of the foetus, of which hypospadias is a mild form", says Paulozzi. Undescended testicles, where the testes do not descend into the scrotum and are retained within the abdomen, vaginal pouches where the penis is covered with a layer of fat, cleft penis, reduced seminal vescicles, etc are among the other features of this incomplete masculinization or "feminization" of the male genitals. Also cases of such genital defects seem to be increasing according to various reports. Such defects have also been found to occur in animal studies using pesticides such as DDT (and its breakdown product DDE) and vinclozolin, a fungicide used commonly on fruits and vegetables.

Similarly, cancer of the testicles in men has been found to be increasing in several countries and the incidence of testicular cancer has been found to be higher among men with developmental defects such as hypospadias and undescended testicles. Researchers say that this indicates that the higher rates of testicular cancer have something to do with events in early life or in the womb itself. Here again, laboratory studies with animals have indicated that estrogens may have a major role in promoting testicular cancer.

Meanwhile, studies done by Frederick vom Saal at the University of Missouri, USA, have shown that mouse foetuses exposed to very small doses of estrogen-like chemicals developed enlarged prostates and the mice later had declined sperm counts.

Decline in sperm counts in men has also been found in studies of semen samples from various regions of the world. In 1992, Danish endocrinologist, Niels Skakkebaek and his colleagues analyzed various studies of semen quality (covering 15,000 men from 20 countries) published over the previous 50 years and found that the mean sperm count had declined nearly 50 percent worldwide over that period - from 130 million/mL in 1940 to 66 million/mL in 1990. This study turned out to be very controversial; while some smaller scale and localized studies of semen quality that followed found a decline in sperm count, a few others did not.



However, in 1997, Shanna Swan and her group at the California Department of Health Services, USA, reviewed and re-analyzed Skakkebaek's data for sperm count, taking into account regional variations in sperm count, and came to the conclusion that there was indeed a sharp drop in sperm count worldwide; if anything, they found, the drop could be sharper than what was estimated by Skakkebaek.

Source: K. Prabhakar Nair, varied researched references, 1999. Graphs taken from Japan Offspring Fund (JOF) information posters for their Endocrine Disruptors Campaign.

Suffer the Little Children...

Theo Colborn, a researcher at the World Wildlife Fund (WWF), has been closely following and synthesizing research on endocrine-disrupting chemicals around the world for years. She put together, for the first time the mounting evidence, collected from all over the world, for the endocrine-disrupting effects of synthetic chemicals, including pesticides and industrial chemicals, in her book, "Our Stolen Future". In this extract here, taken from her interview with "Mother Jones", a U.S. magazine, she talks about the implications of these endocrine-disrupting effects of synthetic chemicals. She says:

"We are neutering the population (as a result of the interference of some organic pollutants and industrial chemicals which act like hormones); we are making females more masculine and males more feminine.

Up until the 56th day from the day of conception, you can't tell the sex of the foetus. The tissue that is there is going to eventually produce testicles or ovaries. It takes just a slight tweak of a hormone to make it grow into a male tissue and become a testicle; a tweak in the other direction, and it will become female tissue.

What we are finding in fish and birds and even mammals now are ovotestes, or testes that have ovarian tissues in them. We have uncovered a new series of subtle effects, which probably take place during embryonic and foetal development and which have long-term effects that keep an individual from reaching his or her full development.

We are seeing an increase in hypospadias in boys. Hypospadia is a condition where the urethra doesn't come out at the end of the penis. This particular developmental process starts on day 56 in the womb and ends on day 84. Hypospadia has nothing to do with genetic pre-disposition. But what can cause this condition is dioxin and DDT. And it is not just this type of hypospadias that is increasing but also the more severe form, where the end of the urethra actually comes out of the scrotum. It is almost impossible to repair this surgically.

Hypospadias and undescended testicles – another condition that results from males not fully developing in the womb – put young men at greater risk of developing testicular cancer, which is one of the fastest-growing cancers in the world, and is occuring in younger and younger men.

Finally, males with hypospadias and undescended testicles always produce less sperm, which means they are more likely to have reproductive problems...

During embryonic and foetal development, the brain isn't developed yet, so you have got an individual that has no feedback mechanism to protect itself. The foetus is still growing new tissue, constructing its nervous system, constructing elements of its immune system and the reproductive tract. When all your organs are formed and fully functioning, it takes a lot more to blow them away.

But we are never going to be able to prove a causal relationship of anything in a human being because we can't feed chemicals to human beings and wait for them to grow up.

In the case of a developmental problem such as attention deficit hyperactivity syndrome (ADHD) in children, for example (this is in answer to the interviewer's question on ADHD—ed.), it is very difficult (to prove a causal relationship) because the syndrome is probably precipitated pre-natally or in early infancy through something that interfered with the development of the brain. And the presence of the chemical in that individual later on in life may not indicate that it was the cause.

Despite the fact that there are a lot of misdiagnosed kids, I still think ADHD is on the increase. And the evidence is almost overwhelming that these chemicals are involved."

Common Pesticides as Endocrine Disruptors

Endocrine-disrupting pesticides vary in their effects since they may involve different receptors and target cells, accumulate at different rates, and have different binding affinities. Consider, for example, vinclozolin, a commonly used fungicide (which has been shown to strongly block the receptors for the male hormone androgen when given to pregnant rats)[1]. When vinclozolin was present during critical periods of foetal development, genital malformations were common and these would affect reproduction later in life. Scientists had difficulty identifying the male offspring at birth because they had genitals that were feminized: i.e., undescended testes, vaginal pouches, reduced seminal vesicles and prostate glands and cleft phalli. The vinclozolin molecule itself is not the culprit but it is broken down in the body into two products which are endocrine disruptors. This provides a classical example of the body's natural chemical detoxification system producing more dangerous chemicals.

Another example is the old DDT. Concerns about the effects of DDT and its metabolites on the health of wildlife and humans have a long history. A variety of abnormalities seen in male sexual development have been linked to DDT. It was earlier thought that these effects (as also the well-documented eggshell thinning) were in part due to DDT's interference with estrogen receptors but recently it has been shown that the primary metabolite (breakdown product) of DDT, p,p'-DDE, blocks androgen receptors. Like vinclozolin, it also binds to the androgen receptor, blocking a switch critical for the development of normal males.[2]

Eggshell thinning is another effect seen in birds exposed to DDT. Scientists had long suspected that this was the result of estrogen-mimicking. However, recent work reveals that the effect is also instigated by DDE which acts to inhibit the production of another hormone, called prostaglandins, which are critical to calcium balance and deposition of calcium in eggshells. This is a very important example that endocrine disruption does not only occur through the receptors that hormones usually signal cells in the body. Many hormones have other specialized ways of communicating other messages that are critical for many other processes.

These are only two examples of endocrine-disrupting behaviours of commonly used pesticides.

Other Offending Pesticides

Herbicides:
2,4,-D
2,4,5,-T
Alachlor
Amitrole
Atrazine
Metribuzin
Nitrofen
Trifluralin

Fungicides:
Benomyl
Ethylene thiourea
Fenarimol
Hexachlorobenzene
Mancozeb
Maneb
Metiram-complex
Tri-butyl-lin
Zineb
Ziram

Insecticides:
beta-HCH
Carbaryl
Chlordane
Chlordecone
Dicofol
Dieldrin
Endosulfan
Heptachlor / H-epoxide
Lindane (gamma-HCH)
Malathion
Methomyl
Methoxychlor
Oxychlordane
Parathion
Synthetic pyrethroids
Transnonachlor
Toxaphene

Nematocides:
Aldicarb
DBCP

References:
1. Gray Jr, L.E., J.S. Ostby and W.R. Kelce, 1994, Developmental effects of an environmental antiandrogen: the fungicide vinclozolin alters sex differentiation of the male rat, Toxicology and Applied Pharmacology, 129: 46-52.
2. Kelce W.R., C.R. Stone, S.C. Laws, L.E. Gray, J. A. Kemppainen and E. M. Wilson , 1995, Persistent DDT metabolite p,p'-DDE is a potent androgen receptor antagonist, Nature, 375:581-585.

Source: Dr. Michael Smolen, World Wildlife Fund, USA.

DDT Can Reduce Breast Milk

Besides the numerous health effects of DDT, the presence of DDT and DDE (a breakdown product of DDT) in breast milk can lead to a decrease in breast milk and shorten the period of lactation, according to some studies. This is significant in that breast milk is the main source of healthy food and nutrition for infants in developing countries, and it is in these countries that DDT is still being heavily used.

Studies of the implications of DDT and DDE in mothers' milk done in Mexico and the U.S. have shown that higher levels of DDE in breast milk are associated with shorter periods of lactation. [1] A fall in estrogen levels partly leads to lactation after child birth, but the presence of estrogen mimics like DDT or DDE in these mothers inhibits full lactation, it is said.

But whether it is as a source of contamination of breastmilk, or as a cause of a reduction in breastmilk and shortening of the period of lactation, it is clear that it is the use of pesticides that need to stop, and not the act of breastfeeding itself. Very often contamination and problems with breastmilk are used as a deterrent to women who want to breastfeed their babies. Breastfeeding is very important to the wellbeing and nutrition of the baby.

Source: Global Pesticide Campaigner, September 1998; and additional comments on breastfeeding from the International Baby Food Action Network, November, 1999.

Reference:

1. Gladen, B.C., W.J. Rogan, 1995, "DDE and shortened duration of lactation in a northern Mexican town", American Journal of Public Health, 85 (4). Rogan W.J., B.C. Gladen, et al, 1987, "PCBs and DDE in human milk: effects on growth morbidity and duration of lactation", American Journal of Public Health, 77(10).

effects on its health later in life.

For example, changes in the developing brain can alter neural pathways leading to altered adult behaviour or alter the functions of many endocrine systems. ***(See also Article: Pesticides and Aggression, in page 37).*** Changes to the thymus and bone marrow cells can lead to immune suppression. Changes to the testis or ovary can reduce sperm or egg quality and quantity.

Scientists studying cancer are also concerned that subtle changes in early development can predispose individuals to certain types of cancer later in life, such as prostate or breast cancers. Therefore, the presence of endocrine disrupting chemicals is particularly serious in pregnant or nursing women, and in developing foetuses or infants. ***(See also Article: Pesticides, Organochlorines and Breast Cancer, in this Section).***

We must therefore assess to what extent pesticides are involved in endocrine disruption, specially those pesticides which are produced in large quantities, widely dispersed and frequently transported over long distances over water or through the air. Many pesticides and other synthetic chemicals do not degrade and persist in the environment. Some breakdown in the body into different chemicals that are more biologically active, and interfere with the function of the normal endocrine systems. Many can accumulate in the fat of animals and are passed through the predator-prey food chain.

Preliminary studies have identified pesticides such as endosulfan, methoxychlor, dicofol, lindane, DDT and its metabolites, vinclozolin, chlordecone, toxaphene, 2,4-D, 2,4,5-T, atrazine, carbaryl, dieldrin, heptachlor, mirex, malathion and chlordane as endocrine-disruptors. There is no battery of tests yet available that can ascertain that specific chemicals are either endocrine-disruptors or are safe. Such screening tests are currently being evaluated but until such tests become available, every chemical, especially pesticides, must be considered potentially disruptive.

Exposure to commonly used pesticides is not restricted only to applicators but consumers too. Vinclozolin residues for instance, can be found in many foods, including beans, peas, and onions [9]. DDE (a breakdown product of DDT) may be a more serious concern since it dissolves in body fat, resists degradation, persists in the body for decades and, transferring through the food chain, gets concentrated to high levels in fish, wildlife and humans worldwide. Even when dietary or occupational exposures to the chemical are low on a daily basis, the concentrations in body tissues increase over the years, and by the time a female reaches reproductive age, the concentrations of chemicals such as p,p'-DDE can be substantial. ***(See Box: Common Pesticides as Endocrine Disruptors).***

A Sensitive Target

As mentioned earlier, the developing offspring is the most sensitive target of endocrine disruption. Many man-made chemicals can cross the placental barrier in the womb and diffuse from the mother's body into the developing offspring. Further, fat-loving chemicals lodged in the fat-rich breast milk are passed on to suckling infants. Thus the exposure to concentrated doses of these chemicals in the womb and in early childhood can be

the highest. This is a matter of much concern. This is because much of the development of the nervous, reproductive and immune systems continues long after birth, and exposure to chemicals such as DDT and its metabolites in the early phase of life can have a wide range of effects on this development. Besides the well-known consequences, there may be other, more cryptic, effects arising from a soup of endocrine disruptors. It has been, for example, reported [10] that higher levels of DDE in women shortens the period of lactation; DDE as a contributing factor in lactation failure is a phenomenon that is being noticed throughout the world. ***(See Box: DDT Can Reduce Breast Milk).***

What Should We Do?

We must begin by recognizing that man-made chemicals have the potential to disrupt the natural endocrine systems of animals, and because the endocrine system is interwoven throughout the life of every animal, effects may vary in site and severity. Disturbances instigated in the developing offspring may not be seen until adulthood, far removed from the early endocrine disruption. Likewise, we cannot assume that processes common to insects, fish, shellfish, amphibians, reptiles, birds and mammals are different from the cellular and molecular processes in humans.

We must realize that we are literally awash in man-made chemicals that have not been rigorously tested for their ability to disrupt endocrine systems, and that we can no longer afford to assume that they are inert baggage that we acquire through life. We must assume that man-made chemicals can be endocrine disruptors, and until tests are implemented to screen and test these chemicals, we must be prudent and adopt the precautionary actions necessary to safeguard our survival.

Dr. Michael Smolen of the World Wildlife Fund (WWF) U.S; is part of the team of scientists who have undertaken extensive work and research on environmental contaminants, a major part of which include pesticides, their impacts – particularly on the human endocrine system – and the serious human health and environmental implications of exposure to such contaminants.

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1. Davis D.L., A. Blair and D. Hoel, 1992, 'Agricultural Exposures and Cancer Trends in Developed Countries', Environmental Health Perspectives, 100:39-44.

2. Giwercman A., E. Carlsen, N. Keiding, and N.E. Skakkebaek, 1993, 'Evidence for increasing incidence of abnormalities of the human testis: A review'm, Environmental Health Perspectives 101, (Supplement 2):65-71.

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4. Auger, J., J.M. Kunstmann, F. Czyglik, P. Jouannet, 1995, 'Decline in semen quality among fertile men in Paris during the past 20 years', New England Journal of Medicine, 332(2):281-285.

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6. Bern, H.A., 1992, 'Diethylstilbestrol (DES) syndrome: present status of animal and human studies', in: Hormonal Carcinogenesis, (J. Li, S. Nandi, and S.A. Li, eds.), Springer-Verlag, New York, 392p.

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9. The Pesticide Register, 1991, Joint publication of MAFF and HSE, Issue 3, March 1991. London.

10. Gladen B.C. and W.J. Rogan, 1995, "DDE and shortened duration of lactation in a northern Mexican town", American Journal of Public Health, 85(4):504-508.

2 Acute Effects of Pesticide Exposure

Three farmworkers were transported to the emergency room by their supervisor. They had been working in a vineyard when a nearby cotton field was aerially sprayed with pesticides. The spray had drifted downwind into the vineyard where about a dozen people were working. Many of the workers began to complain of a variety of symptoms, including difficulty breathing, irritation of the eyes and throat, and nausea. The sickest workers were taken to the emergency room, while others were being seen in a local clinic. There was no information available yet about what the workers were exposed to.

Overview

Acute pesticide poisonings present with rapid onset of symptoms—such as those in the case above—stemming from exposures generally within the past several hours or days. Acute pesticide poisonings are the pesticide-related health effect that practitioners are most likely to recognize and treat. However, large numbers of acute pesticide poisonings each year go undiagnosed and unreported, according to pesticide researchers.[1] The available reporting data indicate that each year between 2000 and 5000 individuals require hospitalization as a result of pesticide poisoning in the United States.[2] Children under six years of age represent more than half of acute reported pesticide poisoning incidents, usually via accidental ingestion or dermal exposure.[3] An estimated 10,000-20,000 farmworkers in the United States suffer from acute pesticide poisonings each year.[4] In California the state's Pesticide Illness Surveillance Program reported nearly 4000 farmworker pesticide poisonings from 1991 to 1996.[5]

Physicians should be aware of the pesticide poisoning reporting requirements under the California Health and Safety Code.[6] The state Pesticide Illness Surveillance Program (PISP) requires that "any physician or surgeon who knows, or has reasonable cause to believe, that a patient is suffering from pesticide poisoning or any disease or condition caused by a pesticide shall promptly report that fact to the local health officer by telephone within 24 hours and by a copy of the report within seven days." Failure to report can result in civil penalties of up to $250. County health officers must then report to county agricultural commissioners, who determine whether the cases are potentially related to pesticides. The state Department of Pesticide Regulation (DPR) administers the program. Pesticide illness records are useful for assessing the public health implications of pesticide use and the effectiveness of current regulations. DPR reports, however, that most pesticide illness data are obtained from workers compensation reports rather than through the PISP.[7]

Careful diagnosis is critical. An EPA model screening protocol is included in the appendix of this resource kit. For a comprehensive guide to protocols for diagnosis, treatment, and follow-up of acute pesticide poisoning, refer to the U.S. EPA handbook on Recognition and Management of Pesticide Poisonings.[8]

Acute Organophosphate and n-methyl-Carbamate Toxicity

Organophosphate and carbamate pesticides are among the most common causes of pesticide poisonings and hospitalizations in the United States.[9]

Organophosphate (OP) insecticides irreversibly deactivate the enzyme acetylcholinesterase, thereby destabilizing neurotransmission at synaptic junctions. This leads to overstimulation of both the sympathetic and parasympathetic nervous systems.[10,11] Specific antidotes and therapeutic protocols are available for organophosphate and carbamate poisonings.

One of the most frequently used OP pesticides is chlorpyrifos (Dursban or Lorsban). It is widely used to kill insects in agriculture, as well as in home insect sprays and in dips to kill fleas. Other common OP insecticides include malathion, azinphos-methyl (Guthion),

methyl parathion, diazinon, demeton, and phosmet. These pesticides are often used in agriculture, homes and gardens.

The N-methyl-carbamate insecticides also deactivate acetylcholinesterase, but the inhibition is reversible rather than permanent. Thus, while the symptoms of carbamate and organophosphate poisoning are identical and may be equally severe, carbamate poisoning generally runs a shorter course.[12] Common N-methyl-carbamate pesticides include carbaryl (Sevin), aldicarb (Temik), fenoxycarb, propoxur, and methomyl.

Signs and Symptoms

The symptoms of OP or carbamate poisoning include bradycardia, dyspnea, wheezing, nausea, vomiting, diarrhea, ocular meiosis, fasciculations, muscle weakness, and hypersecretion, (e.g., lacrimation, perspiration, rhinorrhea, and salivation). Central nervous system signs and symptoms are also prominent, including headache, dizziness, restlessness, and anxiety. Severe intoxication may result in psychosis, seizures, and coma.[13]

Children may present with a different clinical picture from adults. Hypotonia, lethargy, seizures, and coma were more common presenting symptoms in children than in adults, and children rarely present with the classic cholinergic signs of salivation, lacrimation, diaphoresis, bradycardia, or fasciculations.[14]

Theoretically, acute symptoms of organophosphate or carbamate poisoning are classic and easily recognized, but in practice diagnosis can be difficult. Pesticide poisoning can easily be misdiagnosed as gastroenteritis, influenza, bronchitis, or a wide range of other illnesses. Even severe pesticide poisoning requiring intensive care unit admission was misdiagnosed 80% of the time in one series, with diagnoses including pneumonia, meningitis, and epilepsy.[15]

The only way to be sure to correctly diagnose acute pesticide poisoning is to maintain a high index of suspicion and take a screening occupational and environmental history from any patient that presents with suggestive symptoms. Brief questions about occupation, household exposures, and any other potential exposures to fumes, dusts, or gases will allow a rapid assessment of the likelihood that an illness could be related to pesticides or other toxic chemicals.

Diagnosis and Treatment

Plasma or red blood cell cholinesterase levels can be useful in OP or carbamate poisoning, and are readily available through most labs. However, treatment should not be delayed pending results of the laboratory test. Baseline cholinesterase levels, particularly in plasma, are subject to wide variability. As a result, interpretation of the results can be difficult without a baseline for the individual, and a result within the normal range may still represent clinically-significant suppression of cholinesterase for a particular individual.[16] Urinary alkyl phosphates and phenols can be useful for documenting exposure within the first 48 hours, and are more sensitive to low-level exposure than cholinesterase levels.

Therapy for any pesticide poisoning begins with removal of all potential sources of ongoing exposure including gloves and clothing (every effort should be made to ensure privacy when removing clothes in field situations). If residues may be on skin or hair, the patient should be decontaminated with ample soap and water. Supportive care, including continuous cardiac monitoring, oxygenation, airway preservation and aggressive hydration, are all generally indicated.[17]

For many ingested pesticides gastric lavage and cathartics may be indicated. Be aware, however, that gastric lavage is contraindicated with hydrocarbon ingestion (a common vehicle in pesticide preparations), and cathartics may not be needed after ingestion of pesticides such as the OPs and carbamates, which often result in diarrhea.[18] Consultation with a Poison Control Center is highly advisable at this stage.[19]

Atropine sulfate IV or IM is used to control muscarinic symptoms of OP or carbamate poisoning, including lacrimation, salivation, vomiting, diarrhea, and bronchorrhea. This treatment does not affect nicotinic symptoms such as muscle weakness, fasciculations, and

respiratory depression. An atropine challenge can be useful for diagnostic purposes. Atropine is generally administered in repeated doses of 2–4 mg q 15 minutes in adults, or 0.05–0.1 mg/kg q 15 minutes in children until secretory symptoms have reversed. Consult a Poison Control Center or EPA's Recognition and Management of Pesticide Poisonings for current treatment protocols. Repeated doses may be needed for hours, particularly in the case of OP poisoning, and severe poisoning can require very large doses, up to 300 mg/day.[20]

Pralidoxime IV is used to reactivate cholinesterase only in severe cases of OP poisoning. A blood sample for cholinesterase must be drawn prior to administration of pralidoxime. This medication is generally contraindicated in carbamate poisoning. The adult dose of pralidoxime is up to two grams in a slow IV drip, while for children the dose should not exceed 50 mg/kg. Blood pressure and heart rate must be carefully monitored during dosing.[21]

Other Pesticides Associated with Acute Poisoning

Acute symptoms associated with other major pesticide categories are presented in Table 2-1. It is noteworthy that clinical manifestations of acute poisoning have only been studied for a small fraction of pesticides in current use.

Patients who have suffered acute pesticide poisoning require close medical follow-up because certain health effects, particularly neurological impairment, can emerge after apparently successful treatment and recovery.[22]

Preventing Acute Pesticide Poisoning

Advice for Patients

- Avoid using pesticides unless absolutely necessary. Select less toxic alternatives whenever possible. For example, insect baits and traps are almost always safer than broadcast sprays, and non-pesticide alternatives include sealing cracks, cleaning up food scraps, and using soap products to eradicate scents.
- If there are children in the home, make sure that all pesticides are stored out of reach. Do not store any highly toxic pesticides in the home, especially agricultural pesticides or OP pesticides.
- Never store pesticides in containers other than the original, labeled container. In particular, never store pesticides in soft-drink bottles or other food containers.
- If any object, including clothing, containers, or equipment, becomes contaminated with pesticides, discard it or clean it thoroughly and separately. Do not leave any pesticide-contaminated objects in areas where children might come into contact with them.
- Never apply pesticides without following label directions. Always wear protective gloves, long sleeves, and protective clothing. Do not re-enter an area where pesticides were applied until well after any time interval specified on the label.
- If you suspect pesticide poisoning, seek emergency medical care as quickly as possible. Bring along any containers associated with the incident.

