

Chlorinated Pesticides: Threats to Health and Importance of Detection

Walter J. Crinnion, ND

Abstract

Although chlorinated pesticides have been mostly banned from use in the United States, their persistent presence in the environment poses an ongoing threat to health. Because of the lipophilic nature of chlorinated pesticides, they are bioaccumulative and difficult to excrete from the body. A select group of these xenobiotics is also associated with a wide range of health problems, identification of which would aid in disease prevention and reversal. Ongoing research by the Centers for Disease Control and Prevention now provides national standards for some of these compounds, allowing the clinician to evaluate levels in a patient. Serum samples are easily obtained and can reveal the presence of these xenobiotics. Eight of the most commonly found and harmful chlorinated pesticides are reviewed in this article, along with the most common sources of exposure and possible action steps. (*Altern Med Rev* 2009;14(4):347-359)

Introduction

Multiple studies over recent decades have examined the presence of xenobiotic substances in the blood or adipose tissue of a variety of subjects. A number of these compounds, including the chlorinated pesticide dichlorodiphenyltrichloroethane (DDT) and the industrial polychlorinated biphenyl (PCB) compounds, are stored in the fat tissue of the body and, instead of being easily excreted, continue to “bioaccumulate” over time. A portion of these fat-soluble compounds is passed from mother to child; thus, all new life starts with a toxic load. The load is increased incrementally

through eating, drinking, and breathing, increasing the total toxin burden during the aging process.

The Environmental Working Group (EWG) (www.ewg.org) funded and published two studies that specifically tested adults and newborns in the United States to see how many toxins were carried. The EWG originally tested nine adults, none of whom worked in industries that would ordinarily expose them to high levels of environmental poisons.¹ The nine adults in the EWG study had an average of 91 of the 210 toxic compounds that were tested present in serum, including an average of 33 PCBs and four chlorinated pesticides.

Since these compounds can be passed from mother to child, the EWG designed another study to measure how many chemicals would be found in a random sampling of cord blood from infants in the United States. The EWG newborn study looked for the presence of 413 different xenobiotic chemicals in the cord blood of 10 infants born in U.S. hospitals in 2004.² A total of 287 toxic compounds were found in the cord blood samples, including 147 PCBs and 21 chlorinated pesticides.

While these types of articles are alarming, they do not help the clinician determine whether a patient is carrying an abnormally high load of a particular toxin, or whether the toxin is one that carries documented health risk. To answer the first question, the Centers for Disease Control and Prevention (CDC) has been conducting ongoing studies to identify the toxic burden

Walter Crinnion, ND - 1982 graduate of Bastyr University; practice since 1982 with a special focus on treating chronic diseases caused by environmental toxic burden; conducts post-graduate seminars in environmental medicine; professor and Chair of Environmental Medicine, Southwest College of Naturopathic Medicine; contributing editor, *Alternative Medicine Review*
Email: w.crinnion@scnm.edu

Table 1. Levels of Chlorinated Pesticides Most Commonly Found (with the exception of HCB) in the CDC Third National Report (reported in percentiles)³

Compounds	CDC 50th		CDC 75th		CDC 90th		CDC 95th	
	ppb	ng/g lipid	ppb	ng/g lipid	ppb	ng/g lipid	ppb	ng/g lipid
HCB	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Heptachlor epoxide	<LOD	<LOD	<LOD	<LOD	0.102	14.8	0.153	21.6
Oxychlorthane	0.69	11.1	0.143	21.7	0.248	36.3	0.352	49.7
Trans-nonachlor	0.1112	17.9	0.217	33.7	0.389	56.3	0.589	78.2
DDE (p-p)	1.57	250	3.97	597	8.81	1400	15.4	2320
DDT (p-p)	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	0.184	26.5
Dieldrin	<LOD	<LOD	<LOD	<LOD	0.109	15.2	0.146	20.3
Mirex	<LOD	<LOD	<LOD	<LOD	0.101	15.8	0.414	57.1

LOD = limit of detection

carried by U.S. residents. They have published three reports to date that are available at <http://www.cdc.gov/exposurereport/report.htm>. When a new study is published more compounds are added to the list for testing. Within a few years the number will exceed the total number of toxins the EWG has measured, and will include a greater number of individuals.

Not only does the CDC report provide national “normal ranges,” but the majority of these compounds are measured both as total parts per billion (ppb) in the serum as well as a lipid-adjusted value of ng/g of lipids.³ The levels reported in ppb are reflective of the amount of the toxin present in the serum, mostly found in the cholesterol and albumin fractions.⁴ The lipid-adjusted value provides a more accurate picture of the total burden of these toxins residing in adipose tissue throughout the body.⁵ The amounts stored in adipose tissue are a result of bioaccumulation over a lifetime.

Serum levels reflect the recirculation of toxins released from the adipose tissue from lipolysis. If the individual being tested is going through increased lipolysis due to stress, rigorous exercise, saunas, or weight loss, serum levels may be higher due to increased lipolysis, as a portion of these toxins accompanies cholesterol and triglycerides released from the body’s fat stores. Increased levels, however, can also be reflective of current exposures either from air or food sources.

Using the lipid-adjusted value (measuring the amount of lipids present in the blood and adjusting the amount of toxins to that lipid burden), an accurate assessment of the amount of stored toxins can be obtained, allowing an individual following a cleansing protocol to monitor progress in reducing overall burden of these persistent and accumulating toxins.⁵

Table 1 lists the most common chlorinated pesticides found in the CDC

report. Also, by comparing the two values (ppb in serum and ng/g lipids), current exposure can be more readily determined (high in serum, but the lipid-adjusted value shows low or no stores of this compound).

Common Findings with Serum Testing of Chlorinated Pesticides

Close to 100 percent of all persons tested will have p,p-dichlorodiphenyldichloroethylene (p,p-DDE) in their serum. In a study of 5,994 individuals in Texas, researchers found that increasing age, residing on a farm, and being male increased the risk for a chlorinated pesticide burden.⁶ The levels of p,p-DDE found in this group are shown in Table 2.

Table 2. Levels of Chlorinated Pesticides in a Texas Study⁶

Organochlorine Compounds in the Serum of 5,994 Persons		
Compound	Frequency of finding	Range
p,p-DDE	99.5%	≤1 ppb up to 378.6 ppb
p,p-DDT	10.5%	

The CDC's reported mean levels of DDE are 1.57 ppb and 250 ng/g of lipids. One can also readily observe from the above CDC chart that, in addition to DDE, some individuals will also have the chlordanes oxychlordanes and trans-nonachlor. William J. Rea, MD, at the Environmental Health Center in Dallas, has observed that when individuals have more than one chlorinated pesticide present in their blood, they will have some type of immunotoxicity effect.⁷

Adverse Effects of Chlorinated Pesticides

Chlorinated pesticides as a class are insecticidal primarily via the neurotoxic action of disrupting ion flow, thus interfering with axonal transmission. This leads to over-stimulation of the nerves with uncontrolled neuronal discharge. Acute toxicity (poisoning) signs

and symptoms of chlorinated compounds (headache, nausea, vomiting, hyperesthesias, irritability, confusion, convulsions, respiratory depression, cardiac arrhythmias, aplastic anemia, and porphyria cutanea tarda)⁸ are rarely seen as these compounds have mostly been banned from use since the 1980s. However, since they are fat-soluble and tend to bioaccumulate, they can cause a variety of health problems that often begin slowly. The effects of these compounds are most often seen secondary to mitochondrial toxicity in the neurological, immunological, and endocrinological systems, although they can also affect the cardiovascular, respiratory, gastrointestinal, and other bodily systems.

These organochlorine compounds, with the exception of dieldrin, have been shown to induce the function of 1A and 2B cytochromes, which increases the production of free radicals and reduces glutathione levels (needed to clear phase 1 metabolites from the body).⁹

The adverse health effects of the specific compounds listed in the discussion below under each analyte are those that have been documented in the medical

and scientific literature for that specific compound. The health effects across compounds can be very similar, and many of the studies review a number of analytes together, many of which have been found to be causative agents.

Chlorinated Compounds with the Greatest Documented Health Impact

The following compounds are those associated with the greatest amount of documented adverse health effects in the published medical and scientific literature. With the exception of hexachlorobenzene, they are also the most commonly found pesticides in the CDC Third National Report.

Hexachlorobenzene (HCB)

HCB is a byproduct of chemical solvents, other chlorine-containing compounds, and pesticide manufacturing. Small amounts of HCB can be produced by combustion of waste and other compounds. HCB is an industrial byproduct of the chlor-alkali and wood preservative industry. Hexachlorobenzene was widely used as a pesticide until 1965, but is no longer used commercially in the United States. HCB was also used as a fungicide for control of mold and fungi in cereal grains, primarily wheat. The CDC study did not find HCB often enough to establish any standards, so the presence of any level of HCB is considered abnormal and actionable.