Table 2-1: Acute Symptoms Associated With Some Major Pesticide Categories

Pesticide Category	Chemical Examples	Physiological Target	Acute Symptoms	Diagnosis/Treatment
Organophosphates	Chlorpyrifos, diazinon, methyl parathion, malathion, azinphos-methyl, naled	Irreversibly inhibits acetylcholinesterase resulting in muscarinic and nicotinic effects	Vomiting, diarrhea, hypersecretion, bronchoconstriction, headache, weakness	Cholinesterase levels/ Supportive care, atropine, pralidoxime
n-methyl Carbamates	Carbaryl, aldicarb, fenoxycarb, methomyl, bendiocarb	Reversibly inhibits acetylcholinesterase resulting in muscarinic and nicotinic effects	Vomiting, diarrhea, hypersecretion, bronchoconstriction, headache, weakness	Cholinesterase levels/ Supportive care, atropine
Pyrethrins	Pyrethrum	Neuronal paralysis, sensitization	Allergic reactions, anaphylaxis. Tremor, ataxia at very high doses	No diagnostic test/ Treat allergic reactions with antihistamines or steroids, as needed
Pyrethroids Type I	Allethrin, permethrin, tetramethrin	Interference with sodium channel in neuronal cell membranes — repetitive neuronal discharge	Dizziness, irritability to sound or touch, headache, vomiting, diarrhea	No diagnostic test/ Decontamination, supportive care, symptomatic treatment
Type II (cyano-pyrethroids)	Deltamethrin, cypermethrin, fenvalerate	Interference with sodium channel and inhibition of gamma-aminobutyric acid (GABA)	Seizures, dizziness, irritability to sound or touch, headache, vomiting, diarrhea	*Note:* Skin contact may cause highly unpleasant, temporary paresthesias, best treated with Vitamin E oil preparations
Organochlorines	Lindane, endosulfan, dicofol, methoxychlor	Blockade of chloride channel in the GABA receptor complex	Incoordination, tremors, paresthesia, hyperesthesia, headache, dizziness, nausea, seizures	Detectable in blood/ Decontamination, supportive care, cholestyramine to clear enterohepatic recirculation
Chlorophenoxy compounds	2,4-Dichlorophenoxyacetic acid (2,4-D), 2,4-DB, 2,4-DP	Peripheral neuropathy, myopathy, metabolic acidosis, skin and mucus membrane irritant, uncoupler of oxidative phosphorylation	Nausea and vomiting, headache, confusion, myotonia, low fever, acidosis, EKG changes, CPK elevation, myoglobinuria	Detectable in urine and blood/ Decontamination, hydration, forced alkaline diuresis
Dipyridyl compounds	Paraquat, diquat	Corrosive, free radical formation, lipid peroxidation, selective damage to pneumatocytes	Pain, diarrhea, headache, myalgias, acute tubular necrosis, delayed pulmonary edema. Neurologic toxicity from diquat	Urine dithionite test (colorimetric), detectable in urine and blood/ Decontamination, do not administer oxygen, aggressive hydration, hemoperfusion
Anticoagulant Rodenticides	Warfarin, brodifacoum, difenacoum, coumachlor, bromadiolone	Antagonize vitamin K, inhibition of clotting factors	Nosebleeds, hematuria, melena, ecchymoses	Elevated PT and INR/ Vitamin K administration
Chlorophenols	Pentachlorophenol (PCP, Penta)	Uncouples oxidative phosphorylation, skin and mucus membrane irritant	Fever, tremor, thirst, sweating, tachycardia, hypercapnia, chest constriction, abdominal pain	Detectable in blood and urine/ Decontamination, supportive care, control hyperthermia
Nitrophenols and Nitrocreosols	Dinocap	Uncoupler of oxidative phosphorylation	Hyperthermia, tachycardia, anxiety, confusion, headache, diaphoresis	Detectable in serum, bright yellow staining of skin and urine/ Supportive care, control hyperthermia
Fumigants	Methyl bromide	Irritant, inhibits sulfhydryl enzymes and reversibly breaks down ATP	Headache, ataxia, tremor, agitation, visual disturbances, vomiting, seizures, pulmonary edema	Blood or urine bromide levels/ Supportive care, benzodiazipines, dimercaprol
Fumigants	Metam sodium	Decomposes in water to methyl isothiocyanate, severely irritant gas	Mucus membrane irritation, pulmonary edema	No diagnostic test/ Supportive care

Source: J.R. Reigart and J.R. Roberts, *Recognition and Management of Pesticide Poisonings*, Fifth Ed. U.S. Environmental Protection Agency, EPA 735-R-98-003. 1999. Online at http://www.epa.gov/pesticides/safety/healthcare

3 Dermatologic Effects of Pesticide Exposure

An agricultural worker comes in with a rash on her hands and arms. It appeared three days ago, the day after she went into some recently sprayed strawberry fields to pick fruit. She reports that many co-workers have similar rashes but have not sought medical attention: They fear losing their jobs if they report the problem. She does not know the name of the pesticide sprayed, but thinks it is used to control mold. She mentions that she is pregnant and wonders whether the chemical could harm her baby.

Overview

Many pesticides penetrate the skin and cause systemic exposure.[1] Acute illness and death have been reported from percutaneous absorption of pesticides, particularly through damaged skin.[2]

Dermatitis is the second most common occupational disease. Rates in the agricultural industry are the highest of any industrial sector.[3] In California, pesticide-related skin conditions represent between 15% and 25% of pesticide illness reports.[4]

Skin reactions can involve any skin area, including areas covered by clothing, particularly if the pesticide contacts the clothing and soaks through. However, exposed areas, such as arms, hands, face, and neck, are most commonly affected.[5]

Pesticides are reported to cause irritant dermatitis, allergic contact dermatitis, and other skin conditions, including photodermatitis, porphyria cutanea tarda, and chloracne.[6]

Plants alone can also cause dermatitis. Strawberries, mangoes, and some nursery plants are common causes of allergic contact dermatitis. Parsley and limes can cause photodermatitis.[7,8]

Irritant Dermititis

- Soil fumigators can get irritant dermatitis and chemical burns of the lower extremities from methyl bromide, dichloropropene (Telone), and metam sodium. These can be prevented by use of chemical-resistant boots.[9,10]
- Other pesticides frequently associated with irritant dermatitis include the herbicides paraquat and diquat, the miticide propargite, and the fungicides sulfur, ziram, benomyl, and captan. Reactions are generally more severe in the setting of pre-existing skin abrasions, such as those produced by picking or weeding prickly or rough crops.[11]

Allergic Dermititis

- Fungicides are particularly known as potential skin sensitizers. The ethylene bisdithiocarbamate (EBDC) fungicides such as maneb, mancozeb, zineb, and ziram break down to ethylene thiourea, a known sensitizer.[12,13,14]
- Sulfur is one of the most commonly reported causes of skin reactions among agricultural workers. This compound is a skin irritant, but can also cause allergic dermatitis.[15,16]

Table 3.1 Pesticides Reported to be Associated with Allergic Contact Dermatitis

Acephate	Diazinon	Malathion	Pyrethrum
Benomyl	Dienochlor	Mancozeb	Sulfur
Captan	Dimethoate	Maneb	Thiram
Carbaryl	Ethoxyquin	Norflurazon	Vinclozolin
Chlorothalonil	Fenbutatin-oxide	Omethoate	Zineb
Chlorpyrifos	Fluvalinate	PCNB	Ziram
DCNA	Folpet	Permethrin	

Source: M.A. O'Malley, Skin reactions to pesticides, *Occup Med State Art Rev* 12 ([1997]2): 327–45.

- The organic pesticide *Bacillus thuringiensis* has recently been shown to induce skin sensitization in exposed workers,[17] as have the fungicide triforine and the organophosphate insecticide dichlorvos (DDVP).[18]
- Patch testing with standardized concentrations of certain pesticides can be used to confirm sensitization.[19]

Other Skin Manifestations of Pesticide Exposure

- Paraquat and diquat, herbicides that can cause skin burns, are also known to severely damage fingernails.[20,21]
- Various herbicides have been associated with chloracne, potentially due to contamination with dioxins. The principal herbicide that has been associated with chloracne is 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), the now-banned primary constituent of Agent Orange. Other herbicides potentially associated with chloracne include 2,4-D, diuron, and linuron.[22]
- Porphyria cutanea tarda has been reported following exposure to hexachlorobenzene and diazinon.[23]

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4 Pesticides and Cancer

A 41-year-old farmworker comes into your office complaining of fatigue and bone pain. Since teenagers, he and his sister have worked in fields harvesting crops and mixing pesticides. His work-up reveals multiple lytic bone lesions, pancytopenia, and a monoclonal immunoglobulin spike. A bone marrow aspirate confirms a diagnosis of multiple myeloma. He responds well to treatment. He later tells you that his sister was treated for a soft-tissue sarcoma a few years ago at age 36. Both siblings are motivated to encourage co-workers to participate in a study of farmworker health that is being proposed by the Public Health Department. They ask if their diseases could be related to pesticide exposure. How do you respond?

Overview

A wealth of research explores connections between pesticide exposure and neoplasia. Collected clues from the fields of molecular biology, toxicology, biochemistry, and epidemiology may help us chart a course for cancer prevention.

Numerous pesticides are implicated in causing or promoting many types of cancers, leukemias, and lymphomas. Some of these diseases are relatively common, others quite rare. Many of the neoplasms for which association with pesticides is most well-established are among those cancers increasing in incidence in industrialized countries. It is unclear whether exposure to pesticides is causally related to the rising rates of these cancers.

The mechanisms by which pesticides contribute to cancer causation vary, and one pesticide may operate by more than one of the major mechanisms, which include

- Genotoxic effects—producing direct changes in DNA.
- Promotion—causing fixation and proliferation of abnormal clones. This process includes endocrine effects that may stimulate otherwise quiescent but hormonally sensitive cells to carcinogenesis.
- Immunotoxic effects—disturbing the body's normal cancer surveillance mechanisms.

Whereas the usual concept of toxicity follows the principle that "the dose makes the poison," genotoxic chemicals and hormone disruptors may have effects at very low doses without a true threshold below which no risk exists (the stochastic or probabilistic model). Current understanding of carcinogenesis favors the conclusion that even a tiny dose of a genotoxic agent can initiate the process of converting a normal cell to a malignant one.[1,2,3,4,5]

In the field of endocrine disruption, some scientists argue that because background levels of endogenous hormones such as estrogen are known to promote cancer, any additional external hormonally active agents add to an already established risk.[6] For these reasons, at least in theory, even rather low-dose exposure to certain carcinogens may pose a health risk.

Three major lines of evidence relate cancer to pesticide exposure:

1. Cell-culture studies that demonstrate effects such as chromosomal damage or estrogenicity.
2. Laboratory animal studies (see Table 4-1).
3. Human epidemiological investigations.

This section focuses primarily on human epidemiological evidence linking pesticide exposure and cancer.

Overview of the Epidemiological Evidence

Abundant in vitro and animal research on the potential carcinogenic effects of pesticides is available and often leads to important advances in understanding human carcinogenesis. However, to eliminate the variable of cross-species interpretation of tests, we confine

discussion to the study of exposed humans. In the case of pesticides, a number of occupational, home, and other environmental studies illustrate the risks of exposure.

For many human studies of pesticides and cancer, the pesticide specifically responsible for carcinogenesis has not been determined. Because occupations in agriculture involve use of multiple agents (including non-pesticidal chemicals), it is often difficult to determine what agent is linked to a specific endpoint. The same problem occurs with home and environmental exposures, where multiple products may be used, their doses unmeasured, their names long forgotten by those exposed. In this document, whenever studies are specific enough, the class or type of implicated pesticide will be provided.

It is scientifically difficult to prove that something causes cancer. For example, it took a decade of research to confirm the causative link between cigarettes and lung cancer, despite the fact that smoking causes more than 90% of all lung cancers[7] and one third of all cancers in the U.S.[8]

When we refer to the risk of developing various cancers, it should be understood that pesticides are not the only possible cause of any given disease (e.g., leukemia may be caused by some pesticides and also by other chemicals such as benzene). It is usually not possible to know, on an individual basis, all factors that have contributed to carcinogenesis. The following information summarizes those substances that should stimulate suspicion and rigorous study if we are to progress toward prevention.

Pesticides and Cancers of Adulthood

Hematological Malignancies

Non-Hodgkins Lymphoma (NHL)

Sometimes called the "silent epidemic," over the last several decades Non-Hodgkins Lymphoma (NHL) incidence has been increasing by 3–4% per year throughout most of the world.[9,10] In some studies annual increases in incidence are as high as 4.2–8.0%.[11,12,13] These reported increases are corrected for known viral causes of NHL, such as human immunodeficiency virus (HIV), and therefore largely exclude AIDS-related lymphomas.[14,15] Some research on pesticide workers demonstrates associations between occupational exposures (in agriculture or exterminator work) and NHL.[16,17,18] A large number of studies find more specific correlation, especially to phenoxy herbicides such as 2,4-dichlorophenoxyacetic acid (2,4-D).[19,20,21,22] Other research on pesticide workers implicates furan and dioxin contaminants (2,3,7,8-tetrachlorodibenzo-p-dioxin) of the phenoxy herbicides.[23] Although the phenoxy herbicides and their contaminants are the most consistently NHL-associated chemicals, investigators raise concern about other pesticides, including lindane (used in some head and body lice-treatments),[24] organophosphate pesticides,[25] and a variety of others, such as carbaryl, chlordane, dichlorodiphenyltrichlorethane (DDT), diazinon, dichlorvos, malathion, nicotine, and toxaphene.[26] Evidence shows that some fungicides may also be lymphomagens.[27]

Other epidemiologists have studied exposure of persons who are not pesticide workers but live in areas of pesticide use or drift. Herbicide spraying doubled the risk of fatal NHL in a study of persons living in agricultural regions in Canada.[28] The phenoxy herbicides were associated with increased risk of NHL among residents of rice-growing areas in northern Italy.[29] In the U.S., a cluster of NHL and other B cell malignancies has been reported in a Midwestern farming community.[30]

Humans and their dogs live in close proximity, and a study of canine cancer reinforces the above data. Increased risk of canine malignant lymphoma has been associated with pets' exposure to 2,4-D on lawns.[31]

Multiple Myeloma (MM)

Multiple Myeloma (MM) is another hematological malignancy for which age-adjusted incidence seems to have increased during the last several decades. Rates vary, even among industrialized countries: U.S. investigators found an increased incidence of 4% per year from the late 1940s to the early 1980s among white men and women.[32] In contrast, epidemiologists in Spain observed a greater than 10% annual rise from the 1960s to the

mid-1980s.[33] A number of reports cite intermediate increases in several other nations.[34] Many epidemiological studies reveal an association between employment in farming and the chance of contracting MM, with risks as high as 5-fold.[35,36,37,38,39]

Some investigators have more specifically identified possible causative agents. One study of herbicide applicators reports an 8-fold increase in risk of succumbing to MM.[40] The phenoxy herbicides are implicated in this excess risk,[41] an association that should not be surprising since the malignancy is closely related to lymphoma. Chlorinated insecticides are also associated with increased risk for MM in another study.[42]

Hairy Cell Leukemia (HCL)

Increased occurrence of a rare disease is often more obvious to researchers than a similar rise in the rate of a common illness. The latter tends to get "washed out" among the large numbers of expected cases. Hairy Cell Leukemia (HCL) is so rare that multiple recent reports linking it with pesticide exposure raise great interest.[43,44] One study specifically associates organophosphates with HCL.[45]

Myelodysplastic Syndrome (MDS)

Myeloid leukemia and Myelodysplastic Syndrome (MDS) have been associated with occupational exposure to pesticides.[46,47] One case-control study finds significant associations between occupational exposure to pesticides and both acute myeloid and lymphoid leukemia.[48] Review of recent Cancer Registry of Central California data shows correlation of the herbicides 2,4-D and atrazine and the pesticide captan with leukemia among Hispanic males.[49] One cohort study of a group of gardeners known to have been highly exposed to pesticides reveals a nearly 3-fold increased risk for chronic lymphocytic leukemia,[50] an illness for which few possible causes have been proposed.

Soft Tissue Sarcomas (STS) in Adults

As with NHL, development of Soft Tissue Sarcomas (STS) as a function of pesticide exposure is widely studied and frequently correlated. While some studies reveal a simple association with gardening or farming,[51,52] many show a more specific association with the phenoxy herbicides[53,54] or with a combination of exposure to phenoxy herbicides and the pesticide contaminant TCDD.[55]

Occupational exposure to phenoxy herbicides and/or chlorophenol is repeatedly linked to STS.[56,57,58] In one of the most detailed investigations of any tumor/pesticide association, one case-control study of workers with STS derived odds ratios for exposure to three major pesticide-classes—phenoxy herbicides, chlorophenols, and dioxins. The odds of contracting STS after exposure to any phenoxy herbicide was approximately ten times higher than for non-exposed controls; to the class comprising 2,4-dichlorophenoxyacetic acid, 2,4,5-trichlorophenoxyacetic acid, and 4-chloro-2-methylphenoxyacetic acid and to any chlorinated dibenzodioxin or furan, nearly six; and to 2,3,7,8-tetrachlorodibenzo-p-dioxin, greater than five.[59]

Carcinomas and Central Nervous System (CNS) Malignancies in Adults

Skin Cancer and Cancer of the Lip

While elevated risk for skin cancer and cancer of the lip is repeatedly associated with farming,[60,61,62,63] ultraviolet light exposure may be a more likely causative factor than pesticides. Therefore, observation of an association between one specific type of skin cancer—Bowen's disease—and the manufacture of paraquat[64] is of interest because the paraquat-associated skin cancers demonstrate DNA abnormalities which differ from sunlight-induced skin cancers.

Brain Tumors

The age-adjusted incidence of primary tumors of the Central Nervous System (CNS) (particularly astrocytomas, including the rapidly progressive glioblastoma multiforme as well as the benign meningiomas) appears to have increased by 50–100% over the past several decades, with greatest increase among the elderly.[65,66,67] Studies also show increased occurrence of high-grade neuroepithelial tumors, lymphoma, and other primary CNS tumors of 5–13%[68,69] per annum in the elderly. Some observers attribute the apparent increase to the availability of computerized tomography,[70] but disproportionate increase in certain histologic types,[71] parallel increases in mortality,[72] and studies that show diagnostic

imaging only contributes about 20% to case ascertainment all suggest the rise is probably real.[73]

Several studies of workers in farming,[74] gardening and orchard work,[75] pesticide application,[76] and golf-course superintendence[77] show increased risk for primary tumors of the brain. Research analyzing risk of brain cancer among many occupational groups indicates that workers in occupations likely to involve pesticide exposure heighten their liability to brain tumors.[78,79] No studies yet connect specific pesticides to these observed increases.

Respiratory Tract Cancer

Modest increase in cancers of the nose and nasal cavity is reported among workers exposed to phenoxy herbicides and chlorophenols.[80,81] A greater than 2-fold increase in lung cancer (adjusted for smoking) has been observed among structural pest-control workers.[82] Excess cancer of the sinonasal cavities and lungs has been found among women working in agricultural settings.[83]

Gastrointestinal Cancers

Gastric cancer has been associated with work as a farmer,[84] as has colorectal cancer.[85,86] In one retrospective cohort study, colorectal cancer specifically correlated with working in a plant that manufactured the herbicide alachlor. For all exposed workers, risk for developing leukemia or colorectal cancer was 50% higher than for a comparable non-exposed population, while incidence of colorectal cancer among workers with five or more years of the highest alachlor exposure was more than five times greater.[87]

One study finds that biliary and liver cancer correlate highly with work as a pesticide applicator.[88,89] Another study strongly implicates exposure to DDT.[90] Research on workers in plants that manufacture organochlorines shows a nearly 4-fold increased risk from exposure to chlordane, heptachlor, endrin, aldrin, and dieldrin.[91] These pesticides are no longer used in the U.S., but persist in the environment—including termite-protected homes—so exposure may still occur.

A number of studies implicate pesticides in pancreatic cancer. They show that occupational pesticide-exposure increases the risk of pancreatic cancer.[92,93,94] Workers exposed to DDT and related compounds suffer more than a 7-fold increased incidence of pancreatic cancer compared with non-exposed workers.[95] In short, organochlorine exposure appears to be consistently linked with a variety of gastrointestinal malignancies.

Urinary Tract Cancer

The U.S. has recently experienced increased incidence of and mortality from renal cancers. According to the Surveillance, Epidemiology and End Results (SEER) national cancer-monitoring program, the last 25 years have witnessed dramatic increases in disease and death from kidney cancer among black and white Americans of both sexes. During the last 20 years, all white men saw increased incidence at 3.1% per year; white women at 3.9%; and African-American men and women, the steepest at 3.9% and 4.3%.[96] Such rates over a 20-year period cannot be explained by early detection, especially given that screening tests are not routinely employed. An environmental cause is likely.

Occupational exposure to pesticides (work in agriculture) has been correlated with increased risk for kidney cancer (or hypernephroma).[97,98,99] One study shows specific risk associated with pentachlorophenol.[100] Among women occupationally exposed to pesticides, one study observed increased incidence of bladder cancer.[101]

Testicular Cancer

Testicular cancer is another malignancy rising in occurrence for the last several decades in virtually all developed nations. Annual incidence increases range from 2.3% to 5.2% in Europe since the 1940s.[102] In Miyagi, Japan, growth is among the highest, with 6.6% per annum.[103] U.S. data suggest similar trends: The nation's oldest on-going statewide tumor registry finds a mean annual increase in testicular cancer incidence of more than 5.5% over the last 60 years.[104]

Studies of offspring of parents who work in agricultural activities reveal higher rates of testicular cancer, with occurrence manifesting in childhood as well as young adulthood.[105]

Another study shows excess risk of testicular cancer among workers exposed to phenoxy herbicides and chlorophenols.[106]

Prostate Cancer

Numerous studies demonstrate small but significant correlations between prostate cancer and occupational settings likely to lead to pesticide exposure,[107,108] as well as jobs involving direct pesticide or herbicide application.[109]

Breast Cancer

Age-adjusted incidence of breast cancer in industrialized countries has increased 1–2% per year for several decades, both before and after introduction of mammography.[110,111] This observation suggests environmental factors may play a role in this common disease.

Recent years have witnessed great controversy over the possibility of attributing increased breast cancer incidence to hormonally active environmental contaminants, including some pesticides. The organochlorines have received special attention due to their estrogenic effects *in vitro*, lab animals, and wildlife. While we cite studies that seem to support that some pesticides contribute to breast cancer causation, it should be noted that there are negative findings as well, so the precise contribution of pesticides to breast carcinogenesis is not settled.

A case-control study of postmenopausal breast cancer measured serum levels of certain organochlorine compounds (DDE, hexachlorobenzene, mirex, and several polychlorinated biphenyls or PCBs). Some increased risk appeared for women with certain types of PCBs and mirex detectable in their serum, but this effect was predominantly restricted to postmenopausal women who had never breast-fed.[112] It should be observed that PCBs, although organochlorines, are not expected pesticide-components.

Another case-control study analyzed breast tissue from patients with invasive cancer for the presence of organochlorines and compared it with control measurements from women with benign breast biopsies. Some, but not all, classes of PCBs were associated with breast cancer, especially among postmenopausal women with estrogen-receptor positive tumors. Hexachlorobenzene levels were also associated with increased risk of malignancy.[113]

Case-control research from Colombia showed an association between serum dichlorodiphenyl-dichloroethane (DDE, a metabolite of DDT) levels and risk for breast cancer.[114] Another study found serum dieldrin levels associated with dose-related, significantly elevated risk of breast cancer, but other organochlorines appeared not to affect risk.[115]

In an ecological study of breast cancer incidence in an agricultural district heavily contaminated with organochlorine and triazine herbicides, a very modest but statistically significant increased risk of breast cancer is evident.[116]

In summary, organochlorine pesticides may disrupt some actions of estrogens. However, the actual effect on breast cancer risk is likely to vary from compound to compound and even change with different endocrine states of the host.[117]

Thyroid

A large cohort study of workers exposed to phenoxy herbicides and chlorophenols reveals increased risk of thyroid cancer among exposed persons.[118]

In a community exposed to unusually high levels of the organochlorine hexachlorobenzene, excess incidence of thyroid cancer was observed.[119] An agricultural region of Minnesota with heavy use of ethylene bis-dithiocarbamate fungicides (such as maneb, mancozeb, and zineb) suffered a nearly 3-fold increased risk. These fungicides are metabolized to ethylene thiourea, a known thyroid carcinogen in animals.[120]

Pesticides and Childhood Malignancies

Every year approximately 8000 children under age fifteen are diagnosed with a malignant disease, most frequently leukemia and brain tumors. Environmental exposure such as to ionizing radiation, hormones, and antineoplastic agents are accepted to be contributors to these diseases. Some childhood tumors such as gliomas, leukemia, and Wilms' tumor seem

to be increasing in incidence, but the cause for most of these illnesses remains unknown.[121] The clues pertaining to pesticides and children should be treated seriously given pesticides' ubiquitous presence, the tendency of children (especially toddlers) to experience their world by tasting it, and the possible increased sensitivity of children to carcinogens.

Childhood Leukemia

Parental occupational exposure to pesticides as well as home and garden pesticide use may increase risk of childhood leukemia.[122,123,124] Home use of pest strips has been strongly associated with risk.[125]

Non-Hodgkin's Lymphoma

Pesticides have been linked to childhood NHL.[126] Children of parents engaged in agricultural work show higher than expected risk.[127]

Brain and Nervous SystemTumors

A multicenter case-control study finds home use of pesticides increases risk of childhood brain cancers.[128] Other research on home pesticide deployment demonstrates highly significant correlation between pediatric brain tumors and use of sprays or foggers to dispense flea and/or tick pet-treatments.[129] Other pesticides implicated include pest strips, termite-control pesticides, lindane shampoo, flea collars, yard and orchard herbicides, home pesticide bombs, and carbaryl for outdoor use.[130] Occupational pesticide use by parents has been associated with increased risk of childhood neuroblastoma.[131]

Sarcomas

A study of parental occupation and childhood cancer shows a strong association between fathers' employment in agricultural work (from six months prior to conception up to the time of diagnosis) and Ewings' sarcoma in offspring.[132] Yard pesticide treatments have been linked to an increased rate of childhood soft-tissue sarcomas.[133]

Wilms' Tumor

Paternal employment in agriculture has been associated with increased risk of Wilms' tumor.[134] In other studies, both paternal and maternal exposures to pesticides correlates with increased risk.[135,136]

Table 4-1: Carcinogenic Pesticides

Chemical Name	Chemical Use
Arsenic acid	Herbicide
Arsenic pentoxide	Insecticide, wood preservative
Arsenic trioxide	Rodenticide
Cacodylic acid	Herbicide, defoliant
Captan	Fungicide
Chlorothalonil	Fungicide
Chromic acid	Wood preservative
Creosote	Wood preservative
Daminozide	Plant growth regulator
Ddvp	Insecticide
Dipropyl isocinchomeronate	Insecticide
Diuron	Herbicide
Ethoprop	Insecticide
Ethylene sodium	Fumigant
Fenoxycarb	Insecticide
Folpet	Fungicide
Formaldehyde	Microbiocide
Iprodione	Fungicide
Lindane	Insecticide
Mancozeb	Fungicide
Maneb	Fungicide
Metam-sodium	Fumigant
Metiram	Fungicide
Ortho-phenylphenol	Microbiocide
Ortho-phenylphenol, Sodium salt	Microbiocide
Oxadiazon	Herbicide
Oxythioquinox	Insecticide, fungicide, fumigant
Para-dichlorobenzene	Insecticide
Pentachlorophenol	Wood preservative
Potassium dichromate	Wood preservative
Propargite	Insecticide
Propoxur	Insecticide
Propylene oxide	Fumigant
Propyzamide	Herbicide
Pyrethrins	Insecticide
S,S,S-tributyl phosphorotrithioate	Defoliant
Silica aeroge	Insecticide
Sodium dichromate	Wood preservative
Thiodicarb	Insecticide
Thiophanate-methyl	Fungicide
Trichlorfon	Insecticide
Vinclozolin	Fungicide

Source: Pesticides listed as known, likely, or probable carcinogens by U.S. EPA Office of Pesticides Programs as of August 1999, or by the state of California under Proposition 65 and the Safe Drinking Water and Toxic Enforcement Act of 1986.

Chapter 4 Notes

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5 Pesticides and Respiratory Disease

A 24-year-old man comes into an occupational health clinic with a three year history of chest tightness, wheezing, and episodic dyspnea. The patient works in a chemical plant that manufactures pesticides. His symptoms began shortly after his transfer to a captafol production line, are worst in the evening and at night, but resolve on weekends and vacations. There is no personal or family history of allergies or asthma. Review of systems reveals rashes on his wrists above his gloves, chronic burning eyes, and rhinitis. Specific bronchial challenge testing reveals a marked and persistent fall in FEV1.[1]

Overview

Acute organophosphate or N-methyl carbamate overexposure is well known to cause cholinesterase inhibition, resulting in bronchoconstriction, increased airway secretions, and respiratory distress.[2]

A few pesticides are known sensitizers and can result in allergic reactions including asthma.[3,4] An association between low-level pesticide exposure and asthma is controversial, and confounded by the fact that animal, plant, and other antigens cannot be completely ruled out.

A few studies report other respiratory effects from pesticides, including pulmonary hemosiderosis, pneumonia-like infiltrates, chronic bronchitis, pulmonary fibrosis, Wegener's granulomatosis, and respiratory muscle impairment.[5,6,7,8,9]

The main target organ for the herbicide paraquat is the lung. This pesticide is selectively taken up by the lung from peripheral blood, and causes oxidative damage presenting as acute pulmonary edema and hemorrhage or as delayed pulmonary fibrosis. Respiratory failure has occurred following exclusively dermal exposure to this chemical.[10]

Pesticides and Asthma

- Case reports and specific bronchial-challenge testing link several pesticides with occupational asthma. These pesticides include captafol,[11] sulfur,[12] pyrethrins and pyrethroids,[13] tetrachloroisophthalonitrile,[14] and several organophosphate and N-methyl carbamate insecticides that appear to have a methacholine-like effect on the lung.[15,16]
- A cross-sectional study of nearly two thousand farmers in Saskatchewan revealed a significant association between physician diagnosed asthma and reported use of cholinesterase inhibiting pesticides. Potential confounding from exposure to fungi and pollen cannot be completely ruled out.[17]
- Plantation workers in India showed a potential association between pesticide exposure and respiratory impairment. Although overall prevalence of asthma was lower among workers than among controls (perhaps due to the well known "healthy worker effect," in which the working population, on average, enjoys a better health status than the overall population),[18] the pesticide exposed workers revealed an exposure-related increase in both obstructive and restrictive deficits on pulmonary function testing.[19]
- Vineyard and orchard workers in Eastern Europe had significantly higher overall prevalence of dyspnea, chest tightness, chronic cough, and chronic phlegm compared with non-pesticide-exposed controls. Among both smoking and non-smoking workers employed for greater than ten years, FEV_1, FEF_{25}, and FEF_{50} were significantly reduced. Exposed workers also had significantly reduced FVC compared to controls. It was not possible to determine whether findings were due to pesticide exposure or to occupational exposure to dust, pollen, or mold. However, the workers were exposed to a variety of organochlorines, organophosphates, sulfur, and inorganic copper compounds.[20]

- Worldwide population trends indicate that the prevalence of asthma is increasing in the general population, particularly among children and young adults. Severity of asthma, as measured by emergency room visits, hospitalizations, and deaths, is also increasing despite treatment advances.[21] Causes of these trends are not well understood, but it is possible that increasing exposure to pesticides may play a role.[22]
- Children are more susceptible to airborne health hazards than adults for several reasons, such as more rapid respiratory rate and greater volume per unit of body weight, and greater average activity level with faster respiratory rates. Furthermore, very young children are naturally closer to the ground or floor, where chemicals denser than air tend to accumulate. The fact that terminal airways of the lung are not fully developed until several years after birth is also significant.[23]

Other Respiratory Diseases Related to Pesticide Exposure

- An interesting case report describes a young woman who developed diffuse pulmonary hemosiderosis four days after she applied a combination of three synthetic pyrethroids (deltamethrin, cyhalothrin, and bensultap) to a strawberry field. The patient developed sudden onset of dyspnea and severe hemoptysis requiring transfusion. Her chest x-ray showed bilateral cloudy infiltrates, and bronchoalveolar lavage revealed hemosiderin-loaded macrophages. All antibodies were negative. The syndrome responded well to cyclophosphamide.[24]
- One group of researchers proposes the existence of a "biocide lung" following prolonged exposure to pesticides. This syndrome is characterized by intermittent pulmonary infiltrates followed by chronic progressive fibrosis.[25]
- In a survey of about 200 Danish fruit-growers, individuals reported using an average of 13 different pesticides. The most commonly used pesticides comprised captan, paraquat, parathion, azinphos-methyl, diquat, amitrol, benomyl, and simazine. Approximately 40% of the growers reported at least one significant respiratory symptom in connection with pesticide spraying, and nearly 20% had diminished peak flow. These findings were more common among workers who did not wear respiratory protection when applying pesticides. X-ray revealed pulmonary infiltrates or fibrotic changes in nearly one quarter of the subjects.[26]
- A case-control study of 101 patients with Wegener's granulomatosis found that cases reported significantly greater occupational exposure to pesticides compared with both healthy controls and controls with other pulmonary diseases. [27]
- A study questionnaire administered to 54 workers in an Eastern European pesticide plant revealed a 50% prevalence of chronic bronchitis. Approximately two-thirds of the workers had significantly decreased peak expiratory flow. Exposed workers also showed significantly diminished maximum inspiratory and expiratory pressures, potentially indicating respiratory muscle weakness.[28]

6 Neurological and Behavioral Effects of Pesticides

A 52-year-old patient draws your attention to a tremor that has become increasingly bothersome over the past year. On examination, the tremor is pill-rolling and resolves with intention; the patient also has a positive Romberg Sign and an unstable tandem gait. You make a preliminary diagnosis of early Parkinson's Disease. The patient's wife mentions that she recently read in the newspaper that most Parkinson's is from environmental causes, and asks if the fact that her husband is a farmer and has used pesticides for years could be related to his early-onset disease.

Overview

Pesticides have been shown to affect both the central nervous system (CNS), and the peripheral nervous system (PNS) in animals and humans via a variety of mechanisms.

The effects of neurotoxic pesticides can be assessed by measuring changes in neurochemistry, neuropathology, and behavior, including subtle effects on visuospatial function, concentration, reaction-time, learning, and short-term memory.[1,2]

Certain pesticides, for example, the organophosphates and N-methyl carbamates, are designed specifically to damage neurological function in insects and are neurotoxic in humans because of similarities in nervous system function between insects and humans.

Human neurotoxic effects may be acute, may represent the chronic sequelae of an acute poisoning, or may result from chronic exposures in the absence of an acute episode of poisoning.[3] This section focuses on the chronic neurotoxic effects of pesticide exposure.

Pesticides and Parkinson's Disease (PD)

There is increasing evidence that a high proportion of Parkinson's Disease (PD) may be associated with environmental factors.[4]

- Specific pesticides and pesticide classes implicated in PD include paraquat, the organophosphates, dieldrin, and the manganese-based fungicides maneb and mancozeb.[5,6,7]
- The designer heroin-like drug MPTP, known to cause a Parkinsonian syndrome in addicts via the neurotoxic effect of its major metabolite, is chemically related to the herbicide paraquat.[8]
- Numerous studies identify a higher incidence of PD in industrialized countries. Within these countries, people who live in rural areas, live or work on farms, or report a history of pesticide use have the highest risk.[9,10]
- Several population-based case control studies identify a 4-fold increased likelihood of past herbicide exposure among patients with PD, and a 3–4-fold increased likelihood of prior exposure to insecticides.[11,12]
- Several recent studies indicate a possible role for gene-pesticide interactions in the etiology of PD. In particular, higher than expected rates of certain glutathione transferase polymorphisms, the slow acetylator genotype of N-acetyltransferase-2, and the slow 4-hydroxylation of debrisoquine (the CYP 2D6 29B+ allele) have all been reported in patients with PD.[13,14,15] These genetic variants may increase risk from environmental exposure by slowing detoxification of exogenous compounds.[16]

Peripheral Neurotoxicity

- The cholinesterase inhibiting pesticides (organophosphates and N- methyl carbamates) interfere with impulse transmission in the PNS. Chronic effects of exposure can include sensory, motor, and autonomic neuropathies.[17]

- Organophosphate pesticides can rarely cause a distinct syndrome known as organophosphate-induced delayed polyneuropathy (OPIDP), which occurs within five weeks after an acute intoxication.[18] OPIDP is characterized by axonal degeneration and secondary demyelination of long tract neurons.[19] Symptoms of OPIDP include paresthesias of the limbs, leg cramping, motor weakness of the wrist and ankle, and, in severe cases, paralysis.[20] Permanent residua include weakness, loss of reflexes, and sensory impairment.[21]
- In some cases, a so-called "intermediate syndrome" may develop 24 to 96 hours following acute organophosphate pesticide poisoning. The main symptoms consist of proximal muscle weakness, profound weakness of the neck flexors, and weakness or paralysis of the muscles involved in respiration.[22] Sensory function is completely spared.[23] This syndrome may or may not be followed by OPIDP.[24] Neither OPIDP nor the intermediate syndrome respond to therapy with atropine or pralidoxime.[25]
- PNS impairment may also occur following chronic occupational exposure to pesticide mixtures, even in the absence of acute poisoning or frank OPIDP. Several studies report an increased prevalence of neurological abnormalities in exposed workers compared with controls. Abnormalities include hyporeflexia, dysequilibrium, reduced vibration sensitivity, and nerve conduction delays.[26,27] Other studies fail to find peripheral nerve conduction delays in workers who have not suffered high level exposure.[28]
- Workers exposed to mixed pesticides, particularly to the dithiocarbamate fungicides maneb and zineb, have been shown to have slowed peripheral nerve conduction. Motor and sensory conduction were affected equally, with some indication of autonomic dysfunction as measured by reduced respiratory variability.[29]

Neurocognitive Effects of Pesticide Exposure

- Many pesticides are able to penetrate the blood brain barrier, while others exert indirect effects on the brain via disruption of oxygen supply, nutrients, hormones, or neurotransmitters.[30]
- Areas of the brain most commonly affected by pesticides include the limbic system, hippocampus, basal ganglia, and cerebellum.[31]
- Evidence of pesticide-associated neuropsychological deficits is based primarily on studies of workers acutely or chronically exposed to organophosphate pesticides, although some case reports also implicate N-methyl carbamate pesticides in the appearance of similar effects.[32]
- Cognitive symptoms in these populations include impairment of memory and psychomotor speed, and affective symptoms such as anxiety, irritability, and depression.[33] Visuospatial deficits have also been linked to organophosphate exposure.[34] Standardized neuropsychiatric testing batteries confirm these deficits in exposed groups compared with unexposed controls. Long-term memory and language abilities are generally spared.[35]
- The fumigants methyl bromide, sulfuryl fluoride, and dichloropropene (Telone) have been reported to cause personality changes and shortened attention span following exposure. Methyl bromide exposure was related to decreased touch sensitivity and reduced cognitive ability; Telone exposure, to increased depression and anxiety reflected in standardized test batteries; and sulfuryl fluoride, to a range of behavioral and cognitive deficits.[36,37]

Pesticides and Seizures

- Many pesticides are known to increase CNS excitability and to produce seizures with acute high-dose exposure.[38]
- Recent animal studies indicate that some pesticides can cause an electrical kindling response after repeated sub-threshold dosing. Low doses repeated three times a week for ten weeks of the pesticide lindane (used to treat head lice) resulted in enhanced myo-

clonic jerks and seizures at normally subconvulsant doses. Other organochlorine pesticides, such as endosulfan and dieldrin, are reported to have similar effects.[39]

Effects of Pesticides on Neurological Development in Children

Neurological development in children is particularly vulnerable to disruption. Although there is some plasticity inherent in the development of the nervous system, even low-level exposure during the brain-growth spurt have been shown to exert subtle, permanent effects on the structure and function of the brain.

- Animal studies have demonstrated periods of vulnerability, particularly to anticholinesterases, during early life.[40] Recent evidence supports the finding that acetylcholinesterase may play a direct role in neuronal differentiation.[41]
- Children from a region in Mexico with intensive pesticide use were found to have a variety of developmental delays compared with otherwise similar children living where fewer pesticides were used. Although the children were similar in growth and physical development, significant delays were noted among the exposed children in physical stamina, gross and fine hand-eye coordination, and short-term memory.[42]

Table 6-1: Chronic or Delayed Neurotoxic Effects of Pesticides

Pesticide Category	Effects on Central Nervous System	Effects on Peripheral Nervous System
Organophosphates e.g., malathion, chlorpyrifos	Cognitive, affective and perceptive effects	OPIDP; sensorimotor neuropathy; intermediate syndrome
Carbamates e.g., carbaryl	Memory deficits; visual impairment; lassitude	Sensorimotor neuropathy
Organochlorines e.g., kepone	Impairment of cognitive function and personality; seizure kindling	Tremor (Kepone shakes)
Metals e.g.,monosodium methyl arsenate, lead arsenate, zinc phosphide	Impaired visuospatial abilities; deficits in short-term verbal memory	Painful, burning dysesthesias
Fumigants e.g., carbon disulfide, dichloropropene, methyl bromide	Cognitive impairment; mood changes; difficulty concentrating; pyramidal signs	Loss of reflexes and distal motor strength
Fungicides e.g., dithiocarbamates—zeneb, maneb, mancozeb	Reduction of physiologic respiratory arrhythmia; possibly Parkinson's	Reduced nerve conduction
Pyrethroids e.g., fenvalerate, cypermethrin	Reduction of spontaneous motor activity; altered startle response	Cutaneous paresthesia; numbness
Rodenticides e.g., vacor (N-3-pyridylmethyl-N-p-nitrophenyl urea)	Minimal data on cognitive impairment	Autonomic incompetence

Sources: M.C. Keifer and R.K.Mahurin, Chronic neurologic effects of pesticide overexposure, *Occup Med* (Philadelphia) 12 (1997): 291–304; M.M. Amr, E.Z. Abbas, G.M. El-Samra, et al., Neuropsychiatric syndromes and occupational exposure to zinc phosphide in Egypt, *Env Rsrch* 73 (1997): 200–206; D.J. Echobichon and R.M. Joy, *Pesticides and neurological diseases*, 2nd ed. (Boca Raton, FL: CRC Press, Inc., 1994); L. Rosenstock, M. Keifer, W.E. Daniell, et al., Chronic central nervous system effects of acute organophosphate pesticide intoxication, *Lancet* 338 (1991): 223–27.

Chapter 6 Notes

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7 Reproductive and Developmental Effects of Pesticides

A 32-year-old man comes in with concerns about fertility. He has been married four years and his wife has not become pregnant despite regular attempts for the past several years. The man reports that he works at a chemical company that manufactures pesticides and that several other men are having similar problems. The men complained to the union steward; all would be coming in for medical evaluation over the next few weeks. Semen analysis reveals azospermia.

Overview

Pesticides may affect human reproduction by direct toxicity to the reproductive organs or by interference with hormonal function.[1,2,3,4] Effects of pesticides on reproduction may include menstrual abnormalities, male or female infertility, or hormonal disturbances.

The developing fetus and infant are disproportionately susceptible to the health effects of pesticides.[5] Developmental toxicity of pesticides may result in spontaneous abortion, growth retardation, structural birth defects, or functional deficits.[6]

There is often a period of vulnerability to the effects of toxic chemicals—including pesticides—during fetal development and early childhood. This vulnerability occurs during the period of development of various organ systems. Permanent structural birth defects or permanent functional changes may occur.[7,8,9]

Male Infertility: The Example of DBCP

The most thoroughly studied human epidemic of pesticide-induced reproductive dysfunction began in the 1970s when men at an Occidental chemical plant in Southern California sought medical care for infertility. Many were sterile, and subsequent investigation found that a fumigant manufactured in the plant, dibromochloropropane (DBCP), was responsible for effects on spermatogenesis and for germ-cell mutations.[10] In many cases, effects were permanent. Rodent studies performed decades earlier found dramatic testicular toxicity in animals, yet this evidence was disregarded until the human outbreak occurred.

Although use of DBCP has been discontinued in the U.S., it is persistent in soil and still present in groundwater in some parts of California. Thus there is potential for ongoing low-level human exposure.[11] The long-term effects of such exposures over the reproductive life-span are unknown. DBCP was still used until recently in Central American banana plantations, resulting in epidemics of sterility in agricultural workers.[12]

Effects of Pesticides on Fertility

Use of chlordecone (Kepone) was discontinued in the U.S. after incontrovertible evidence that it causes decreased sperm mobility and viability, in addition to serious neurological effects in workers.[13]

Exposure to carbaryl has been associated with increased frequency of morphologically deformed sperm, but longitudinal studies have not been conducted to confirm adverse reproductive outcomes.[14]

- The herbicide 2,4-Dichlorophenoxyacetic acid (2,4-D) is spermatotoxic in laboratory animals. A correlation between increased exposure to 2,4-D and decreased sperm density along with increased percentage of abnormal sperm was reported in agricultural pesticide applicators.[15]

- A study of over eight hundred couples undergoing *in-vitro* fertilization revealed that men moderately or highly exposed to pesticides at work had significantly decreased fertilization rates compared with unexposed males, with only one-third the likelihood of successful *in-vitro* fertilization. These effects persisted after adjustment for all other known exposures, including smoking, alcohol, caffeine, and other chemical use.[16]
- Wives of male fruit growers in the Netherlands have shown an increased time-to-pregnancy, particularly during the spring and summer growing season when pesticides are applied. During that season, time-to-pregnancy more than doubled. Twenty-eight percent of farm couples sought medical attention for infertility, compared with only 8% in the control (unexposed) population.[17]
- Increased time-to-pregnancy was also found to be significant in Canadian farm families. During periods when both husbands and wives applied pesticides, fecundability dropped to between 50% and 80% of expected, whereas when only the husband or neither partner applied, fecundability was within normal ranges. There was no clear link to particular pesticides or pesticide classes.[18]

Developmental Abnormalities: Growth Retardation and Spontaneous Abortion

- Numerous studies report an increased rate of spontaneous abortions and stillbirths among female agricultural workers. These studies are limited by potential recall bias, and by difficulties in exposure assessment since workers are exposed to a complex mixture of chemicals and doses are unknown. Some studies of wives of agricultural workers also show an increased risk of spontaneous abortion and stillbirth.[19,20,21,22,23,24]
- A California study demonstrated an association between pesticide exposure at work or in the home and stillbirths, particularly those with congenital anomalies. Elevated risks ranged from a 70% increased risk of stillbirth for home exposure to pesticides, to a 240% increased risk for occupational exposure.[25]
- Higher levels of organochlorine pesticides have been found in abortuses and pre-term infants than in full-term babies.[26]
- Women living in communities supplied with drinking water contaminated by a variety of herbicides, including atrazine, cyanazine, and metolachlor, had an 80% increased risk of intra-uterine growth retardation compared with similar communities with uncontaminated water.[27]
- Teachers working in day care centers in Germany where wood was treated with the pesticides and wood preservatives pentachlorophenol and lindane were significantly more likely to give birth to lower birthweight and smaller size infants. These preservatives are known to volatilize off wood for years and become entrained in air or dust particles.[28]

Pesticides and Birth Defects

Numerous epidemiological studies and case reports associate pesticide exposure at work or home with increased risk of various types of congenital malformations.[29]

Particular birth defects associated with pesticides include

- Cleft lip and palate—a doubling of risk with exposure during the first trimester.[30,31,32]
- Limb defects—a 3–4-fold increased risk for garden or workplace exposure, and greater than doubling of risk with household exposure, particularly if pesticides were applied by a professional pest eradication service.[33,34,35,36]
- Cardiovascular malformations, particularly Total Anomalous Pulmonary Venous Return—a 2–3-fold greater risk found in the Baltimore-Washington Infant Study.[37]
- Spina bifida and hydrocephaly—a 2.7- and 3.5-fold increased risk respectively in one study, and a 50% increased risk with residence within a quarter-mile of an agricultural field in another.[38,39]

- Cryptorchidism and hypospadias—2–3-fold greater rates of orchidopexy in highly agricultural areas; a 50% increase in hypospadias also reported.[40,41]

- A California study using the state birth defects monitoring program found that infants with limb reduction defects along with other anomalies were 60% more likely to have parents involved in agricultural work and 2.4 times more likely to live in an agricultural county compared with unafflicted infants.[42]

- One Minnesota study of pesticide applicators revealed that their children were at higher risk of a variety of birth defects, including circulatory/respiratory anomalies, and urogenital, musculoskeletal, and integumental defects. These same trends and birth defects, although less marked, were paralleled among the general population in heavily agricultural regions of the state. Defects were most significantly associated with use of

Table 7-1. Developmental and Reproductive Toxins

Chemical Name	Chemical Use	Developmental Toxin	Female Repro. Toxin	Male Repro. Toxin
1080	Rodenticide			Y
2,4-Db acid	Herbicide	Y		Y
Amitraz	Insecticide	Y		
Arsenic acid	Herbicide	Y		
Arsenic pentoxide	Multiple uses, insecticide, wood treatment	Y		
Arsenic trioxide	Rodenticide	Y		
Benomyl	Fungicide	Y		Y
Bromacil, Lithium salt	Herbicide	Y		
Bromoxynil octanoate	Herbicide	Y		
Chlorsulfuron	Herbicide	Y	Y	Y
Cyanazine	Herbicide	Y		
Cycloate	Herbicide	Y		
Diclofop-methyl	Herbicide	Y		
Disodium cyanodithioimido carbonate	Microbiocide	Y		
Eptc	Herbicide	Y		
Ethylene oxide	Fumigant		Y	
Fenoxaprop ethyl	Herbicide	Y		
Fluazifop-butyl	Herbicide	Y		
Hydramethylnon	Insecticide	Y		Y
Linuron	Herbicide	Y		
Metam-sodium	Fumigant	Y		
Methyl bromide	Fumigant	Y		
Metiram	Fungicide	Y		
Myclobutanil	Fungicide	Y		Y
Nabam	Fungicide	Y		
Nicotine	Insecticide	Y		
Nitrapyrin	Microbiocide	Y		
Oxadiazon	Herbicide	Y		
Oxydemeton-methyl	Insecticide		Y	Y
Oxythioquinox	Insecticide, fungicide, fumigant	Y		
Potassium dimethyl dithio carbamate	Microbiocide	Y		
Propargite	Insecticide	Y		
Resmethrin	Insecticide	Y		
Sodium dimethyl dithio carbamate	Microbiocide	Y		
Streptomycin sulfate	Fungicide	Y		
Tau-fluvalinate	Insecticide	Y		
Thiophanate-methyl	Fungicide		Y	Y
Triadimefon	Fungicide	Y	Y	Y
Tributyltin methacrylate	Antifoulant, microbiocide	Y		
Triforine	Fungicide	Y		
Vinclozolin	Fungicide	Y		
Warfarin	Rodenticide	Y		

Source: Proposition 65 List of Chemicals Known to the State of California to Cause Cancer and Reproductive Harm (Sacramento: California Office of Environmental Health Hazard Assessment, 29 December 1999). United States Environmental Protection Agency Toxic Release Inventory database.

2,4-D and various fungicides. Risks for children of both pesticide applicators and the general public in the agricultural region were greatest among those conceived in the spring, a time of greater pesticide use.[43]

- Communities in Iowa with elevated levels of the herbicide atrazine in their drinking water showed a 2–3-fold increase in all birth defects—specifically, a 3-fold increase in cardiac defects, a 3–4-fold increase in urogenital defects, and a nearly 7-fold increase in limb reduction defects.[44]
- Numerous case reports and case series present various combined severe congenital anomalies following occupational or accidental exposure of pregnant women to pesticides.[45,46,47]
- Many pesticides are reported to cause birth defects in animals. Pesticides listed as reproductive or developmental toxicants by the State of California or by U.S. EPA are listed in Table 7-1.

Disruption of Hormone Function

Various pesticides mimic estrogen, while others block androgens or thyroid hormone.[48]

- Estrogenic pesticides that have been studied in some detail include numerous banned and still used organochlorine pesticides, such as DDT, chlordecone, dicofol, methoxychlor, endosulfan, and lindane.[49] Fungicides such as vinclozolin and iprodione are antiandrogens.[50] In addition, some triazine herbicides such as atrazine interfere with estrogen via indirect pathways.[51]
- Pentachlorophenol (PCP), a pesticidal wood preservative, binds to human transthyretin and may directly reduce uptake of thyroxine (T4) into the brain.[52,53] Other currently used pesticides, including dicofol and bromoxynil, have similar effects on thyroxine binding, as does dinoseb, now banned.[54]
- Health effects of endocrine disrupting pesticides in animals include altered circulating hormone levels, hypospadias, nipple development in males, cryptorchidism, decreased semen quality, altered time to sexual maturity, and abnormal behavior.[55,56,57]
- Male pesticide factory workers in China exposed to the organophosphate pesticides ethyl parathion and methamidophos had significant abnormalities in their reproductive hormone profiles. Increased pesticide exposure correlated positively with serum LH and FSH levels, and negatively with serum testosterone. In addition, workers with higher exposure tended to show greater risk of abnormal semen parameters.[58]
- Workers applying ethylene bisdithiocarbamate fungicides (such as Maneb or Zineb) in Mexico developed elevated levels of TSH without changes in thyroid hormone levels. Although findings were subclinical in these healthy adult males, they could be relevant to a developing fetus were a pregnant woman exposed.[59]
- In the fetus or neonate, disruption of endocrine homeostasis can result in permanent alterations in sexual development, whereas disturbance in adulthood is less likely to create lasting health effects.[60]

Table 7-2: Endocrine-Disrupting Pesticides

Chemical Name	Chemical Use
Alachlor	Herbicide
Aldicarb	Insecticide
Atrazine	Herbicide
Benomyl	Fungicide
Carbaryl	Insecticide
Chlorpyrifos	Insecticide
Cyanazine	Herbicide
Endosulfan	Insecticide
Lindane	Insecticide
Malathion	Insecticide
Mancozeb	Fungicide
Maneb	Fungicide
Methomyl	Insecticide
Methyl parathion	Insecticide
Metiram	Fungicide
Metolachlor	Herbicide
PCNB	Fungicide
PCP	Wood preservative
Pyrethrins	Insecticide
Resmethrin	Insecticide
Simazine	Herbicide
Tributyltin methacrylate	Antifoulant, Microbiocide
Tributyltin oxide	Antifoulant, Microbiocide
Vinclozolin	Fungicide

Sources: L. Keith, *Environmental endocrine disruptors* (New York: Wiley Interscience, 1997); J. Liebman, *Rising toxic tide* (San Francisco: Pesticide Action Network/Californians for Pesticide Reform, 1997); Illinois EPA, *Report on endocrine disrupting chemicals* (Illinois EPA, 1997); T. Colborn, D. Dumanoski, and J.P. Myers, *Our stolen future* (New York: Penguin Books, 1996), 253; C.M. Benbrook, *Growing doubt: A primer on pesticides identified as endocrine disruptors and/or reproductive Toxicants* (The National Campaign for Pesticide Policy Reform, September 1996).

8 Effects of Pesticides on the Immune System

A family comes into a local clinic because the state health department recently informed them of pesticide contamination in the well water in their small town. They want to know whether their children's persistent respiratory infections and skin rashes might be associated with the water contamination problem. They are particularly concerned about immune problems and want to have their immune functions tested. They also want to know whether switching to bottled water is sufficient to protect them.

Overview

There is limited evidence that exposure to certain pesticides may compromise the immune system. Findings are based primarily on animal studies that demonstrate damage to immune organs, suppression of immune-mediating cells, and increased susceptibility to infectious disease.[1,2,3,4,5,6]

The intrinsic variability of immune parameters between and within individuals makes study of the effects of environmental or occupational exposure on human immune function extremely difficult.

Pesticide exposure has been associated with

- Hypersensitivity reactions ranging from dermatitis to asthma or anaphylaxis.
- Suppression of immune function and consequent susceptibility to infectious pathogens.
- Autoimmune responses.
- Cancers of immune cell lines *(see Section 2. Pesticides and Cancer).*

Allergic Responses

- Some pesticides may cause immediate hypersensitivity symptoms such as rhinitis, asthma, or anaphylaxis.[7,8] Pesticides reported to cause hypersensitivity reactions in humans include atrazine, parathion, dichlorvos, captafol, folpet, captan, naled, maneb, zineb, dithianone, and dinitrochlorobenzene.[9,10]

Immune Suppression

- Adults occupationally exposed to organophosphate or organochlorine pesticides were found to have increased frequency and severity of respiratory infections such as tonsillitis, pharyngitis, and bronchitis. These workers also showed diminished neutrophil response—related to duration of exposure to pesticides—including impaired phagocytosis, respiratory burst, and adhesion.[11,12]
- In humans, one now—banned organochlorine pesticide, chlordane, was associated with abnormal T-cell and B-cell subsets, decreased proliferation response to mitogen, and suppressed antibody-dependent cell cytotoxicity. These findings were statistically significant among people whose homes were sprayed with this pesticide for termite control.[13]
- A study of Nebraska farmers showed slight but significant reductions in serum complement activity in the most highly pesticide-exposed group. No consistent differences in total leukocyte count, mitogen-stimulation of T-cell or B-cell proliferation, or serum IgG and IgM concentration among the groups were detected.[14]
- Women who consumed aldicarb contaminated groundwater in a potato farming area had significantly decreased CD8 cell subsets when compared with women drinking uncontaminated groundwater.[15]
- The environmentally persistent wood preservative pentachlorophenol (PCP) is consistently associated with a range of abnormal immune parameters, from increased levels of serum IgM and increased immature leukocytes to greater incidence of infection and

aplastic anemia. Proliferative responses to mitogen and antigen have been reported to be significantly depressed in residents of log homes preserved with PCP.[16,17]

Autoimmunity

- Metal-based pesticides such as arsenic and copper are repeatedly associated with autoimmune responses.[18]
- A small four year follow-up study of people overexposed to chlorpyrifos reveals persistently higher levels of antibiotic sensitivity, autoimmunity, and CD26 cells.[19]
- Other pesticides reported to be associated with indications of autoimmunity in humans include chlordane/heptachlor, pentachlorophenol, and formaldehyde.[20]

Other Possible Immune Effects

- Some researchers hypothesize that several controversial and poorly understood syndromes, including Multiple Chemical Sensitivity Syndrome, Chronic Fatigue Syndrome, and Gulf War Syndrome, may be due to an immunotoxic response to pesticides and other chemicals. Testing of immunologic parameters in these individuals yields conflicting results.[21,22,23,24] At present, the etiology of these syndromes is unknown and the effects on the immune system have not been established.

Table 8-1: Immunotoxicity of Pesticides

Pesticide	Immune Effect
Organophosphates	
Dichlorvos	Inhibits complement Interferes with lymphocyte DNA repair Suppresses serum antibody titers to *S. typhi*
Malathion	Stimulates macrophage respiratory burst and phagocytosis Suppresses humoral immunity
Parathion	Decreases resistance to viral and bacterial infection Decreases T-cell proliferation Delays antibody production
Chlorpyrifos	Increases CD26 cells, autoimmunity, and antibiotic sensitivity
Carbamates	
Carbaryl Carbofuran Aldicarb	Decreases macrophage cytotoxicity Inhibits T-cell activation to mitogen (worse with multiple low doses) Decreases CD8 cells Increases response to *Candida* antigen Increases total lymphocytes[a]
Pentachlorophenol	Reduces humoral response Decreases IL-2 production Decreases CD4 cells Increases immature leukocytes Increases chronic cutaneous inflammation
Metam sodium	Increases complement activity Decreases NK cell activity
Organochlorines	
Chlordane Heptachlor	Produces abnormal B- and T-cell subsets Decreases mitogen response Decreases antibody-dependent cytotoxicity Increases autoantibody production Delays macrophage activation
Aldrin Dieldrin	Decreases resistance to viral infection suppress macrophage activity
Lindane Benzene hexachloride	Decreases macrophage activation Decreases resistance to giardia
Tributyl tin oxide	Decreases ability to resist bacterial and parasitic infection Creates immune dysfunction at low dose levels[b]

Source: I. Voccia, B. Blakley, P. Brousseau, and M. Fournier, Immunotoxicity of pesticides: A review, *Toxicol Ind Hlth* 15 (1999): 119–32. Notes: *a* T. Vial, B. Nicholas, and J. Descotes, Clinical immunotoxicity of pesticides, *J Toxicol Env Hlth* 48 (1996): 215–29. *b* P. A. Botham, Are pesticides immunotoxic? *Adverse Drug React Acute Poison Rev* 9 (1990): 91–101.

HIDDEN DIMENSIONS OF DAMAGE

Pesticides and Health

MONICA MOORE

THE IDEA THAT PESTICIDES ARE DANGEROUS IS NOT CONTROVERSIAL. *After all, pesticides are created and released into the environment in order to kill organisms considered pests, be they insects, weeds, bacteria, fish, snails, birds, rodents, or other forms of life. Yet most people do not realize just how dangerous many pesticides are, either individually or in combination with one another, or how far beyond their intended targets the harmful effects of pesticides actually reach. Ultimately, pesticides affect all members of an ecosystem, from the tiniest invertebrates to humans and other large animals living at the top of their food chains.*

Although the true extent of pesticide-related damage has never been (and may never be) fully quantified, enough is known to indicate that these chemicals are very costly to the health of present and future generations. The long list of known and suspected health problems linked to pesticides grows steadily as new scientific discoveries reveal more of the intricate systems in and around us that influence our health and development from the moment of conception until we die. Numerous studies document disturbing levels of pesticide poisonings and other damage in wealthy as well as poor countries, and knowledgeable sources agree that these documented cases represent only a fraction of the actual total. And of course human poisonings are only the beginning of a much larger story of poisonings.

The fact that spreading billions of pounds of toxic pesticides throughout the environment each year results in extensive harm should not be surprising to policy makers, growers, or the general public. Yet somehow it remains not just surprising, but eternally so. This never-ending lack of awareness of the true scale of damage keeps people from challenging assumptions that societies benefit more than they lose from continuing their dependence on pesticides. Meanwhile, the true dimensions of pesticide damage to human health and the environment remain among the best kept, least acted on secrets of agricultural, public health, development, and regulatory authorities around the globe.

This article considers the extent of pesticide-caused damage to human health. Because we humans are an integral part of the environment, environmental health impacts are an important part of this discussion. Assessing pesticide damage requires pulling together information of many different types from many sources, including acute and chronic effects of different kinds of pesticides; fate and transport of pesticides in the environment; pesticide poisonings statistics; and information on pesticide use, sales, and markets. Some of this information is reasonably easy to find, if you know where to look for it. Other pieces of the puzzle exist only as educated guesses or closely guarded commercial secrets that require determined efforts, dumb luck, huge sums of money, or all of the above to bring to light.

Understanding the true dimensions of pesticide health effects also requires the consideration of factors that shield pesticide damage from public scrutiny and outrage. For example, how is it that so many people have no idea either of the scale of pesticide use in conventional agriculture or of the extent of public health problems caused by these chemicals? Why is the public so unaware of the unavoidable exposures to pesticides they endure daily through their food, water, air, workplaces, and living environments? Most disturbingly, why is agricultural reliance on pesticides growing despite often heroic efforts of ecologically minded farmers to meet consumers' preferences for organically produced food and fibers?

Addressing these questions means examining both the biological mechanisms of pesticide poisonings and pest resistance, and the mechanisms of power that operate in corporate boardrooms and national capitols. This means looking directly at the economic, social, and cultural contexts that grant official invisibility to epidemic levels of poisonings and other forms of pesticide damages. Seen from this perspective, pesticides can be important teachers that help us see interconnections among seemingly distant people, places, and ecological communities. But insights into interconnections without actions to reduce pesticide use and promote safer, ecologically based alternatives will not prevent further damage. Unfortunately, the lesson that pesticides should be teaching us — that an ounce of prevention is much better than a pound of cure, especially when the damage is avoidable and no cure exists — has yet to be learned. For this reason, exposing the hidden dimensions of pesticide damage remains an urgent public and environmental health priority and a continuing challenge for the sustainable agriculture movement.

HOW PESTICIDES DAMAGE HUMAN HEALTH

Pesticides can affect human health through acute (short-term) effects, chronic (long-term) effects, or both. Chronic health effects can be delayed effects from an individual exposure or the result of repeated low-level expo-

sures, whose impacts build up over time. Most pesticides have acute toxic effects; many also present serious chronic hazards. Pesticide exposures can also worsen existing illnesses and medical conditions, including asthma and other respiratory illness, liver and kidney disease, and many others.

Acute Pesticide Poisoning. Symptoms of acute pesticide poisonings may be local, causing irritation or damage to the skin or eyes. Some pesticides can cause allergic reactions, another type of acute effect. Acutely toxic pesticides can also affect the body systemically, causing problems as they begin moving through the blood. Many pesticides generate both local and systemic effects. Specific symptoms vary according to the type of pesticide and also within types. The following section describes a range of acute poisoning symptoms for several major types of pesticides, illustrating some of the many ways that pesticides affect human health.

Nerve Poison Pesticides. Two closely related types of nerve poison pesticides, the organophosphates and the methyl carbamates, are responsible for most acute pesticide poisonings and deaths in the United States and worldwide. Both of these compounds kill insect pests by stopping a critical nerve impulse–transmitting enzyme from functioning normally. Unfortunately, they block the same enzymes in the bodies of non-target insects, birds, fish, reptiles, mollusks, amphibians, and mammals, including people. Mild systemic poisoning symptoms produced by these pesticides include blurry vision, headache, dizziness, fatigue, diarrhea, nausea and vomiting, heavy sweating, and muscle or abdominal pain. As the level of poisoning increases, a victim's pupils shrink and he or she experiences difficulty walking, talking, and concentrating. Twitching muscles and generalized weakness are also symptoms. Signs of severe poisoning include pinpoint pupils, convulsions, unconsciousness, difficulty breathing, coma, and death. Organophosphate and carbamate pesticides are widely produced and used throughout the world.

Organochlorine Pesticides. This category includes DDT, the world's most notorious pesticide, along with other less famous compounds. Organochlorines affect the brain and increase the sensitivity of neurons. While better known for their chronic effects, many organochlorines are highly acutely toxic as well. Convulsions are the classic acute poisoning symptom for this category,

and may or may not be accompanied by other symptoms, including headache, dizziness, nausea, vomiting, tremors, lack of coordination, and mental confusion. Organochlorines can also cause local irritant effects, including allergic reactions. Although some older organochlorine pesticides have been widely banned, use of others remains common in the United States and throughout the world.

Pyrethrins and Synthetic Pyrethroids. Pyrethrins are naturally occurring compounds derived from chrysanthemum flowers, and pyrethroids are their synthetically manufactured chemical cousins. These compounds also affect the brain and nervous system, although differently than do the two pesticide types mentioned above. Acute poisoning symptoms produced by these pesticides include local skin irritation, multiple allergic reactions, dizziness, tremors, irritability to sound or touch, headache, vomiting, and diarrhea. Because this class of insecticide tends to break down sooner in the environment than do many organochlorines, they are often substituted for them, and are used widely in agriculture, as well as in homes and gardens.

Dipyridyl Pesticides. This category includes the herbicides paraquat and diquat, highly toxic compounds responsible for many acute poisonings in the United States and internationally. These pesticides are very strong irritants that can severely damage the skin, eyes, mouth, nose, and throat, including causing blindness and fingernail loss. They destroy lung tissue and cause failure of the kidneys, liver, and other organs. Symptoms of poisoning by these pesticides include pain, vomiting, diarrhea, headache, nosebleeds, loss of appetite, and death. Paraquat in particular is in wide use throughout the world.

Chlorophenoxy Herbicides. This category of herbicides includes the well-known weed killer 2,4-D and also 2,4,5-T, an ingredient of the Vietnam War–era defoliant known as Agent Orange. Products containing 2,4-D are big sellers in both agricultural and over-the-counter home and garden products. While the long-term health effects of phenoxy herbicides are usually considered more serious, acute poisoning symptoms can include skin irritation, headache, nausea, vomiting, low fever, mental confusion, abdominal pain, and temporary changes in heartbeat.

"Inert" Pesticide Ingredients. The already daunting task of evaluating pesticide harm is made much more difficult by the unidentified "inert"

ingredients found in all formulated pesticide products. These falsely named ingredients include solvents, emulsifiers, and other substances added to a pesticide product to make it easier to blend or apply or for any other reason not directly related to killing a target pest. So-called "inert" ingredients may have serious negative health effects, and some are even used as pesticides in other products. Although they often make up over 95 percent of the formulated product, the true identity of "inert" ingredients is classified as "confidential business information" and kept secret from both product users and the public. The result is that no one has any idea of what chemical combinations they are being exposed to when they come in contact with pesticides.

CHRONIC HEALTH IMPACTS

Many pesticides are known to cause chronic effects in people, laboratory animals, and/or wildlife. Such effects include many types of cancers, neurological effects, reproductive and developmental illness, and disruption of the endocrine system. Whether subtle or drastic, the pesticide origins of these long-term health impacts are more difficult to prove than are acute poisonings. While not comprehensive, this section presents summary information about several types of chronic pesticide health effects.

Pesticides and Cancer. Many pesticides used in agriculture and in homes, gardens, buildings, and public spaces are linked to different kinds of cancers. According to the Environmental Protection Agency (EPA), 112 currently registered pesticides are known, probable, or suspected carcinogens. Pesticides can increase cancer causation through several mechanisms, including by promoting abnormal cell proliferation, directly altering DNA, or disrupting the immune system. Evidence linking pesticides to cancer comes from three major sources: human epidemiological investigations, studies performed on laboratory animals, and cell-culture studies. The following examples emphasize epidemiological studies:

- The agricultural and home-use weed killer 2,4-D has been associated with malignant melanoma in several studies. One study in the *Journal of the American Medical Association* found that farmers who mixed or applied 2,4-D more than 20 days per year had a six times higher risk of non-Hodgkins lymphoma.
- Overall incidence of childhood leukemia in the United States increased by 27 percent between 1973 and 1990. One National Cancer Institute study found that in homes where pesticides were used even just once a week, children's risk of leukemia increased 400 percent. Other studies show that children whose fathers work in jobs that expose them to pesticides have a threefold increased risk of leukemia.
- Use of the pesticide lindane has been linked with aplastic anemia.

One study found that use of lindane shampoos to treat head lice is associated with higher incidence of aplastic anemia in children. Lindane has also been linked with lymphoma and breast cancer in adults.

- Childhood brain cancer has increased by 33 percent in the past 20 years; risks of childhood brain cancer were found to be elevated two- to sixfold in homes where pesticides are used. One study in *Environmental Health Perspectives* found brain cancer rates to be five times higher in homes where "no-pest" strips were used and six times higher in homes where pets wore flea collars.

Neurological and Behavioral Effects of Pesticides. As mentioned, pesticides that affect the nervous system cause more acute poisoning cases than any other pesticide category. But these pesticides also have serious long-term effects on both the central and the peripheral nervous systems. Many years after the fact, large numbers of people who have suffered serious acute organophospate poisoning have significantly impaired hearing, vision, intelligence, coordination, reaction time, memory, and reasoning. Cognitive symptoms of chronic damage to the nervous system include personality changes, anxiety, irritability, and depression. A growing body of evidence indicates that Parkinson's disease may be linked to exposures to certain pesticides and pesticide classes, among other environmental factors. Specific chemicals implicated in this particular type of damage include the herbicide paraquat, the organophosphates, dieldrin (an organochlorine), and the fungicides maneb and mancozeb. Other herbicides and insecticides also appear to be associated with development of Parkinson's disease. Several fumigants, including methyl bromide, Telone, and sulfuryl floride, are linked with a range of behavioral and cognitive effects. As they are with other health effects, children are particularly vulnerable to chronic neurotoxins, and exposures during key periods of brain growth can result in permanent effects on the structure and function of their brains.

Reproductive and Developmental Effects. Pesticides can damage men's and women's fertility by affecting their reproductive organs directly or indirectly, or by disrupting the normal functioning of their hormones. Fertility can be impaired by occupational exposure to pesticides, as indicated by increased time-to-pregnancy documented in spouses of farmers and agricultural workers and other types of studies in North America and Europe. It may even be destroyed forever, as hundreds of men exposed to the pesticide DBCP in the United States, Central America, and Africa have learned to their deep and lasting sorrow. Widely used pesticides that are known to be reproductive toxins in men, women, or both include the herbicides 2,4-D and chlorosulfuron, the rodenticide 1080, the insecticides oxydemeton-methyl and hydramethylnon, and the fungicides benomyl, myclobutanil, and triadimefon.

Most pesticides can cross the placenta and enter the body of a fetus. Developmental effects of pesticides can include spontaneous abortion, stillbirth, birth defects, low birth weight and smaller infants, and functional impairment. Many studies show that mothers' occupational exposure to pesticides increases risks of congenital birth defects. Others demonstrate that increases in a variety of birth defects are associated with fathers' employment as pesticide applicators. The timing of exposure can be critical: the periods of fetal development and early childhood, in which the body's organ systems are formed, are especially vulnerable times for this type of health effect.

Endocrine Disrupting Pesticides. Many pesticides can disrupt normal functioning of the endocrine system in people and other animals. Such pesticides may strengthen or weaken, imitate or block the effect of naturally occurring hormones, leading in turn to serious problems, including cancer, reproductive illness, or developmental effects. Most pesticides have not yet been studied for their potential to affect hormones or otherwise disrupt the endocrine system, and the tests capable of detecting such effects are still being developed. Pesticides that have been identified as having this type of effect so far include the popular weed killers atrazine, alachlor, cyanazine, and simizine; the insecticides aldicarb, carbaryl, lindane, endosulfan, resmethrin, and other synthetic pyrethroids; the fungicides vinclozalin, metiram, benomyl, mancozeb, and maneb; and the wood preservative pentachlorophenol. Many pesticides that persist for long periods in the environment are known to be endocrine disruptors, including DDT, aldrin, endrin, dieldrin, chlordane, heptachlor, and other organochlorine insecticides.

PESTICIDE POISONING AND CHILDREN

Children are more susceptible to the acute and chronic heath effects of pesticides than adults are for several reasons. Because their bodies and organs are still growing and developing, children's bodies do not process these poisons as well as those of adults. Children are also more exposed to pesticides. Pound for pound, children eat more food, drink more liquids and breathe in more air than adults, so they take in more pesticides per unit of body weight than adults do. Because they are smaller, children's bodies have a relatively greater surface area in contact with the world then adults do, and most pesticide exposures occur through the skin. Children also have more contact with pesticides and other environmental toxins because they crawl around on all kinds of surfaces, often put their hands in their mouths, hug pets more frequently, and generally are in more intimate physical contact with the world than are adults. Children in agricultural settings face particularly high risks. Children ten years and older may work legally on farms, and younger children of farmworkers often join older family members in the fields out of economic necessity. As a result, farm kids often have much higher exposures to pesticides than other children do.

There is some evidence that acute poisoning of children tends to be noticed and treated more readily than occupationally related poisonings, especially when caused by swallowing a pesticide, a spill, or some other specific event in the home. Reports from the national network of Poison Control Centers show that more than 50 percent of pesticide poisoning emergencies reported in the United States each year involve children less than six years old. Poisonings that occur away from home are less likely to make it into the official record.

U.S. AND GLOBAL POISONING ESTIMATES

Although acute pesticide health effects, which occur within moments or days of exposure, are more easily identified than chronic poisonings, most acute agricultural poisonings go unrecognized or unreported. There are many reasons for this. Many symptoms of acute poisonings (e.g., headache, nausea, dizziness, diarrhea, vomiting, and skin rashes) are also associated with other common conditions, making accurate diagnoses difficult even when health care professionals are informed enough to consider pesticide poisoning as a possibility. The fact that most of the world's agricultural workers have no access to health care obviously contributes to a lack of reliable data on agriculture pesticide poisonings. Furthermore, many farmworkers fear being fired or getting labeled as troublemakers if they seek medical help or take time off work to recover when poisoned by pesticides on the job.

Where reliable numbers are unavailable, educated guesses become increasingly important. In the United States, government estimates indicate more than 20,000 farmworkers out of an estimated population of 5 million workers in this country suffer acute pesticide poisonings annually. Yet authorities also acknowledge that their estimates are based on very little knowledge regarding the extent of actual pesticide exposures and resulting health effects. In terms of chronic impacts, no serious effort to develop estimates of annual cases has been attempted.

At the global level, the World Health Organization published an estimate in 1990 that 3 million severe acute pesticide poisonings occur in developing countries each year, including some 220,000 fatalities. This figure is still widely cited today, although another study by the same expert indicates it is a serious underestimation. Based on hospital records in four Asian countries, this expert concluded that between 2 and 7 percent of the agricultural labor force in developing countries is poisoned annually, which would revise his previous estimate upwards to well over 25 million poisoning cases each year in developing countries alone.

Another more in-depth field study of 228 farmers and pesticide sprayers in Indonesia found that 21 percent of all pesticide applications over the study-

season resulted in symptoms that strongly indicated organophosphate pesticide poisoning. Asked if they remembered ever having been poisoned by pesticides, 9 percent of the farmers reported at least one incident serious enough that they sought medical attention. The study noted that the farmers "tended to accept this level of illness as part of the work of farming." Most of the farmers also reported pesticide storage, disposal, and other practices that put their family members at risk.

Translating these figures into a fictional nonagricultural setting helps to highlight the social and economic assumptions that allow such astounding rates of occupational hazard to persist without consequences to the suppliers of the injurious product or adoption and enforcement of regulatory measures sufficient to reduce the rate of injury. Consider the following: word processors are basic tools for many firms and industries, and millions of people rely on them for personal uses as well. Now imagine that 21 percent of the time you, or anyone else, used a word processor at work you would receive an electrical shock. That's on average, so it wouldn't be every time, and the shock wouldn't be enough to kill you — at least not most of the time. Then imagine that nearly 10 percent of the people using word processors got shocked severely enough to require medical treatment at some point in their careers and that their families were at risk from the word processors that they kept at home.

Does it seem reasonable for you to be forced to accept being shocked repeatedly as "part of the work of word processing"? Or that the computer company whose products kept shocking you should be allowed to stay in business?

THE BENEFITS OF CHRONIC UNCERTAINTY

In the early stages of learning about pesticide dangers, many people get frightened or overwhelmed and don't want to know more. This is easily understandable. Professionals deeply familiar with pesticide health effects also may numb themselves to the pain and suffering they encounter in laboratory animals, wildlife, and men, women, and children exposed to pesticides in order to stay focused on the task at hand. But overwhelmed individuals and psychic numbing among experts should not prevent public acknowledgement of massive, unnecessary proliferation of dangerous pesticides into the air, water, food, and public spaces we all share.

Chronic pesticide poisonings provide an instructive case in point in considering factors that blunt public awareness of pesticide damages. No one disputes that such poisonings occur, but the extent, frequency, significance, and implications of these poisonings are endlessly controversial. From a public health and welfare perspective, acting to reduce use of hazardous

chemicals, getting them off the market, and replacing them with less or non-hazardous alternatives is the obvious and most effective way to address individually the damage — impossible to prove but collectively very real — caused by chronic pesticide poisonings. The same course of action flows easily from an environmental frame of reference.

But somehow, this is not what happens. Instead, industry scientists, regulators, pesticide users, and public interest groups all agree that chronic pesticides are health hazards, but disagree on how hazardous and what to do about it. This is where you start to see the qualifying phrases stack up in both industry and government regulatory positions: yes, they are hazardous . . . *but* they can be applied in a safe and harmless manner when applied according to label instructions. *But* our research indicates that this product presents no significant hazard to the public. *But* by controlling the exposure, we can control the risk, and the exposures are at safe levels. *But* alternatives are not available or cost effective. *But* we don't know enough about the extent of harm to justify taking "extreme measures" (code for removing a product from the market). *But* the harm done (to many) is outweighed by the economic benefits (to increasingly few) of using the pesticide.

Driven by such assertions, which are rarely if ever subject to open scrutiny, scarce public funds and the greater resources of industry are spent documenting that long latency periods, confounding exposures, and other factors make it difficult to estimate individual and aggregate exposures or quantify risks from chronic pesticides. This is true. Yet such "insights" do little to prevent further damage or develop alternatives to more pesticide use. In this intentionally endless quest for greater knowledge, new studies are designed to better understand a pesticide's mode of action, establish clearer causal relationships, identify so-called "safe" exposure levels, quantify the extent of harm more precisely, etc. Meanwhile, serious measures to reduce and eliminate the source of harm never make it onto the list. As the wheels of investigation grind on, uncertainty is "resolved" in favor of pesticide manufacturers and users, who continue to develop their plans and project future profits based on continuing use of chronic poisons.

PESTICIDES IN THE ENVIRONMENT

Ultimately, most pesticides in the environment degrade upon exposure to air, sunlight, and water, or as they are broken down within plants, animals, and microorganisms. How long this takes and how much damage is done in the meantime varies greatly from case to case, however. Different types of pesticides break down differently, and while the chemical breakdown products of these processes are usually less harmful than the original material, some are even more dangerous than the parent compound, as in the cases of the insecticides aldicarb, malathion, and ethyl parathion, and the herbicide atrazine.

How pesticides break down in the environment is also influenced strongly by temperature, moisture, presence or absence of other chemicals, and many other factors. But predicting the environmental conditions of where a pesticide may end up is no simple matter. Pesticides are highly mobile and can travel vast distances. Once released into the environment, they are like genies let out of their bottles — impossible to put back in. Pesticides applied by aircraft can drift many miles from their supposed targets, evaporating in and out of a solid state within air currents, only to land, revolatilize, and set off again. Tiny pesticide droplets suspended in fog can be deposited onto birds, wildlife, leaves, and any other living or nonliving surface touched by the mist. Rain, storms, and irrigation ditches routinely sweep huge loads of pesticides into streams, lakes, wells, and rivers, with often devastating effects on fish, amphibians, and aquatic invertebrates. Pesticides cross the seas on prevailing currents to contaminate Arctic and Antarctic environments, native peoples, and the animals they depend on for sustenance, thousands of miles from the original application. They also move through aquifers and groundwater, to the horror of those who depend on these sources for their drinking water.

Another way pesticides travel is through food chains. In this mode of transport, pesticides that are taken up within smaller organisms and not broken down or excreted remain stored there until the organisms are eaten by another creature, whose body burden of pesticides increases accordingly. The same thing happens again when that creature becomes a meal for another predator, and so on and so on. These pesticide body burdens travel with their "hosts," and migratory animals such as marine mammals, birds, and fish often carry pesticides over long distances before being eaten by predators.

The continuing process of adding and passing on new loads of pesticides and other toxins through the food chain is called bioaccumulation, and it is the reason that top predators like birds, sharks, some whales, bears, and people carry high concentrations of certain poisons in their bodies. Breast-feeding infants of mammalian predators, including human babies, are at the pinnacle of the food chain, since large amounts of bioaccumulating chemicals collect in breast milk and are passed into the infants' bodies as they feed.

PERSISTENT ORGANIC POLLUTANTS

One particular type of pesticide combines several characteristics that make it a special threat to life. Persistent Organic Pollutant pesticides (POPs) — such as DDT — are linked with serious chronic health effects; they last for long periods without breaking down; they travel far and wide in the environment; and they build up to ever higher and more harmful levels in the food chain. Since their widespread production and use began, less than 60 years ago, POPs pesticides and other POPs chemicals have moved throughout the global environment to threaten human health and ecosystems around the world. All living organisms on earth now carry measurable levels of POPs in their tissues, and evidence that exposure to even tiny amounts of POPs during critical periods of development can cause irreversible damage is strong and increasing. Effects of such exposures can take years to appear, sometimes appearing first in the offspring of exposed parents. In this tragic legacy of damage, children can end up suffering from a parent's exposure to a POPs chemical that occurred decades before they were born.

In an encouraging example of coordinated action to reduce chemical hazards, nations around the world recently recognized and began addressing the extraordinary threat of POPs chemicals with an international treaty. The new POPs treaty mandates global phase-outs of production and use of POPs chemicals. This type of global approach to eliminating chemical damages is both inspiring and much too rare.

WHAT LIES BENEATH

The fact that pesticides continue to be promoted and accepted as the most efficient and desirable form of pest management is a symptom of a different kind of chronic poisoning. Driven by economic policies that put short-term profits and agricultural exports first, and address health and social concerns only later, if at all, extractive, chemical-intensive industrial agricultural is gaining ground despite our greenest intentions and desires.

The approximately $35-billion-a-year pesticide business lies at the center of these expansive lies. Dominated by ten corporate giants based in the United

States and Western Europe that control nearly 90 percent of the global pesticide market, this industry is directly (but not solely) responsible for the release of several billion pounds of pesticides into the environment every year. And that's just one piece of the agro-industrial complex. Increasing use of pesticides and other harmful agrochemicals, despite their negative health and environmental impacts and the sustained growth of the organic sector, underscores the power of these industries to thwart attempts toward biologically based pest management and ecological agriculture. Continuing public confusion regarding the true extent of pesticide damages, weak national regulatory and enforcement systems, and a pervasive lack of public investment in already existing and promising new alternative pest management approaches are additional symptoms of these industries' poisonous influence.

Ironically, many people believe that agriculture is gradually giving up its dependence on pesticides. Agrochemical and related industries' investments in marketing, public relations, and political campaigns help explain this misperception. For example, most people mistakenly believe that when pesticide producers proclaim their environmental commitments, this means they are reducing production of environmentally harmful materials. Many people also believe that because organic agriculture is making such rapid gains, levels of pesticide use must be falling as well — and national regulatory authorities neither collect nor publish pesticide-use data showing that exactly the opposite is true. People also assume that pesticides on the market must be safe, or pesticide regulatory agencies would not allow manufacturers to sell them, reflecting a widely held but dangerously inaccurate understanding of these agencies' role. And of course psychic numbing and feelings of being overwhelmed also help shield corporations and governments from scrutiny.

REGAINING GROUND

Acknowledging such barriers and the power of corporate interests to maintain them in no way implies that our societies can never awaken from the health and environmental nightmares of conventional industrial extractive agriculture. Rather, it points directly to the need for multiple and reinforcing strategies of public education, analysis, and actions over time. To be effective, these strategies must facilitate the development of new leadership and other resources needed to transform agricultural policy and practice. They must also address the social contexts in which massive unnecessary pesticide damages are considered "normal" and acceptable and in which companies responsible for these damages are rewarded for inflicting them.

Changing how our societies deal with the uncertainty that surrounds the hidden dimensions of pesticide damage is an important element of stemming the rising toxic tide of pesticides. When protecting people and the environment from pesticide harm, it is not reasonable to require iron-clad

scientific proof or multi-stakeholder consensus that a pesticide causes a certain number of deaths or percentage of cancers or other types of health effects before taking action to reduce harm. Even where our knowledge of the mechanisms and extent of damage is not complete, awareness of harm should automatically trigger actions to protect the health of our families, communities, and environment.

Protecting health in highly contaminated and otherwise compromised environments is extremely difficult, and mitigating and healing damages to health from such contamination is generally prohibitively expensive and often impossible. That is why preventing the release of harmful chemicals and other forms of environmental contamination is the most effective, economical, and morally justifiable approach to safeguarding people and ecosystems from costly and often irreversible damages, such as those described here. Acceptance of this straightforward approach is guiding efforts toward cleaner production in several industries and gaining credibility in some nations, most notably in Scandinavia. Although increasing global pesticide sales and marketing of new genetically engineered pesticides show that the conventional agriculture industry has yet to embrace this precautionary approach, those of us convinced of the wisdom of moving toward cleaner production in agriculture have much to work with.

Knowing that we need to move toward an agriculture capable of supplying the foods and fibers we need without destroying people's health, environments, and cultures in the process helps us target our efforts. Since preventing pest problems is essential to healthy and successful agriculture, for example, we know we must figure out much better ways to do this than reflexively using pesticides. Similarly, preventing pesticide-related damage to health implies rapid elimination of pesticides known or suspected to cause such damage. This means we need effective mechanisms for targeting major uses of hazardous pesticides, removing those products from the market, and replacing them with safer alternative approaches.

Fortunately, many proven alternative methods and products are available to reduce our current massive dependence on pesticides, and more are becoming available with time. Where such alternatives already exist, we must move far more quickly to implement them. Wherever they are not available, we need to move urgently to apply the human creativity, financial resources, and other support to ensure they are developed and implemented as rapidly as possible.

Meanwhile, the secrecy and misinformation surrounding the true scale of pesticide use in agriculture remain a huge obstacle to the development of safer pest management alternatives. Think of the billions of pounds of pesticides being released into the environment each year as straws being loaded onto camels' backs. We and our families, communities, and environment are the

camels, and the burdens we bear are packed for us by experts who swear they are essential to keeping us fed and are otherwise not a problem — and who make a commission on every straw we carry.

All of us have the right to know what pesticides we are exposed to intentionally or unintentionally, and to be heard in decision-making processes that affect whether or not these exposures continue. In addition to ensuring our right to know, public reporting and disclosure of pesticide use is also crucial to creating effective demands for safer alternatives. The same corporate advertising expertise that helped create chemically dependent agriculture now churns out messages telling us about the greening of agriculture. These messages are all the more easily swallowed because they are partially true — despite industrial agriculture's stalling tactics and thanks to the ceaseless efforts of a small but growing number of farmers leading the way to more ecologically and socially beneficial agricultural systems.

Separating agricultural fact from profitable fantasies requires more information than the public is presently allowed access to. Using new information technologies, it could be easy for any man, woman, or child to find out whether the use of specific pesticides on specific crops in specific places is going up, down, or staying the same in their county, state, and country. Other questions that should be easily answered include whether public and private funds dedicated to research and extension programs designed to reduce pesticide use are increasing, and where to find detailed information about farm ownership and the ecological and labor conditions under which food and fibers are grown and processed. Without constant public tracking of these and other indicators of progress toward ecologically based agricultural production, all we have is assurances from people whose words we know from experience cannot be trusted without independent verification. ❖

As the use of pesticides increases, so does the rate of breast cancer. According to the National Cancer Institute, 50 to 60 percent higher levels of organochlorine pesticides are found in the breast tissue of women with breast cancer than in the tissue of healthy women. Yet the cancer-establishment has actively denied the connection to pesticides, no doubt due to its own involvement in the petrochemical industry. No wonder that surgery is considered a remedy for cancer, while reducing pesticide use is not.

I. DDT AND OTHER CHEMICALS USED IN VECTOR MANAGEMENT PROGRAMS

A Brief History

DDT (dichlorodiphenyltrichloroethane) is an organochlorine insecticide used mainly to control mosquito-borne malaria. DDT's insecticidal properties were discovered in the 1930s by Swiss chemist Paul Müller. Considered harmless to mammals this odorless, tasteless, white crystalline chemical was used during the Second World War for crop protection as well as protection of troops from malaria and typhus. DDT's characteristics of insolubility in water, persistence, long half-life of 10-35 years and high-contact toxicity made it appear to be the ideal insecticide. As a consequence, Müller was awarded the Nobel Prize in 1948. Only a few years later, Swiss scientists confirmed the connection between unborn and functionally-impaired calves whose mothers had been grazing on pastures that had been sprayed with DDT. Previously, U.S. agricultural researchers had linked similar severe impairments in calves whose mothers had been eating feed salted with DDT for pest control (IEM on POPs, Annex II). Still others had found that young roosters treated with DDT had severely underdeveloped testes and failed to grow the normal combs and wattles roosters use for social display (Colborn *et al.*, 1996).

Regardless of these effects, DDT's efficacy and low-production costs made it the most widely used agricultural insecticide in the world from 1946 to 1972. Total world production of DDT during this period has been estimated from 2.8 million tonnes to more than 3 million tonnes (IEM on POPs, Annex II).

The effects of DDT on wildlife reproduction and its residues appearing in food products that had been sprayed with DDT became evident in the 1960s. Long term studies showed that DDT was found at alarming levels in many animal species including fish, birds, and mammals. Many birds such as peregrine falcons, California condors, and bald eagles with high levels of DDT in their bodies began producing weak eggshells, which were crushed upon incubation. The result was a decline in the bird populations and a threat to their very existence. These findings led to DDT use restrictions and bans in the U.S., Canada, and most European countries in the early 1970s. DDT is now banned in 34 countries and severely restricted in 34 (IEM on POPs, Annex II).

Insecticides Currently in Use

The World Health Organization (WHO) approves use of DDT in controlling malaria, provided several conditions are met, including limiting its use to indoor spraying, taking appropriate safety precautions, and using materials that meet WHO specifications. Four major groups of insecticides are available for indoor spraying: organochlorine chemicals (DDT), organophosphates, carbamates, and the synthetic pyrethroids (Table I-1). The undesirable effects of DDT are widely known; they have driven the restrictions on DDT that have occurred to date and are responsible for DDT being targeted in international POPs negotiations. The organophosphates and carbamates are acutely toxic to humans, and pose a high hazard in particular to those who work with them (Herath, 1995). The synthetic pyrethroids are not as toxic as the carbamates or organophosphates, and are widely used as an alternative to DDT or used to impregnate bednets. Because most reports of wide-scale applications of pesticides for vector control involve DDT or the synthetic pyrethroids, the discussion that follows focuses mainly on these pesticides.

Table I-1: Vector-Control Insecticides and Their Known Health Effects

Health effects modified from Lars et al. (1996), WHO Environmental Health Criteria or Health Safety Guides, EXTOXNET-Extension Toxicology Network, Co-operative Extension Offices of Cornell University, The University of California, Michigan State University, and Oregon State University. (Listing adapted from Chavasse and Yap, 1997)

Insecticide	Type	Indoor	Bednets	Toxicity to Humans/Mammals	Environmental Toxicity
Bendiocarb	C	+		High acute toxicity	Moderate-high toxicity to fish, birds, crustaceans; neither bioaccumulative nor persistent
Propoxur	C	+		Very high acute toxicity; suspect carcinogen, mutagen	Moderately persistent; very high acute toxicity to birds, fish, bees, crustaceans; low bioaccumulation
DDT	OC	+		Possible carcinogen	Highly toxic to fish and aquatic species, moderately toxic to birds and mammalian species; eggshell thinning in birds; very persistent and bioaccumulative
Chlorpyrifos	OP	+		Medium-high oral toxicity, dermal and inhalation; delayed neurotoxin; sterility and impotence	Moderately persistent in soil; high-very high acute toxicity to birds, molluscs, crustaceans, bees; long-term effects on reproduction/growth of fish; bioaccumulative in aquatic organisms
Fenitrothion	OP	+		Moderate - high acute toxicity	Moderately bioaccumulative; high toxicity to some birds; high toxicity to crustaceans; not persistent
Malathion	OP	+		Medium-high acute toxicity; suspect mutagen; delayed neurotoxin	Not persistent; high toxicity to birds, fish crustaceans; highly toxic to bees, amphibians
Bifenthrin	SP1		+	Very high acute toxicity; suspect carcinogen, mutagen	Moderately persistent; very high acute toxicity to fish, crustaceans, aquatic invertebrates
Cyfluthrin	SP2	+	+	High acute toxicity	Moderately bioaccumulative; highly toxic to fish, crustaceans, and bees
Cypermethrin (includes α)	SP2	+	+	Moderate-high toxicity	Highly toxic to aquatic invertebrates and fish
Deltamethrin	SP2	+	+	High acute toxicity	Moderately bioaccumulative; highly toxic to fish, crustaceans, and molluscs
λ-cyhalothrin	SP2	+	+		Highly bioaccumulative; highly toxic to fish and crustaceans
Permethrin	SP1	+	+		Highly toxic to aquatic arthropods and fish

Key: C = carbamate; OC = Organochlorine; OP = Organophosphate; SP = Synthetic Pyrethroid (Type 1 or Type 2)

Chemical Properties

DDT is available in several different forms: aerosol, dustable powder, emulsifiable concentrate, granules, and wettable powder. Technical grade DDT is actually a mixture of three isomers of DDT, including the p,p'-DDT isomer (85%) with the o,p'-DDT and o,o'-DDT isomers present in much lesser amounts (ATSDR, 1994). The content of these isomers is important because the o,p'(ortha-para) isomer is said to be five to nine times less toxic in tests with rats than the p,p'(para-para) isomers. While DDT is highly resistant to degradation, some microbes can degrade DDT into a variety of metabolites. Among the more important of these is DDE and TDE (DDD). The latter is also manufactured as a commercial product (IEM on POPs, Annex II).

Persistence and Transport Characteristics

At present, most of the millions of tonnes of DDT that have been produced in the past continue to be transformed and redistributed throughout the environment. DDT and its metabolites have been detected in virtually all media throughout the world. An extremely stable chemical compound, 50 per cent of the DDT sprayed on a field can remain in the soil 10 to 35 years after its last application. For example, an Oregon (U.S.) orchard still had 40 per cent of the original DDT used 20 years later. DDD has also been shown to be even more persistent in soils, sediments, and waters, lasting 190 years and longer (IEM on POPs, Annex II).

These compounds do not remain in the soil, but are transported into the general environment by the processes of volatilization, through wind and water erosion. Although more than 20 years have passed since the last applications of DDT, soils in the southern U.S. cotton belt are estimated to be volatilizing 110 tonnes of DDT and its metabolites annually into the atmosphere. These small particles are transported long distances on air currents, and are returned to the land surface by precipitation.

DDT in the Arctic Food Web

There has been very little local use of DDT in the high arctic, therefore the presence of DDT in arctic biota is indicative of the global or hemispherical transportation of this compound. DDT has been found at various concentrations in all trophic levels of the arctic food chain. Table I-2 is a summary of DDT concentrations found in the lower trophic levels of the arctic marine food web. Table I-3 shows concentrations of DDT in the blubber of arctic mammals.

Table I-2: DDT Concentrations (ppb lipid wt.) in Marine Biota in Various Locations in High Arctic (adapted from Canadian Arctic Contaminants Assessment Report, DIAND)

Biota	Region	Total DDT
Epontic Particles	Ice Island	20-70
	Barrow Strait	150-360
Zooplankton	Ice Island	8-150
	Barrow Strait	2-20
Amphipods		
Pelagic	Ice Island	<350
Pelagic	Barrow Strait	3-60
Benthic	Arctic Ocean	2,200-25,900
Benthic	Barrow Strait	15-1,590

Biota	Region	Total DDT
Fish		
Arctic Cod	Lancaster Sound	66-120
	Barrow Strait	15-255
Turbot	Cumberland Sound	626-1,044
	Beaufort Sea	659-1,1251
Four-horn Sculpin	Wellington Bay	93
	Cambridge Bay	1,225
	Hall Beech	135
Bivalves		
Clams	Sanikiluaq	34
Septentrion sp	Manitounuk Sound	13

Table I-3: Mean Concentrations (ppb wet wt.) of Total DDT in Blubber of Arctic Mammals (adapted from Canadian Arctic Contaminants Assessment Report, DIAND)

Species	Female	Male
Ringed Seal	473	959
Harp Seal	486	NA
Beluga Whale	1,940	4,974
Narwhal	NA	3,232
Walrus	744	1,744
Adult Polar Bear (Bernhoft et al., 1997)	372	340

Water runoff provides another mode of transportation. DDT sticks to soil particles by the process of adsorption. These particles are transported to lakes and rivers and are the principal route by which lakes and streams become contaminated. In an experimental plot of cotton, runoff waters transported 2.8 per cent of the DDT applied in six months.

Under tropical conditions, residues continue to be detected in major water bodies in the Philippines despite DDT's restricted-use status. Fish, as well as duck eggs, from lake areas also show residues (IEM on POPs, Annex II).

While DDT will evaporate and photo-oxidize from soil surfaces to a certain degree, it is a robust and long-lived chemical compound. Even when its use is banned globally, DDT and its various metabolites will continue to travel in the winds and waters and accumulate in the bodies of the world's organisms for decades to come.

Bioaccumulation in Organisms

Bioaccumulation Potential

Bioaccumulation reflects the relationship between how much is taken into an organism by exposure versus how much is lost through metabolism and excretion. The key in pesticide exposure scenarios is whether the rates of metabolism and excretion remove enough of the substance to prevent a gradual increase in the organism. If the rates of metabolism and excretion are not rapid, an organism will accumulate ever-increasing concentrations, adding to the concern about chronic, low-dose exposures.

Chemicals that are water soluble are more easily excreted, as well as more easily mobilized to sites responsible for metabolism of the compound. On the other hand, a chemical with high solubility in lipids (fats, oils, or waxes) has bioaccumulation potential. Such lipophilic chemicals easily move into cells and

are sequestered in fat where they can become more persistent. DDE is an example of a lipophilic chemical that resists enzymatic degradation and, therefore, rapidly bioaccumulates. DDT is also lipophilic, however, it is more readily degraded and excreted from the body.

Bioaccumulation in the Great Lakes Food Web

DDT continues to be deposited in North America's Great Lakes basin despite restrictions on its use in the United States and Canada. It appears that much of the DDT currently being deposited in the basin is atmospherically transported from Central and South America.

Even though the concentration of DDT in plankton is 1/100 part per million, the flesh of a fish-eating bird in the same lake system may contain 630 times that concentration (Colborn *et al.*, 1990).

Table I-4: Bioaccumulation of DDT in Lake Ontario Food Web (Colborn et al, 1990)

Species	Concentration (ppm wet weight)
Plankton	0.01
Mysis	0.03
Pontoporeia	0.10
Sculpin	0.40
Smelt	0.40
Lake Trout	1.10
Herring Gull	6.30

Synthetic pyrethroids are also lipophilic though they are more like the isomers of DDT in that they can be metabolized to more water soluble forms that can then be excreted. Furthermore, the sites where they can be metabolized are not limited to the liver and therefore, metabolism is much quicker. For example, the elimination half-life for deltamethrin in plasma of the rat is 33 hours (Anadon *et al.*, 1996) with almost complete elimination from the body by day 4 (Ruzo *et al.*, 1978). Cypermethrin is more resistant to elimination; 90% is lost in the first four days, however, total elimination may take as long as 17 to 26 days (WHO Working Group, 1992).

Synthetic pyrethroids (permethrin, deltamethrin) are rapidly distributed in the body (Anadon *et al.*, 1991, 1996). The primary sites of deposition are the central nervous and peripheral nervous systems, which can have concentrations of permethrin ranging from 1.5 to 7.5 times higher than those observed in plasma. In another study, a single topical application of deltamethrin (0.75%), cypermethrin (10%), or cyhalothrin (4.5%) to dairy cows was detectable in both the cows' blood and milk for 28 to 35 days (Bissacot *et al.*, 1997). In these situations, bioaccumulation results in much lower peak concentrations since the differences between exposure and intake are not widely different from metabolism and excretion. The concern would be if the exposure is periodic, with a span shorter than the rates of excretion. Chronic, low-dose exposures may lead to slightly increased concentrations in the body. There is little known about the pharmacokinetics of the synthetic pyrethroids.

DDT Bioaccumulation in Humans

In surveys around the world of human blood, fat tissue, and breast milk, DDT and its metabolites are found in substantial quantities (Thomas and Colborn, 1992; Jensen, 1990). For example, Table I-5 reviews concentrations of two isomers that are known endocrine disruptors. Since DDT is very lipophilic, it accumulates in all fats, including the 3% fat

found in breast milk (Rogan *et al.*, 1986). The quantity of DDT and DDE varies with the age of the individuals with young individuals having higher concentrations than older individuals. This is probably the result of a combination of a pesticide-rich food source (breast milk) and a lower total body fat content in the baby. As the baby matures, fat accumulations increase the available pool which in effect dilutes the DDT/DDE enriched fetal fat reserve.

Table I-5: Concentrations of o,p'-DDT or p,p'-DDE (endocrine-disrupting isomers of DDT) in Breast Milk of Women (standardized to ppm fat)

Country	# Women	Year	o,p'-DDT	p,p'-DDE	Σ DDT	Citation
Canada	497	1995	0.003	0.22		Newsome et al., 1995
U.S.A, New York	7	1985-87	-	0.54	-	Schecter et al., 1989
Mexico, Veracruz	43	1994-95	0.27	5.02	6.44	Waliszewski et al., 1996
Mexico, Mexico-City	50	1994-95	0.14	0.59	0.93	Torres-Arreola et al., 1998
Germany	150	1985-87	-	0.75	-	Schecter et al., 1989
Spain, Madrid	51	1991	-	0.60	0.66	Hernandez et al., 1993
Norway	20	1988		0.97		Skaare et al., 1988
United Kingdom	193	1989-91	-	0.40	-	Dwarka et al., 1995
France	20	1990-91	-	2.18	-	Bordet et al., 1993
Slovakia	50	1994		1.20		Prachar et al., 1996
Yugoslavia,						
Krk Island	33	1986-87	-	1.10*	-	Krauthacker, 1991
Labin	20	1986-87	-	0.55*	-	Krauthacker, 1991
Croatia	50	1981-82		1.90		Krauthacker et al., 1986
Nigeria	10	1987		0.99		Atuma and Okor, 1987
Nigeria, Benin	35	1981-82	-	1.1	-	Atuma and Vaz, 1986
Kenya	68	1983-85		1.73		Kanja et al., 1986
New Guinea, Papua	41	1990	-	0.45	0.89	Spicer and Kereu, 1993
Uganda	143	1992-93	0.06	2.35		Ejobi et al., 1996
Zimbabwe, Kariba	39	1994		13.60	25.26	Chikuni et al., 1997
Australia, Victoria	60	1995		0.96		Quinsey et al., 1995
India	60	1985-86	1.43	7.28		Zaidi et al., 1989
India	25	1988		2.00		Tanabe et al., 1990
Jordan, Amman	15	1989-90	0.23	2.04	3.31	Alawi et al., 1992
Saudi Arabia	115	1995-96	-	-	0.27	Al-Saleh et al., 1998
Turkey	104	1995-96	-	2.01	2.36	Cok et al., 1997
Thailand, Bangkok	3	1985-87		3.61		Schecter et al., 1989
Vietnam	7	1985-87		6.70		Schecter et al., 1989

Note: Methodologies used to quantify isomers varied, however they allow for comparisons of geographical differences.
* Median

A breast feeding baby can acquire concentrations of lipophilic chemicals at extraordinary rates. Mes *et al.* (1984) estimated that babies could acquire 1.8 micrograms of p,p'-DDE per gram body fat (or 1.8 ppm) by the 14th week of breast feeding from breast milk alone. Furthermore, the infant's DDT levels could reach those of the mother in the first three months of breast feeding. The levels of DDT in the blood begin to decline at about 3 years of age, again probably reflecting the shift in diet to a less contaminated food, and an increase in new fats.

Based on observations in South Africa of DDT (and DDT derivatives) in breast milk, Curtis (1994) estimated that a baby fed entirely by breast milk exceeds the allowable daily intake (ADI) for DDT (0.02 mg/kg), as determined

by FAO/WHO (1985), by 5 to 18 times. Rogan and Ragan (1994) estimated that over a nine-month period of breast feeding, an infant can acquire 21.5 mg of DDE based on the 90th percentile level. Such estimates identify breast feeding as a principal source of exposure to DDT and DDE. It must be pointed out that this exposure of the newborn coincides with development of their brains (Eriksson, 1997), so such exposure has implications for neural development, behaviour, and susceptibility to insecticides later in life (Johansson *et al.*, 1996). This concern for exposure during early development will be discussed in more detail later in the paper.

The DDT and DDE a nursing baby acquires from breast milk come directly from the low background exposures and accumulation over the years in the mother. In one study, levels of DDE were found to be 17% lower in women who had breast fed previously as compared to those who had not. This was confirmed in studies of women who were followed through two pregnancies, where there was a 23% difference in DDE levels between their first and second child.

The quantity also changes with the length of the nursing period – declines in DDE in milk of 20% at six months, and a 40% decline by the 18th month of nursing (Rogan *et al.*, 1986). The amount of DDT and DDE in the bodies of the women participating in this study was the result of low background exposures beginning early in their lives. It was not the result of accidental or agricultural exposures.

The persistence of lipophilic chemicals is cause for concern because exposure to low concentrations over an extended period of time may lead to substantial burdens later in life. In an analysis of DDT exposures associated with indoor application of DDT for malaria control in KwaZulu, Africa, Bouwman and colleagues (1991, 1993, 1994) found that household members in regions that used indoor pesticide applications for vector control had significantly higher DDT levels in their sera than those in regions where no spraying occurred.

Lactational transfer is not limited to DDT or DDE. Any pesticide that enters the body can be excreted in breast milk (Rogan and Ragan, 1994). Synthetic pyrethroids have also been reported in the milk of dairy cows when pesticides were applied as a part of an ectoparasite control program. Deltamethrin, cypermethrin, or cyhalothrin were reported in milk within 24 hours of application (Bissacot and Vassilieff, 1997), with concentrations of 0.51, 0.36, and 0.19 ppm, respectively. The organophosphate pesticide, chlorfenvinphos, another topical treatment for ectoparasites in cattle, was recorded to vary from 1.18 to 10.40 ppb in milk from cows in Kenya (Kituyi *et al.*, 1997). Only recently has attention been focused on the transfer of synthetic chemicals in human or cow milk, and therefore, it is not known what the magnitudes of transfer are for commonly used pesticides.

The fact remains that this lactational transfer is the rule of pharmacokinetics of synthetic chemicals, and not the exception. The concern increases as the transfer rate and concentration of the chemicals increases, which is related to both the application rate, frequency, and the bioaccumulation potential of the chemicals.

DDT in the blood of Indians has been found to be 10 times more than in most other countries. In Delhi it has been found to be 26 ppm on an average – well above the MRL of 1.25 ppm – the highest in the World.

These residues are far more dangerous when combined with a low-protein diet as in the case with the majority of Indian population .....

Inadequate Testing of Pesticides

A new study in the journal of Toxicology and Industrial Health identifies significant shortcomings in toxicological testing protocols currently used to register pesticides in the United States. The five year study, released in March 1999, suggests that combinations of commonly used agricultural chemicals in concentrations that mirror levels found in groundwater can significantly influence immune and endocrine systems as well as neurological health.

"The single most important finding of the study is that common mixtures, not the standard one-chemical-at-a-time experiments, can show biological effects at current concentrations in groundwater," said Warren Porter, lead author and University of Wisconsin professor of zoology and environmental toxicology.

The experiments performed by Porter's group suggest that children and the developing foetus are most at risk from pesticide-fertilizer mixtures. Their influence on developing neurological, endocrine and immune systems portend change in the ability to learn and in patterns of aggression. *(See Box: Household Pesticides and Childhood Leukaemia)*

Chronic Effects in U.S. Farmworkers

Despite the fact that millions of farmworkers in the U.S. are exposed over extended periods of time to multiple pesticides, few studies have addressed the relationship between exposure and subsequent illness in this population. Although very limited data are available, studies which have been conducted show disturbing evidence of chronic effects of pesticide exposure among farmworkers. The following is a brief summary of some of the findings of studies on farmworkers done in the U.S.

Cancer: One cancer study conducted in the USA in 1993 found that when compared to the general population, both farmers and farmworkers have increases in multiple myeloma and cancers of the stomach, prostate and testis. In addition, farmworkers show unique increases in cancers of the mouth, pharynx, lungs and liver.

Birth defects and stillbirths: Although increased numbers of birth defects have been recorded among farm area residents, very few studies have looked at birth defects among farmworkers. In one study of 990 single births, limb reduction defects occurred among offspring of agricultural workers three to 14 times more frequently than among the general U.S. population. The risk was greatest for mothers residing in countries with high agricultural productivity (2.4 times) and high pesticide use (3.1 times). In another study, occupational exposure of pregnant women to pesticides during the first and second trimesters increased the risk of stillbirths and early neonatal deaths by 5.5 and 4.8 times respectively, compared to unexposed groups.

Developmental effects: Many pesticides are known to disrupt the human endocrine system. The endocrine system is a complex array of glands, organs and tissues that secrete hormones (chemicals produced by the body) into the bloodstream and regulate a range of physiological and neurological systems. Reproductive organs appear to be at particular risk for development abnormalities when pregnant women are exposed to endocrine-disrupticy chemicals (EDCs). In both sexes, the brain, thyroid, liver, kidney and the immune system are also potential targets for EDCs. Since EDCs persist in body fat, they may also exert their effects long after exposure.

Thus even with limited data available a startling picture emerges of the dangers facing farmworkers.

Source: "Fields of Poison, California Farmworkers and Pesticides", by Margaret Reeves and Kristin Schafer (Pesticide Action Network North America), Kate Hallward (United Farm Workers of America) and Anne Katten (California Rural Legal Assistance Foundation), Californians for Pesticide Reform (CPR) Series, 1998.

Household Pesticides and Childhood Leukemia

The use of pesticides in homes is generally increasing. They are used as indoor pest controllers, on indoor plants and in home gardens. However, exposure to some of these pesticides, particularly exposure before birth (foetal exposure) increases the risk of children developing leukemia, according to a recent study.

A comparative study of the pesticide exposure background of nearly 500 children with (acute lymphoblastic) leukemia and a similar number of children without the disease by a group of researchers from McGill University in Montreal, Canada, has shown that "indoor use of some insecticides ... and pesticide use in the garden and on interior plants increased the risks up to several-fold". The study was published in the journal, 'Epidemiology', in September,1999.

Sourcing the paper in the journal, 'The Sun', Malaysia, recently reported (1): "According to the authors, the use of insecticides in the garden and inside the house, particularly frequent pre-natal exposure, was associated with increased risks of leukemia. For example, foetal exposure to household cockroach, ant and/or wasp-fighting compounds during pregnancy increased a child's risk of developing leukemia by 79 per cent, the investigators report, compared with children without such exposure. The researchers also noted that foetal exposure to moth-killer compounds was associated with more than double the risk for childhood leukaemia. "Household insecticides used in the study included compounds such as organophosphorus, chlorpyrifos, diazinon, dichlorvos, malathion, cygon, propoxur, carbaryl and chlordane."

The study also found that such cancer risks were much higher in children who possessed genes linked to the activity of certain enzymes (P 450) which, they suggest, can activate the carcinogens in the pesticides. However another researcher, writing in the same journal, said that these results must be cosidered priliminary as the study was "one of the first, if not the first, to evaluate gene-environment interactions for pesticides and childhood leukemia".

Source: The Sun, Malaysia, August 23, 1999.

Fertilizer and Pesticide Combinations

The study focused on three commonly used farm chemicals: aldicarb, an insecticide; atrazine, a herbicide; and nitrate, a chemical fertilizer. All three chemicals are in wide use worldwide and are the most ubiquitous contaminants of groundwater in the United States.

In a series of experiments, when mice were given drinking water laced with combinations of pesticides and nitrate, they exhibited altered immune, endocrine and nervous system functions. Those changes, according to Porter, occurred at concentrations currently found in groundwater. Effects were most noticeable when a single pesticide was combined with nitrate fertilizer.

The apparent influence of pesticide and fertilizer mixtures on the endocrine system, the system of glands such as the thyroid that secretes hormones into the bloodstream, may also result in changes in the immune system and affect foetel brain development. "Thyroid disruption in humans has multiple consequences", Porter said. Some of these include effects on brain development, level of irritability, sensitivity to stimuli, ability or motivation to learn and altered immune function.

A curious finding of the study is that animals may be more vulnerable to the influence of such chemicals depending on the time of year: "Our current working hypothesis is that animals are seasonally vulnerable because of subtle modulation of natural seasonal variation in hormone levels," according to Porter.

Need for New Testing Methods

This new study, Porter contends, adds to a growing body of evidence that current testing methods required for the registration and use of chemical pesticides in the US are fundamentally flawed. The study lists 6 important deficiencies in current testing protocols.

- Current tests do not require chemicals to be tested at low dose pulse exposure. Pulse doses of low levels of pesticides at critical times when developmental windows are open and body defenses are unable to respond may lead to permanent changes in a foetus. It is important to remember that the embryo has almost no defensive systems against chemicals and no feedback systems to modulate chemical concentrations early in its development.
- Toxicological tests have typically focused on cancer and mutation endpoints and have not looked at other critical concerns such as endocrine and immune system effects that can occur.
- Standard toxicological tests only evaluate one route of exposure at a time, rather than all pos-

Flyers Beware! Pesticides on Aircraft

Airline passengers and crew can be exposed to hazardous pesticides without their knowledge, according to a report released by the Northwest Coalition for Alternatives to Pesticides (NCAP), USA. The report, "Flyers Beware: Pesticide Use on International and Domestic Aircraft and Flights" states that pesticides are commonly used on both cargo and passenger aircraft in the U.S and in other countries. Some airlines spray voluntarily, while others spray to comply with national regulations or requirements of other countries. Pesticides are used in occupied and unoccupied passenger cabins, galleys, cockpits and cargo holds.

On flights to at least six countries (Trinidad and Tobago, Grenada, Madagascar, Kiribati, India and Uruguay) passengers are directly sprayed with pesticides after landing while still strapped in their seats. According to one airline attendant, passengers' clothing, skin and hair may be soaked with the pesticide.

On flights to many other countries, passengers are exposed to pesticides sprayed prior to boarding without their knowledge. This type of spraying leaves long-lasting insect-killing residues in the passenger cabin. It is currently required on some or all flights to Australia, New Zealand, Jamaica, Barbados, Panama, Fiji and Guam.

Passengers on U.S. domestic flights may also be exposed to insecticides residues sprayed on aircraft.

Several insecticide active ingredients commonly used on aircraft, including permethrin, cypermethrin and piperonyl butoxicide, are classified by the U.S Environmental Protection Agency as possible human carcinogens. Others are classified as reproductive hazards or suspected endocrine-disrupting chemicals.

NCAP says airlines should use non-toxic pest prevention and management practices, and that governments should prohibit or discourage use of hazardous pesticides on aircraft.

Source: Northwest Coalition for Alternatives to Pesticides (NCAP) published in Global Pesticide Campaigner, April 1999.

The full report on aircraft spraying is available on NCAP's website at www.efn.org/~ncap/AirlineSpray.pdf. For further information contact: Northwest Coalition for Alternatives to Pesticides (NCAP), P.O. Box: 1393 Eugene, OR 97440. Tel: (541) 344 5044 Fax: (541) 344 6923.

sible routes i.e. oral, cutaneous and respiratory.

- Most testing is done with pure forms of pesticidal active ingredients rather than with commercial formulations. There are three types of chemical additives missing from most testing protocols; i.e. contaminants of manufacturing processes, toxic waste deliberately added from chemical reactor cleaning processes and "inert" ingredients.
- Current testing requirements do not evaluate exposure effects from chemical mixtures. While it is impossible to examine all possible mixtures, common combinations generated in specific areas due to crop rotation and tillage practices could be examined.
- Laboratory animals generally live in an environment where climate, nutrition and disease are carefully controlled. Researchers know that when additional stresses are present, toxic responses to registered chemicals occur that may not appear under current standard testing procedures.

"Toxicology testing so far has been extremely limited in scope and focused on mechanisms that require extensive mutations or cell damage to show any effects," said Porter. "They do not adequately assess the potential for biological effects under real world exposure scenarios." *(See Box: Flyer Beware! Pesticides on Aircraft)*

Source: Global Pesticide Campaigner, Volume 9, No. 1. PAN North America, April 1999, Original Source: Warren Porter et al., "Endocrine, Immune and Behavioural Effects of Aldicarb (carbamate), Atrazine (triazine) and Nitrate (fertilizer) mixtures at groundwater concentrations," Toxicology and Industrial Health (1999), 15, 133-150, University of Wisconsin-Madison Press Release, March 15, 1999.

5 Recommendations: Protecting Farmworkers from Pesticides

"Pesticide exposure can cause serious acute illness among farmworkers. In the incident described in this report, workers entered a field well before the end of a label-specified restricted entry interval (REI) and incurred pesticide exposure that resulted in a moderately severe illness. The incident demonstrates that 1) posted and oral warnings based on the REI are necessary to prevent illness among workers performing hand labor in fields recently treated with pesticides and 2) failure to adhere to an REI can result in substantial morbidity [illness] among exposed workers. Because this incident demonstrates that sole reliance on these control measures may be inadequate, the substitution of safer, less toxic alternative pesticides should be adopted when feasible" (CDC 1999).

As demonstrated in the above excerpt from a recent Center for Disease Control (CDC) report, reliance on notification measures alone is in many cases inadequate to prevent farmworker poisoning by pesticides. Farmworker experiences show that even pesticide applications which follow the letter of the law can result in exposure or illness.

The most important and urgently needed step to reduce exposure is eliminating use of pesticides which endanger the health and well-being of farmworkers throughout the state. Phasing out use of the most dangerous pesticides—those that cause cancer or reproductive harm, or are extremely toxic to the nervous system—would represent tremendous progress toward a more sustainable, healthy and humane agricultural system. Substituting safer alternatives for toxic materials is a well-established first step in worker protection as outlined in the widely accepted principles of industrial hygiene (Soule 1991). Specific steps needed to reach this goal and effectively promote viable alternatives are outlined in Recommendation #1 below.

To reduce the level of farmworker exposure to those pesticides which remain registered, we recommend outlawing several hazardous use practices, improving protection from drift and residue exposure, and significantly strengthening the existing enforcement system. Improved reporting and treatment of pesticide illnesses are also critical, as is access to accurate information on pesticide use, violations and illnesses for both farmworkers and the general public. Below we explore these recommendations in greater detail, including some of the specific steps needed to reduce farmworker exposure to dangerous pesticides.

1. Rapidly phase out use of the most toxic pesticides and promote healthy and sustainable alternatives.

- California's Department of Pesticide Regulation (DPR) should develop and implement a plan to phase out use of pesticides that cause cancer or reproductive harm or are highly poisonous acute nerve toxins. In addition, the agency should develop and implement a plan for reducing use of all pesticides, including setting annual goals for total use reduction and ensuring, at the same time, that toxicity is not increased.
- DPR should immediately prohibit use of pesticides that are most hazardous to workers (highly acute nerve toxins, carcinogens and pesticides that cause reproductive harm) on labor-intensive crops.
- California Environmental Protection Agency (CalEPA) should commit significant resources to organic agricultural research and programs to assist farmers in pesticide use reduction and in the transition to sustainable alternatives.[37]

- CalEPA and California Department of Food and Agriculture (CDFA) should increase their research and training budgets in each of the following areas: organic agriculture, biointensive and integrated pest management programs and pesticide use reduction programs. These expenditures should be analyzed annually and compared with expenditures in support of conventional agriculture. Results of this analysis should be made public and widely available.

2. Improve regulations to reduce farmworker exposure.

- DPR should ban aerial spraying of agricultural pesticides, and prohibit use of backpack spraying for all restricted use pesticides and acute systemic toxins.
- DPR should expand posting requirements to apply to all agricultural pesticide applications. Warnings should be required prior to application along the perimeter of all areas where application occurs in such a manner that the warnings are highly visible to workers and other people who might enter the area. All posting signs should include pesticide name and reentry date and be written in the primary language(s) of the farmworkers.
- DPR should require that employers notify farmworkers 24 hours in advance of all pesticide applications in fields they work in or near.
- DPR should extend restricted entry intervals (REIs) to take into account multiple pesticide exposure and prevention of chronic health effects. Early reentry exceptions should be eliminated, and DPR should document and make public the scientific basis for REIs.
- DPR should establish and/or expand worker buffer zones for all fumigants and air-blast spraying.
- Growers should be required to provide washing and laundry facilities for farmworker use on any farm where pesticides are applied.
- Training requirements should be improved and enforced for all pesticide applicators and workers who enter fields or handle crops.
- Agricultural workers should be covered by OSHA's Hazard Communication Standard.

3. Strengthen enforcement of existing laws.

- DPR should set minimum mandatory penalties that county agricultural commissioners must issue for violations of pesticide laws that could endanger the health and safety of workers. The option of issuing "Notices of Violations" and "Letters of Warning" should be abolished.
- DPR should increase fine levels for moderate and serious violations and enforce the automatic "serious" designation for repeat "moderate" violations, as specified in pesticide regulations.
- DPR should require pesticide users to be familiar with regulatory requirements. The "ignorance excuse," a policy of leniency towards violators if they claim to be unfamiliar with relevant requirements, should be abolished. (The DPR Pesticide Policy Manual currently recommends issuance of a "Notice of Violation" rather than a fine for a violation that is a possible health and safety hazard if the violator is judged unfamiliar with pesticide regulatory requirements.)
- An independent review board should be established to annually evaluate the performance of each county agricultural commissioner, with participation from agricultural workers. Elected county officials should receive copies of all agricultural commissioner workplans and evaluations. DPR should exercise its authority to withhold funding from agricultural commissioners' offices that inadequately enforce regulations.
- DPR should require that every county agricultural commissioner's office have at least one bilingual investigator on staff.

- DPR should require special investigations of all pesticide illnesses resulting from legal use practices, rather than allowing agricultural commissioners to take no action in cases where no specific violations are found.
- Poisoning investigations should always involve the Department of Health Services' Occupational Health Branch and/or OSHA, in addition to DPR.
- State agencies should assess stiff penalties for employer retaliation against whistleblowers and for interference with workers' right to organize.
- Agricultural inspectors should enforce existing law (CCR, Title 8, Section 3457), which mandates a minimum $750 fine for inadequate sanitation facilities, as specified in CalOSHA regulations.
- DPR should mandate that egregious violators whose actions endanger workers shall be referred for civil or criminal prosecution and/or have pesticide use permits and licenses revoked for a full growing season.

4. Improve reporting of pesticide poisonings.

- Work "safety incentive" contests that provide bonuses or prizes to work crews when no injuries or illnesses are reported in a given time period should be prohibited.

Jocelyn Sherman, UFW

DPR should expand posting requirements.

- Insurance companies should be required to immediately forward copies of "Doctor's First Report of Occupational Illness or Injury" involving pesticides to the Department of Health Services (DHS) and DPR Worker Health and Safety Branch.
- DHS should establish and fund a program to monitor long-term health impacts of pesticide exposure among farmworkers.
- DHS should expand its existing program to train doctors about pesticide poisoning diagnosis, treatment and reporting requirements. Crop-sheets highlighting symptoms of pesticide poisoning should be widely distributed to migrant health clinics and other physicians or health care providers.
- CalOSHA and the Medical Board of California should exercise their authority to fine doctors who fail to report pesticide poisonings promptly to the county health authorities.

5. Improve farmworker access to medical treatment.

- Failure of agricultural employers to provide workers and doctors with full information about chemicals involved in a possible exposure incident should constitute "interfering with access to medical treatment" and should be enforced aggressively. Regulations requiring employers to take exposed workers promptly to a doctor should be enforced.
- The federal government should increase funding for migrant clinics and other health care providers for farmworkers, including funding for free annual physicals to screen for symptoms of pesticide exposure. These free physical exams should be available to all, regardless of immigration status.
- Agricultural employers should be required to provide health insurance and/or establish a fund to finance farmworker health care costs.
- DHS should expand cholinesterase monitoring programs to include all field workers who could be exposed to organophosphates or carbamates during the course of their work.

6. Ensure farmworker and public right-to-know.

- DPR should expand workers' right-to-know to include posting of REIs and descriptions of acute and chronic health effects associated with each chemical. The information should be posted in a neutral location on the farm in an understandable format and language.
- The Office of Environmental Health Hazard Assessment should ensure that all farmworkers are guaranteed "adequate warning" about exposure to carcinogens and reproductive toxins, as required under Proposition 65.
- County agricultural commissioners should document all drift inquiries; monitor, analyze and publish trends in inquiries and complaints; and institute mandatory site visits in response to repeated inquiries and/or complaints.
- County agricultural commissioners should make the results of pesticide investigations available to DHS and the public within three months of an investigation.
- DPR should release pesticide use and illness data no later than six months after the end of the year for which the information is reported, and should produce an analysis of pesticide use trends and reported poisonings.
- DPR should establish a public database with information on the amount of pesticides used, violations reported, number of workers affected by the violations and number of pesticide illnesses for each user grower. This integrated database could be an expansion of the Agricultural Civil Penalties database of pesticide enforcement actions, and would be analogous to the national Toxic Release Inventory and the statewide Hot Spots database for air polluting chemicals.

In India the blame for direct pesticide poisoning can be put partly on the easy availability of toxic pesticides, often with little effective restriction or controls, in conditions where the necessary safety precautions are highly unrealistic.
Dr. Copplestone of WHO has said, "In all societies, but especially where literacy is low, probably the most effective single measure to promote safety is the restriction of the availability of the more toxic pesticides to those who have been trained in their use, and who have a specific need for them"

— Amita Baviskar —

IG Farben: Participating in State-Sponsored Human Rights Atrocities

"Its bricks were ... cemented by hate; hate and discord, like the Tower of Babel, and ....in it we hate the insane dream of grandeur of our masters, their contempt for God and men, for us men."

- Primo Levi describing the Carbide Tower in the middle of the Auschwitz rubber plant, built by concentration camp inmates[22]

The relationship between government authorities and corporate human rights abuses is a significant and sometimes confusing issue. Governments cannot legally authorize corporations to violate human rights, but corporations often act as if government's permission or encouragement will suffice. Perhaps the most notorious example of this relationship occurred under the Nazis' genocidal rampage in Europe. A cartel of chemical companies known as IG Farben took part in, and benefitted from, the Nazis' atrocities.

In 1925, Bayer, BASF Hoechst and other German companies joined forces to form the cartel — in German an Interessegemeinschaft, or "IG," which they named "IG Farben." This became the linchpin of Hitler's military/industrial complex, providing synthetic nitrates, fuel and rubber[23]. The Nazis' Dr. Josef Mengele conducted gruesome experiments on the inmates in the concentration camps at Auschwitz and elsewhere. IG Farben is alleged to have experimented on inmates against their will with drugs and other chemicals.

Except from "Victims No More," National Catholic Reporter, John L. Allen Jr.

During the unwilling testing of new drugs on concentration camp prisoners, Nazi Doctor Josef Mengele favored experiments on twins because twins have the same genetic code[24]. Eva Mozes Kor was injected with unknown chemicals several times. After one such set of injections, Eva developed an intense fever and was sent to the prison infirmary. During the first two weeks Eva was hospitalized, her twin sister Miriam was put under constant SS guard. The guards were poised to kill Miriam as soon as Eva died, so doctors could perform comparative autopsies.

Kor's lawyers say they have evidence of ampules of drugs bearing the Bayer label found in Auschwitz; records of doctors who were Bayer employees and who conducted experiments in the camps; and correspondence suggesting that Bayer officials knew about the experiments and collected results[25]. The lawsuits name specific people as IG Farben employees, such as SS Dr. Helmut Vetter and Dr. Bruno Weber.[26] [Bayer denies the allegations.]

"In response to requests from Bayer, they experimented with drugs Bayer was in the process of developing," Kor's lawyer Richard Shevitz said. "This was [research and development] conducted in the context of the Holocaust."

Some previously published documents seem to buttress parts of that argument. One of the most sensational is a Nov. 19, 1943, letter from an IG Farben official, Wilhelm Mann, to Otmar von Verschuer, Mengele's mentor. In the letter, Mann — director of pharmaceutical sales at Leverkusen — thanks Verschuer for acquainting him with Mengele, and says he found Mengele's demonstrations "very impressive." He says he will take up the question of funding, and refers to an enclosed "first check."[27]

An Austrian association that maintains records from the Matthausen-Gusen camp system confirmed that Dr. Helmut Vetter did inject inmates with drugs labeled "Ruthenol" and "Praeparat 3582" in block 27 of the Gusen camp.

Shevitz says he believes the concentration camp experiments helped Bayer develop products that are in use today. "We know they were used to develop conventional medicines. It's a matter of asking Bayer how much profit can be traced to those experiments. It's a significant amount of money," he said.

As much as she wants — and believes she is owed — financial compensation and an explanation of what was done to her in Auschwitz, Kor believes her fight with Bayer has broader significance too. "Companies must treat human beings with respect."

While in no way diminishing the incomparability of the Holocaust, Michael Bazyler, a professor at the Whittier

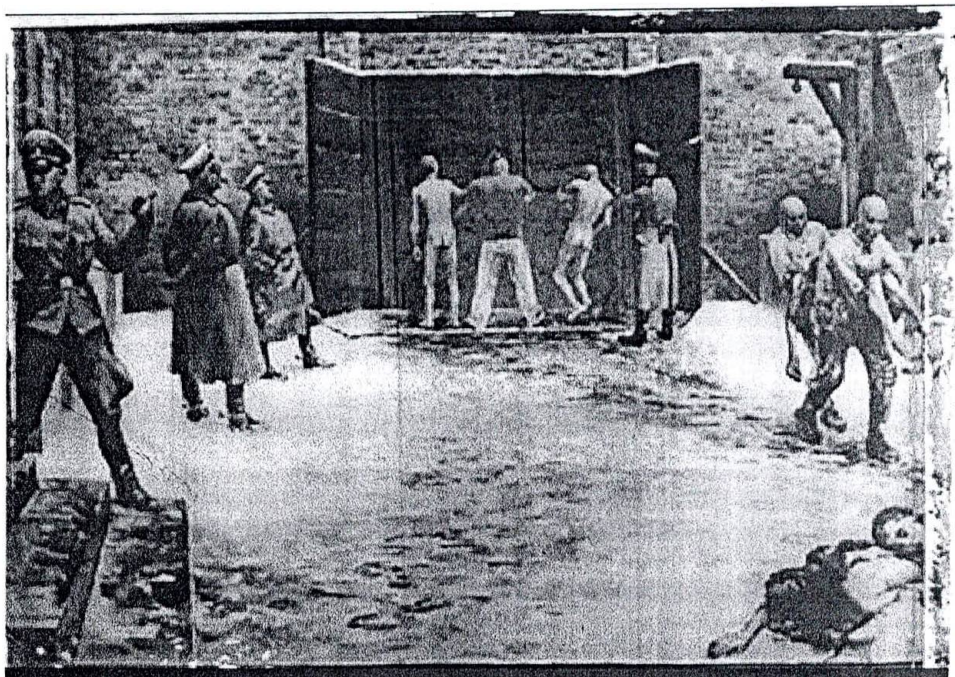


Photo taken by Gary Cohen in Auschwitz Museum. Source of original photo unknown.

Nazi guards executed concentration camp inmates for failing to keep up with the pace of work.

freight cars all day long at a running pace. Prisoners who broke down were beaten by the German IG foremen as well as by the kapos until they either resumed their work or were left there dead. I saw such cases myself. I also noticed repeatedly, particularly during the time when the SS accompanied our labor unit themselves, that the German IG foremen tried to surpass the SS in brutalities."

Workers unable to keep up with the pace were put to death. Paul M. Hebert, one of the judges at the postwar trial of IG Farben wrote: "It was Farben's drive for speed in the construction of Auschwitz which resulted indirectly in thousands of inmates being selected for extermination by the *SS* when they were rendered unfit for work..."[31]

Law School in Costa Mesa, California said Kor's lawsuit and those of other survivors form a dramatic new front in the broader fight to hold corporations accountable for their conduct.[28]

"Obtaining compensation from bankers and industrialists who profit from human rights abuses sends a message that they cannot hide behind the cloak of 'business as usual' when they become joint venturers with a dictatorial regime," he said.

IG Farben was the only business to operate its own concentration camp. IG Farben erected the Monowitz camp, with guard towers, barbed wire and gallows. The forced labor of inmates was used to erect the chemical operations. Norbert Wollheim, a German Jew who was brought with his wife and three-year-old son to Auschwitz in 1943, testified after the war that he was separated from his wife and child and taken to a camp.[29] There he was robbed of all possessions, deloused, registered, and tattooed with the number 107,984. The next day he was brought to a synthetic rubber plant being built for IG Farben[30] by slave labor of concentration camp inmates:

> "As initiation, as was the general rule, we were given only the hardest and most strenuous work, such as transportation and excavation work. I came to the dreaded "murder detail 4," whose task it was to unload cement bags or construction steel. We had to unload the cement from arriving

IG Farben: A Human Rights Analysis

One thing the Farben case showed clearly was that the chemical industry's officials were capable of objectifying humans, even to the extent of making their lives expendable. And the industry's technologies had the capacity for large-scale killing. The atrocities of the era violated the Right against Genocide.

Some Farben officials were ultimately prosecuted for their part in the atrocities. At Nuremberg, twelve senior executives were jailed for terms ranging from one to eight years. The allies then split the company back into its original constituents: Hoechst, Bayer and BASF. One of the company's dominant figures, the scientist Fritz ter Meer, got seven years. When he emerged from jail, he was immediately appointed chairman of Bayer. IG Farben proved able to survive the political regimes with which it was intimately associated.

Philosopher Hannah Arendt, in attempting to understand the Nazi era, has written that evil was not only committed by fundamentalist zealots, but by people who were simply doing their jobs– embodiments of the "banality of evil." In this way, thousands of petty officials could fulfill their small, seemingly innocuous jobs, connected together

1925	1933	1938-1943	1940	1943
Major German chemical companies form IG Farben.	First Nazi concentration camps built.	IG Farben made profits of 100-200 percent from stock holdings in Degesch, the manufacturer of Zyklon B.	The building of Auschwitz begins with oversight by IG Farben officials, and labor from concentration camps.	IG Farben official Wilhelm Mann writes letter expressing appreciation of S.S. Dr. Mengele's experiments.

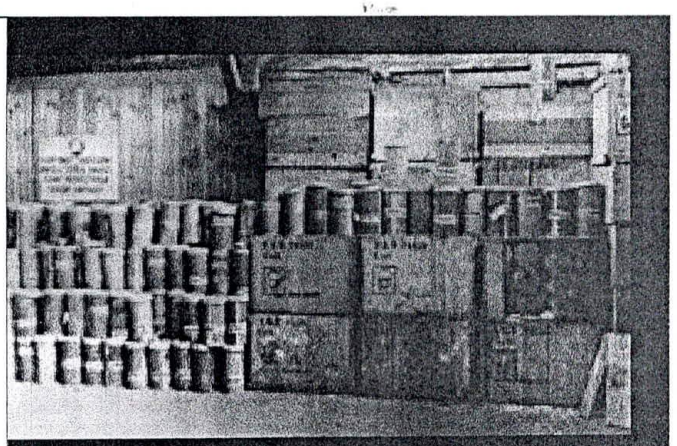
in a vast machinery of brutality and injustice that sent millions of people to their deaths.

The Nazis' efficient technological and corporate structure was an effective mechanism both for removing individual responsibility and in dehumanizing its victims. Jews in transport trains to the death camps were called "pieces". Train officials processed 15,000 pieces from Hungary, 10,000 pieces from Greece, a million pieces from Poland, etc.

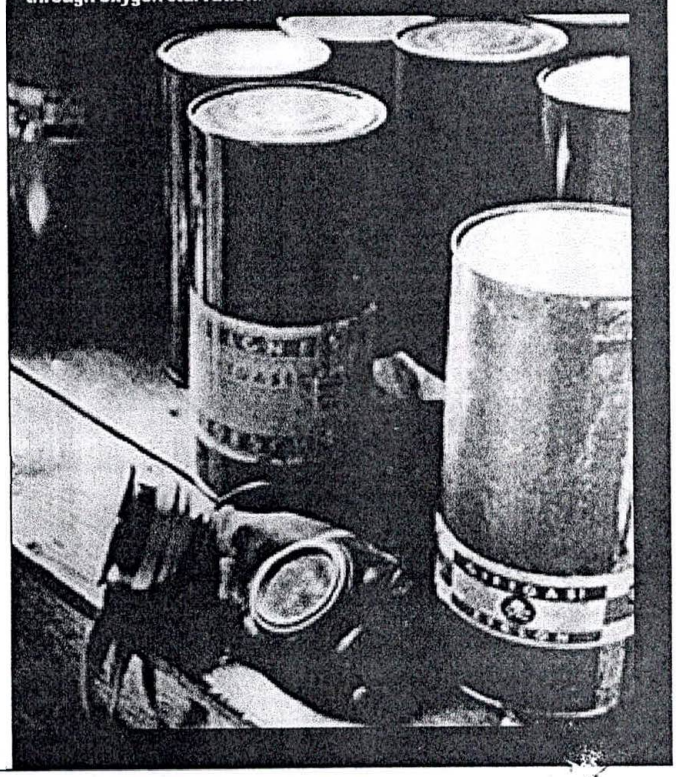
The IG Farben case is the only one in which chemical industry executives were prosecuted and convicted of crimes against humanity. The activities of Farben were undertaken on behalf of the most evil regime of the century, whereas the other case studies in this paper involve more purely commercial endeavors.

After the Nazi era, the chemical corporations shifted to justifying their most harmful activities not in terms of the need for extermination of people, but in terms of *acceptable risks* and the need to advance their product lines and profitability. Underlying both Nazi and corporate logic, however, a similar dehumanization finds expression. A certain group of human beings is made expendable, a certain amount of destruction must be tolerated, in the name of progress and profit.

One result of the Nazis' experimentation was the establishment of the Nuremberg Code, which provides that experimentation on human subjects shall not be committed without willing participation of the subjects. While that code was established in a medical context, the same ethical rationale applies to the industry's global experiment on involuntary humans. Yet, as far as we know, the obvious connection has yet to be made in any courts.



Zyklon-B, used to kill concentration camp inmates, was provided to the Nazis by IG Farben. This is a Stockpile of Zyklon-B poison gas pellets found at Majdanek death camp in 1944 and close-up of the containers and a gas mask. The containers hold Zyklon-B pellets (hydrocyanic acid) that vaporize when exposed to air. Originally intended as a disinfectant and insecticide, the Nazis discovered through experimentation that the gas could be used to kill humans. Prisoners were forced into air-tight chambers disguised by the Nazis to look like shower rooms. The Zyklon pellets were dumped into the chambers via special air shafts or openings in the ceiling. The pellets would vaporize, giving off a noticeable bitter almond odor. Upon being breathed in, the vapors combined with red blood cells, depriving the human body of oxygen, causing unconsciousness, and death through oxygen starvation.



Photos Courtesy of US Holocaust Memorial Museum Photo Archives

1944	1948	1953	1956	1995
The IG Farben industrial complex at Auschwitz is bombed by the Allies. Gassings end in November after more than a million are dead.	Nuremberg trials convict twelve Farben executives of human rights violations, including Fritz ter Meer.	IG Farben's assets divided between Hoechst, BASF, Bayer and other firms.	IG Farben executive Fritz Ter Meet is released from jail and elected Chairman of the Board of Bayer.	Ernest Krienke, chairman of IG Farben Board, rejects demands that the surviving IG Farben slave laborers be paid reparations by the company.

Globalization: Free Trade in Toxic Products, Technologies, and Wastes

Thor employs a lot of casual labor...and when they become ill from the poisons they are fired for carelessness.

– Eric Ncube, former shift leader at Thor Chemicals, South Africa, a plant that received mercury wastes from US and British manufacturers

Exporting Toxic Pesticides

Excerpt from "Human Rights Implications of the Export of Banned Pesticides," by Beth Gammie[33]

A disturbing pattern has emerged. A chemical company will spend large amounts of money to manufacture a pesticide, and obtain its registration to be sold in the United States. The pesticide's harmful health and environmental effects then become apparent, either through incidents of pesticide poisoning or further research. After a slow and laborious process, the EPA eventually determines that the pesticide causes harm to human health or the environment, and the pesticide is removed from the American market. However, the chemical company continues to export the banned pesticide to foreign countries or transfers production out of the United States. Thus a "circle of poison" is created: a pesticide is manufactured in the United States, is exported, and returns to the United States on pesticide-tainted fruits and vegetables.

The unfortunate reality is that corporations often know or suspect the detrimental impacts of their products, but do not act on what they know.

Early studies by Shell and Dow revealed that DBCP caused sterility and precancerous lesions in lab animals.[34] However, these results were not revealed to the workers in the DBCP manufacturing plants nor to the agricultural workers who were exposed to DBCP in the field.[35] Widespread use of DBCP throughout the banana industry was prevalent in all major banana plantations during the 1970's. The EPA suspended the sale of DBCP for most uses in 1977 after Occidental workers brought suit for sterility in California. The potential for profit and the drive to keep businesses in operation too easily overrides the concerns about health. While Dow, Occidental, and Shell ceased production of DBCP after California banned its use, a smaller company, American Vanguard Corporation (Amvac), seized the opportunity to fill the vacuum in the DBCP market by manufacturing and exporting DBCP.[36] Amvac produced and

"[Q]uite frankly, without DBCP, Amvac would go bankrupt."

– Former Amvac executive

In a report to the U.S. Securities and Exchange Commission, Amvac stated:

[M]anagement believes that because of the extensive publicity and notoriety that has arisen over the sterility of workers and the suspected mutagenic and carcinogenic nature of DBCP, the principal manufacturers and distributors of the product (Dow, Occidental, and Shell Chemical) have, temporarily at least, decided to remove themselves from the domestic marketplace and possibly from the world marketplace. Notwithstanding all the publicity and notoriety surrounding DBCP, ... it was [our] opinion a vacuum existed in the marketplace that [we] could temporarily occupy... [we] further believed that with the additional DBCP, sales might be sufficient to reach a profitable level.[38]

sold DBCP for export. Dow also profited; although the company no longer manufactured DBCP, it received a three percent royalty on all DBCP sold due to a patent agreement.[37]

The effects of DBCP exports proved to be just what one would expect based on the numerous studies on DBCP exposure. DBCP sterilized many men, including those working in factories where DBCP was manufactured and those who applied DBCP in the field.[39] As of 1992, approximately 15,000 male banana workers in 12 banana-growing countries, including 12,000 in Costa Rica and the Philippines alone, had been sterilized by their exposure to DBCP in the field.[40] These men, unable to father children, suffer a wide-range of secondary effects, including depression, impotence, and divorce, as well as cancers possibly linked to their exposure.[41]

Approximately 29% of all pesticides sold abroad are either banned, restricted, or unregistered in the United States.[42] Over a three month period during 1990, an estimated 3.5 million pounds of banned, canceled, discontinued, or withdrawn compounds were exported, equaling almost a ton per hour.[43]

These figures represent an enormous amount of exports of illegal pesticides. The effect of this pesticide "dumping" on foreign countries is considerable.

In 1990 it was estimated that 25 million people are severely poisoned every year by agrichemicals. The World Health Organization (WHO) estimated in 1985 that over 70,000 deaths resulted worldwide from accidental pesticide poisoning. Some specific instances illustrate the devastation of toxic chemical exports:

- DDT was sprayed heavily on cotton fields in Guatemala. Researchers found that villagers living near the fields had blood levels of DDT seven times higher than those living in urban areas, and thirty-one times higher than United States residents.[44]

- Residues of heptachlor have been found in the breast milk of mothers in Perth, Australia, in amounts fifteen times international standards.[45]

- The WHO estimated approximately 37,000 cases of cancer annually from pesticide exposure.[46]

Free Trade in Poisons: A Human Rights Analysis

Respect for human rights is seldom an obstacle to the global trade in poisons. In our era of globalization, chemical companies increasingly move around assets, products and wastes on a global chessboard to maximize their profits and minimize their costs. Asbestos, long banned in the U.S. because of its devastating impacts on workers, is sold by Canadian companies to "developing" countries. Waste incinerators, discredited in the United States due to their emission of dioxins and other pollutants, are being financed by World Bank grants to more than 20 countries trying to grapple with their burgeoning waste streams. The chemical industry's human rights violations are repeated in every corner of the Earth.

All persons have the right to freedom from pollution...and activities that adversely affect the environment, threaten life, health, livelihood, well-being or sustainable development within, across or outside national boundaries.

UN Commission on Human Rights[47]

Chemicals that sterilize men or women, or otherwise endanger pregnant women and the health of the fetus in utero, violate the right to family. The Universal Declaration of Human rights articulates this Right to Family: "men and women of full age ... have the right to marry and found a family."[48] The U.N. recognized the Right to Family as including the right of parents to decide when and whether to bear children.[49] By taking away the opportunity to bear children, the chemical industry's involuntary sterilizations of men and women violates this right.

The chemical industry's shifting of pollution and products often constitutes a violation of the right against discrimination, in this context often referred to as environmental racism. This is the discriminatory imposition of

Type of Pesticide	Millions of Pounds Exported in 1995-1996
Banned or Forbidden in U.S.	21
"Never Registered"	9.4
"Extremely Hazardous"	28

Pesticide	Most Uses Banned by EPA	Export Year and Company
DDT	1973	1979-1980 Monsanto
Chlordane and Heptachlor	1978	1987 - mid-1989 Velsicol Chemical Company
DBCP	1977	Dow, Occidental, Shell - Pre-1977 American Vanguard Corporation (Amvac) - Post 1977

pollution on poor and minority ethnic populations.[50] This is frequently displayed in the movement of hazardous wastes, products and production technologies from richer to poorer locales. Waste disposal facilities and chemical production clusters are notorious for their locations in poor and ethnic minority neighborhoods. Wastes and toxic substances run "downhill" in the direction of poor countries and communities just as surely as water runs down a mountain.

Cancer causing pesticides banned in the U.S. and Europe have been freely exported to farmers in Asian, African and Latin American countries for many years. But pesticides exports are going through a transformation. Increasingly, chemical corporations are moving pesticide *production* facilities outside of traditional strongholds in the U.S. and Europe, especially for older technologies.[51] The Bhopal pesticide plant was an early example of this tendency. Asia is seen as the choice region for expansion, especially India and China.[52] Expansion in Latin America, where many large facilities already exist, remains strong.[53] In addition, transnationals are beginning to expand on a very small base in Africa, and to explore new opportunities in Eastern Europe.

Recently, the global trade in poisons has accelerated under the banner of Free Trade, and a new international agency called the World Trade Organization (WTO). Under this new regime, global corporations are free to export dangerous products and technologies to 134 nations, as they shop for the cheapest labor costs and weakest environmental and public health protections. When individual nations try to impose strict regulations to defend their citizens from toxic exports, the exporting nation can appeal to the WTO to strike down those environmental laws as an unfair restriction of trade.

In the course of enforcing its free trade policies, the WTO has ruled against the ability of nations to apply the Precautionary Principle in their decisions to regulate product imports. As Jim Puckett of the Asia Pacific Environmental Network has written, the Precautionary Principle is a common sense concept encapsulated in well worn adages passed on from generation to generation such as "a stitch in time saves nine", "look before you leap", "an ounce of prevention is worth a pound of cure,"

Precautionary Principle

"In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation."

Principle 15 of the Rio Declaration on the Environment and Development

Containers of hazardous waste in New Jersey bound for Asia, Greenpeace investigation.

Donna DeCesare/Impact Visuals.

"fools rush in where angels fear to tread", "better safe than sorry," and "when in doubt, do without."[54] Put in the language of public policy, the Precautionary Principle posits that: *where an activity raises serious or irreversible threats of harm to the environment or human health, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically.*

The relevance to the activities of the chemical industry is clear. The industry over the last century has repeatedly shown the dire results of a nonprecautionary approach. By delaying action until there is scientific certainty, the public and environment suffer enormous harm.

Yet the WTO made a decision in 1998 that limits the ability of all governments that are party to the agreement to apply the precautionary principle. The WTO struck down a European Union ban on the sale of beef grown with artificial growth hormones. The European countries had adopted their ban based on studies that showed risks of cancer and male sterility for consumers of the beef. The issue is subject, as so many issues are, to continued scientific debate. However, the precautionary approach taken by the European countries was based on a conclusion that there was enough evidence to assert that the synthetic hormones should be considered unsafe until they are proven safe. Shockingly, the WTO barred this approach. It requires that regulating nations provide more scientific justification before acting. The WTO's approach threatens to leave humanity helpless to intervene in the face of indications of harm. The WTO has endorsed the chemical industry's nonprecautionary approach, under which scientific uncertainty becomes an excuse for inaction.[55] The rationale is cropping up in opposition before the WTO of various nations' efforts to keep toxic materials out of their economies and environments. As Peter Montague has written:

> France wants to ban asbestos, but is being challenged by Canada on several grounds; one is that there is no worldwide scientific consensus that a ban is warranted. Denmark has announced its intention to ban 200 lead compounds, but the Clinton/Gore administration is challenging this as illegal because there are less trade-restrictive ways to achieve the same public health objective, Mr. Gore says. The European Union has said it wants to ban lead, mercury and cadmium in electronic devices, but the Clinton/Gore administration is challenging this before the WTO.[56]

Current globalization trends erroneously allow profitability to trump health concerns, and corporate rights to supersede human rights. Fortunately, international human rights law may provide recourse against global trade trends that undermine human rights. For instance, the American law which authorizes the export of banned pesticides, the federal Insecticide, Fungicide and Rodenticide Act,[57] could be challenged in US courts as contravening human rights law.[58] Similarly, international human rights law may override inconsistent activities under international treaties, including trade treaties. In international law, certain human rights including Rights to life, rights against genocide and against the arbitrary deprivation of life occupy a special status known as *jus cogens*.[59] These higher status rights are those for which violations are deemed to "shock the conscience of mankind" and thus are considered absolutely essential to the maintenance of the international community. Any treaty that contravenes a *jus cogens* norm is null and void.[60] Activities of trade organizations like the World Trade Organization and of financial institutions like the World Bank, in their prioritizing of economic interests above the sanctity of life, have quickly moved to a point of violating these *jus cogens* principles.

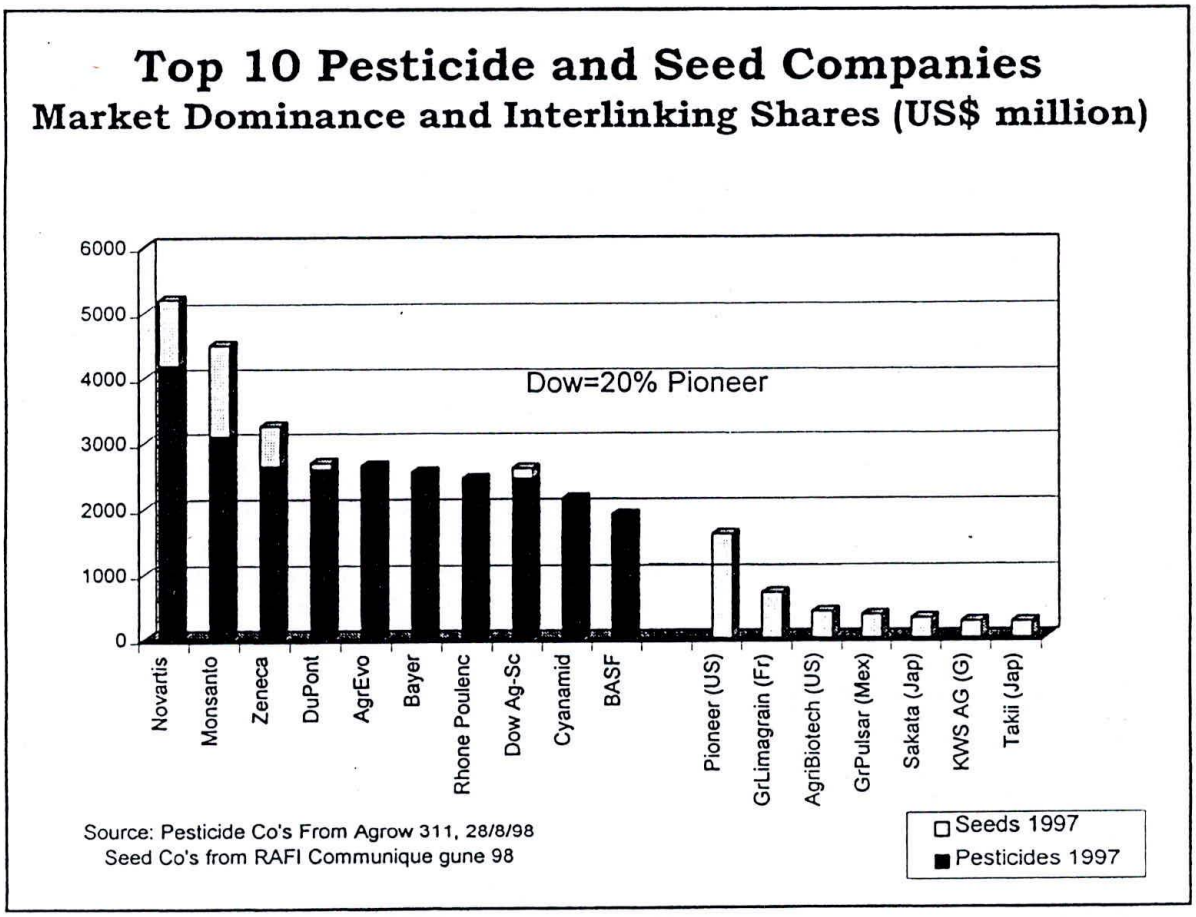
Unless human rights are enforced, we can expect global chemical and biotech companies to accelerate movement of their most harmful activities to the places where they can experiment or sell their wares most freely. Hazardous technologies and toxic substances may flow seamlessly across boundaries of geography and state with little recourse to those whose rights are irreparably harmed.

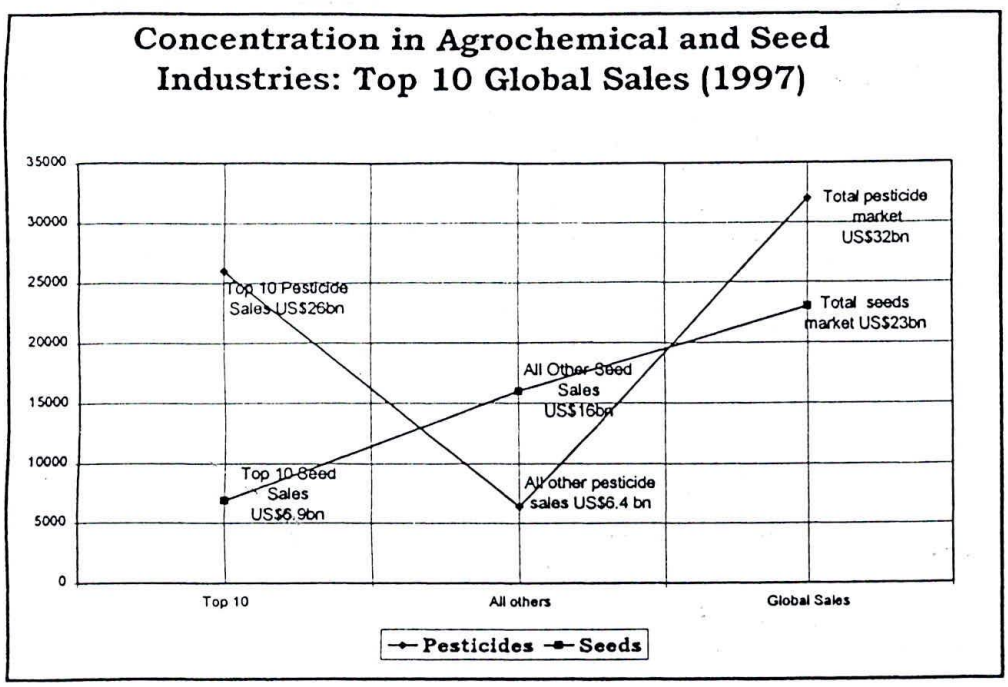
Impact of Corporate Control - The Pesticide TNCs

by Barbara Dinham

The challenge facing us is to achieve wider acceptance of the understanding that food security is about access to and distribution of food, and not about production. Industry promotes the view that increasing production can eliminate hunger, and many decision-makers accept this perspective. The vision of food security needs to be continually asserted against the barrage of productionist propaganda from the pesticide industry.

Agriculture is a complex sector. Unlike industry, agriculture is a way of life, it involves stewardship of the environment, it supports the rural social structure, and the products of agriculture feed the cities. In the forum of the World Trade Organization, these facts are disputed, and under the development of Uruguay Round Agreement on agriculture, the outputs treated like any other product. In the jargon of trade negotiations, this became reduced to an argument about the 'multifunctional nature of agriculture' – with pro-free traders refusing to acknowledge that protection was essential to protect the way of life intrinsic to agricultural production.





Part of the challenge to this view is continually drawing attention to the role of the transnational corporations that draw their profits from agriculture. Developments in this sector cut across the global agenda of liberalization, but to expand the companies constantly push for access to all markets.

Concentration and Control in the Pesticide Industry

In the last 50 years agriculture has been increasingly industrialized: first in Europe and North America and then with the development of Green Revolution techniques in developing countries. Monocultural production brought increasing use of agrochemicals and by 1997, the global sales of pesticides amounted to US$32 billion. The market is dominated by ten companies, which between them take about 80 per cent of global sales. These companies have elbowed out, or taken over, their competitors that do not have the financial resources to invest in the extensive research now needed to stay in the business.

These companies dominate the market, but there is also a growth of national pesticide industries in developing countries (India, Taiwan, China, South Korea, Mexico, Brazil) as well as a growth in the 'generic' pesticide producers. There is also an increase in the activities between the market leaders and companies appointed to market their older products.

The main markets for products remain in North America and Europe as regions; though India is now the second largest pesticide user in the world. As these markets are 'saturated', the big growth areas are targeted to be Asia and Latin America. The Chinese market is particularly interesting: China spends $6.7/ha on pesticides, compared to $752/ha in Japan, yet the Chinese yield is second only to Japan.[1]

The Seed Companies

More recently, concentration has begun to take place in the $23 billion seeds industry. Takeovers and mergers escalated throughout the 1990s and are continuing rapidly. In 1997, the sales of the top three companies accounted for 17 per cent, and are continuing rapidly. The companies were Pioneer Hi-Bred (20 per cent owned by DuPont), Monsanto and Novartis – all leading agrochemical companies. Changes in chemistry and economic, health and environmental pressures led these companies to develop a variety of strategies to continue extracting profits from agriculture.

The agricultural industries encourage monoculture, an agricultural system which inherently reduces agrobiodiversity (the FAO says more plant diversity has been lost to industrial agriculture than any other cause!), but which also increases pests attack and loss of beneficial animals (including insects) and crops. Some scientists have shown that reductions in biodiversity have led to the evolution of aggressive pests and diseases which are more difficult to control than those from which they have been derived. [2]

The full impact of a consolidation of interests is difficult to predict, but this trend now seems inevitable. One industry analyst observed: 'The days of seed companies selling commodity seed products that will be sprayed with pesticides marketed by a separate industry are clearly numbered. Seed companies are now selling seed brands engineered to express pest resistance genes or to be tolerant to specific herbicides'. [3]

The gains for industry could be phenomenal. Some industry analysts predict that the wave of agricultural biotechnology: herbicide tolerance and insect resistance traits could take the global agrochemical market up to a US$100 billion a year industry. [4]

Together the agrochemical and seed industries are reinventing themselves, and no longer market

1998 Top Ten Agrochemical Companies

Nearly all the major agrochemical companies increased sales in 1998, according to Agrow: World Crop Protection News. DuPont's combined agrochemical and biotechnology sales increased by over 25%, the highest rate of increase for the top ten corporations. DuPont recently announced that it had agreed to acquire the outstanding 80% stake in Pioneer HiBred International that it did not already own. Pioneer, the world's largest seed company with sales of US$1,835 million in 1998, controls about 42% of the U.S. maize seed market.

Monsanto's growth rate was a close second with combined agrochemical and seed sales increasing by more than 23% in 1998. This was due to a 25% increase in volume sales of the herbicide glyphosate (Roundup) and a tripling of the area planted with Monsanto's genetically modified crops.

Novartis was the overall sales leader in 1998 with pesticide sales reaching US$4,152 million and seed sales at US$1,005 million. Novartis, as well as Cyanamid, DuPont, Rhone-Poulenc and Zeneca were all hit by lower than expected herbicide sales in the U.S. due to low commodity prices and weather conditions as well as other factors.

Agrochemical and seed sales in Asia, Eastern Europe and Latin America were generally lower due in part to economic problems in these regions. However, Cyanamid, Dow AgroSciences, Novartis, Rhone-Poulenc and Zeneca all reported increased sales in Latin America. Cyanamid, Dow and Novartis also had high sales in Asia.

Top ten agrochemical companies — 1998 sales

Company	Sales*	% Change**
Novartis (Swiss)	$4,152	-1.1%
Monsanto (U.S.)	$4,032	23%
DuPont (U.S.)	$3,156	26%
Zeneca (U.K.)	$2,897	8.3%
AgrEvo (Ger)	$2,410	2.5%
Bayer (Ger)	$2,273	0.2%
Rhone-Poulenc(Fr)	$2,266	2.9%
Cyanamid (U.S.)	$2,194	3.5%
Dow Agro-Sci. (U.S.)	$2,132	11%
BASF (Ger)	$1,945	4.9%

* Millions of US$
** Since 1997

Sources: Agrow: World Crop Protection News, March 26, 1999 and April 16, 1999. Kindly forwarded via PAN North America (panupdates@igc.apc.org), May 7, 1999.

themselves as agrochemical and seeds companies, but as the LIFE SCIENCES companies: playing with life through the manipulation of genes.

Corporate Strategies for Influence

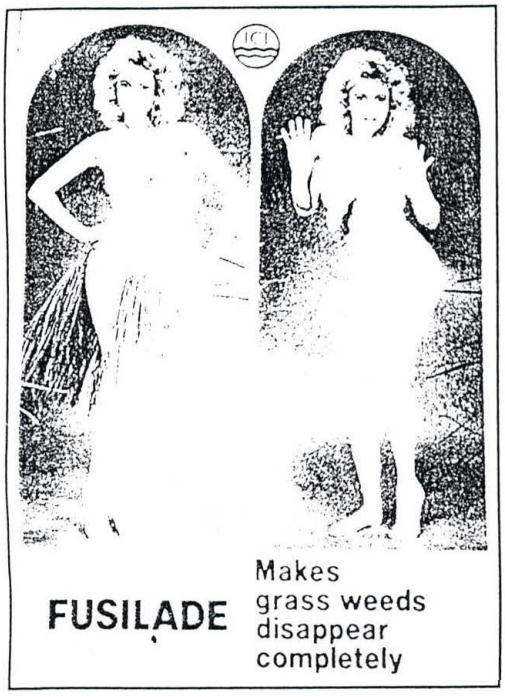
The interest in expanding from the pesticide market to other areas of profitability can probably be traced back to the early 1980s, when environmental concerns began to influence the agrochemical industry. This period began to see the division between research-based agrochemical companies and others; the cost of bringing new products onto the market was a high but essential, price to pay for staying in the game. Companies opting for this route inevitably sought ways to cover the cost of the research. With relatively flat sales through the eighties, a range of expansionary and defensive strategies were devised which kept the industry in a dominant position. Being a 'life science' company implied heavy investment in research. So the underlying tactics continue:

- Expansion of sales of older products, whose research costs have been recouped. These are cheaper and sell particularly well in developing countries. Most companies aim to increase sales in developing countries, particularly, but not exclusively, of older products. The lucrative Asian market has been a major target.
- Registration requirements. Industry faces tighter registration requirements. Its response is to promote the 'science' of risk management as the basis for product acceptability. The worker and consumer demand for precedence of the precautionary principle is undermined in the face of widespread regulatory acceptance of the infallibility of 'science', which puts regulators in a defensive position. Speaking at the British Crop Protection Council conference in 1997, B. Thomas of AgrEvo noted that data requirements on environmental fate and ecotoxicology have increased in recent years, particularly in Europe, and that industry is collecting data to lobby for a relaxation of the criteria.

The Public Image

Aware of the poor image of pesticides triggered by Rachel Carson's *'Silent Spring'* and sustained by publications such as *'A Growing Problem'* and the work of PAN, the agrochemical industry was on the defensive for some time through the 1980s. It is now more aggressively repackaging itself to claim the moral high ground. Its approaches seek to persuade decision-makers, and the public, that the industry is benign and promotes the common good through claims like:

- ***Feed the world***
- ***Protect the environment***
- ***Can be used safely in developing countries***
- ***Are IPM friendly***



Examples of advertising of pesticides by the ICI company that drew heavy criticism from citizen groups and the PAN Global Network. The poster above came from a shop of an ICI distributor in Quetzaltenango, Guatemala, 1992. The advert below, stating that "paraquat works in harmony with nature", was the focus of action by Consumer groups in Malaysia in 1993.

Feeding the World

A key approach is the public relations strategy: winning hearts and minds by 'demonstrating' that pesticides are essential in the battle to feed the 'world's relentlessly increasing population'. This public relations onslaught will continue as companies seek to gain the moral high ground: convincing the public and decision makers that pesticides are needed because only by use of high input agriculture will a population of 8 billion (estimated global population in 2020) be fed. However, food production in China has kept pace with population growth, while meeting policy objectives of maintaining reserves of 17 per cent of a year's food needs.

Protecting the Environment

Companies argue that intensive agriculture will prevent expansion onto wilderness areas, which are an important residue of biodiversity.

Safe Use

Industry recognizes that pesticides have caused health and environmental problems in developing countries, and safe use campaigns are intended to address bad press. This can be a cheap and effective way of advertising. As one company spokesman said: "If we teach farmers to use pesticides correctly, there will be no lack of customers for our products; indeed there might well be an increased demand for the safer and more sophisticated products which we are now making", David McDonald – Novartis (Ciba Plant Protection Farmer Support Team established in 1991)

Industry has invested mainly in only three safe use projects under the Global Crop Protection Federation (GCPF): in Kenya, Thailand and Guatemala. These projects promote awareness of protective clothing; pre-harvest intervals; labeling; good practice on mixing and spraying, 'not decanting' pesticides, training distributors, improving registration, raising formulation standards. The safe use programmes provide an opportunity to promote pesticide use much more cheaply than through advertising, for example, children can be targeted through the school curriculum: many companies provide cartoon comic papers to schools. Furthermore, government or development agency funds can be sought to support safe use programmes, in direct competition with funding alternatives. The approaches learned from these countries are being applied in other countries. Industry should pursue safe use programmes, but real cost of pesticide use should be reflected in the products, and not compete with the potential to train farmers in Integrated Pest Management (IPM) alternatives which will reduce or eliminate pesticide use.

Corporate IPM

When presenting information at a global level, industry asserts the importance of IPM and the GCPF encourages all products to be marketed under an IPM umbrella. Some companies, notably Novartis and Zeneca, have developed a small number of flagship IPM projects. These have generally been in areas where profound problems have been identified as a result of pesticide overuse. The industry approach to IPM is based on management of pesticides, mainly to ensure that pests do not develop resistance to pesticides. Their work undermines the work by the FAO, many other research institutes and NGOs which have developed an approach to IPM based on no, or minimal, use of pesticides. These alternatives draw on farmer-participatory, knowledge-based strategies which make full use of agricultural biodiversity, beneficial insects, understanding of economic loss, principles of rotation and other good farming practices.

What Industry Doesn't Like?

- ***The precautionary principle***
- ***'Cradle to grave' responsibility for products***
- ***Economic instruments such as pesticide taxes and subsidies for ecological agriculture***
- ***Regulation – instead "always opt for voluntary controls. But codes are also important"***

Strategies for a Sustainable Future

In spite of industry's assertions, most decision-makers recognize that access to food is as important as production. However most could not envisage a pesticide-free agricultural strategy. In the last 50 years, agrochemicals have become so much part of production, that the way out of dependence will take some time, many strategies, and struggles on different fronts. These could include

- Documenting the continuing health and environmental costs of pesticides.
- Demonstration of the continued environmental threats of pesticides, which include, e.g. loss of wild and 'free' food e.g. wild fruits, berries and fish.
- Water pollution: effect on health of humans and animals.
- The benefits of agricultural biodiversity.
- Emerging knowledge: the impact and costs of past ignorance: pesticides, POPs.
- The importance of on farm inputs including recycling of nutrients, preservation of beneficials, and farming knowledge that debunks the myth that low input = low output. Demonstration of successful IPM alternatives should be emphasized.
- Issues related to Food Security. Access is the key word: i.e. access to food, to natural resources and land, access to education, water, credit, seed supplies, technology; access for women; and access to mechanisms of public decision making.
- Developing hunger maps and documenting case studies of impacts: i.e. who are winners and losers at regional/national and sub-national level?
- Asserting the multi-functional role of agriculture: i.e. it is about livelihoods, sustainability and a way of life. Developing countries need to push for recognition of the multi-functional role of agriculture.
- Governments to regulate TNC activities: point to role of TNCs in global trade – it is absurd to pretend that trade is merely between governments.
- Codes of conduct: government, industry and civil society – with adequate monitoring.
- Legally binding mechanisms: e.g. trade rules with environment and social rights.
- Trade rules which provide guidance, not to increase or decrease trade.
- Alliances with sympathetic stakeholders: the public sector and non-corporate agricultural research institutions; development agencies, UN institutions, and academics.
- Influencing the influences: e.g. World Bank, development banks, and government policy makers.

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End note
- Nityanand jayaraman

At least 32 countries have banned its use. Endosulfan's irreparable and often fatal damage to humans and animals is an established fact. Even in Kasaragod, villagers and residents had suspected its role in the numerous health problems and deformities observed in their cattle as early as in the early 1980s. In the year 2000, the extent of the endosulfan disaster in Kasaragod became common knowledge. If that be the case, why did it take more than two years to even temporarily ban it in Kerala? If that be the case, why is aerial spraying banned in Kerala, while in the rest of the country, it continues to be used with similarly devastating effects? And if that be the case, why does the poison remain the most widely used pesticide in India? What engaged our agricultural scientists in the conspiracy of silence, and what made others, including regulators paid by taxpayers, mouth lines fed by the chemical industry?

The answer goes back to the access enjoyed by chemical industries to the corridors of power and decision-making. For instance, at the 221st meeting of the Pesticides Registration Committee held on 18 April 2002 to deliberate on the fate of Endosulfan in the context of the Kasaragod's endosulfan disaster, five non-governmental entities were invited -- Plantation Corporation of Kerala; Excel Industries; Aventis Crop Protection Ltd; Hindustan Insecticides Ltd; EID Parry Ltd. The first company is the accused in the Endosulfan poisoning case; the others are chemical companies whose bottomlines stand to be seriously affected if endosulfan is restricted or banned. None of those affected by endosulfan or their appointees were granted access.

Chemical industries are no strangers to poisons and poisoning. Germany's Hoechst, the original developer of endosulfan, has in its closets skeletons from the Nazi era, when as part of IG Farben -- a chemical industry cartel comprising Hoechst, Bayer and BASF -- it ran the research and development for the Nazis. Farben's numerous, usually fatal, experiments on jewish prisoners yielded products including chemical pesticides and pharmaceutical drugs that are still in use. Hoechst, now Aventis, was invited by our government (see above) to present its views on whether its pesticide endosulfan ought to be banned.

If chemical industries are getting away with murder, the blame lies squarely on our regulators who are either in the pay of the industry or are desensitized into believing that the numerous poisoning cases and deaths caused due to pesticide exposure are actually an acceptable cost for food security. Surely, those killed or maimed or their near and dear ones would not share our regulators' enthusiasm for "food security".

To an extent, the blame also lies with those working in the public interest for not having been able to launch a concerted and well-strategised fight against chemical pesticides. More and more, though, activists, NGOs and other public interest organisations are coming together to correct this inadequacy. In this effort, they have to be able to equip themselves with the skills necessary to assess, quantify and communicate health damage by pesticides, and use such information in fighting for policies discouraging pesticides. Key to this endeavor is a sound knowledge of the enemies -- chemical pesticides and the ways of their manufacturers -- and the putting in place of the infrastructure for pesticide-free agriculture.

Just as wars cannot be ended without strengthening peace, chemical pesticides cannot be eliminated without strengthening natural agriculture.