HCB is the only compound reviewed here not found commonly in the CDC study, but is included in this discussion because of its serious health effects when present.

Exposure Sources of HCB

The CDC estimates the average exposure to HCB from foods is 1 mcg/kg body weight. The foods in which HCB was found most commonly, as a part of the Food and Drug Administration's Total Diet Survey, are mostly from the beef/dairy industry, with 25 percent of ground beef, 66 percent of non-organic butter, and 18 percent of American cheese samples containing HCB.¹⁰ HCB was also found in 41 percent of lamb chops and 50 percent of Atlantic salmon (farmed). Of these, the highest levels were found in non-organic butter. High blood levels may also be caused by industrial exposure, such as living close to a waste facility. To help assess these exposure sources one can utilize the databases at www.scorecard.org and <http://www.epa.gov/epahome/commsearch.htm>.

Adverse Health Effects from HCB

The following is a list of the adverse health effects of HCB exposure.

- ▶ Diabetes risk is increased (odds ratio [OR] 4.5) by the presence of HCB.¹¹
- ▶ Childhood obesity is increased (OR 2.5-3.0) with maternal serum levels of HCB.¹²
- ▶ Testicular cancer rates are higher in men (OR 4.4) whose mothers had high serum HCB levels.¹³

- ▶ Reduction of total T4 occurs with increased levels of HCB (T4 dropped 0.32 mcg/dL per each unit increase [in ng/mL] of HCB).¹⁴
- ▶ Increased rates of soft-tissue sarcomas and thyroid cancers occur in persons living close to an industry that emits HCB.¹⁵
- ▶ Porphyria with neurological manifestations can result from HCB exposure.¹⁶
- ▶ Risk for childhood otitis media (OR 2.38) is increased when HCB is present along with DDE.¹⁷
- ▶ Epstein Barr early antigen and risk for non-Hodgkins lymphoma (OR 5.3 with "above median levels of HCB") increase.¹⁸
- ▶ HCB exposure may be related to increased risk of autoimmunity (animal study evidence).¹⁹
- ▶ Chronic fatigue syndrome patients have higher levels of HCB and/or DDE.²⁰
- ▶ HCB can suppress gamma-interferon production.²¹

Heptachlor Epoxide (HCE)

Heptachlor and its metabolite heptachlor epoxide (HCE) are chlordanes, a group of chlorinated compounds used agriculturally until 1974 and as termiticides commonly throughout North America until 1988. Bacteria in the soil, as well as the livers of humans and animals, can transform heptachlor through cytochrome 1A1 (phase 1) to the much more toxic and biologically persistent epoxide form. Once a house has been treated with chlordane, the chlordanes can be found in dust for the life of the home, contaminating anyone living in the home who breathes the dust. Heptachlor is still approved for the treatment of fire ants in underground transformers.

Exposure Sources of HCE

A major source of exposure is living in a home built before 1988 in which chlordanes were applied in the crawl-space under the house where the furnace or air conditioning ducts now run. If those ducts have leaking connections (common in older duct work), the chlordane-contaminated dust can be sucked into the pipes and distributed throughout the house. This can occur no matter what grade of filter is on the furnace, because the contamination occurs post-filter. Since heptachlor

and HCE stick to soil and dust, another source comes from tracking dirt into a building from outdoors. In many areas of the country, former orchards or farmland have been turned into housing developments. The areas where heptachlor was used before 1974 include soil contaminated with HCE that can make it into the dirt and dust of the home, especially homes where shoes are worn indoors and that have wall-to-wall carpeting to which HCE can adhere.

The next greatest source of exposure is through foods, typically seafood, dairy, meats, and poultry. In the Total Diet Survey,¹⁰ the foods highest in heptachlor epoxide are very similar to those in which HCB was found. Fifty percent of the butter samples (non-organic) contained HCE. It was also found in 34 percent of cream cheese and salmon steaks (farmed Atlantic salmon), 32 percent of ground beef, and 30 percent of Swiss and cheddar cheese samples. Interestingly, 25 percent of samples of Hubbard squash also contained HCE.

Chlordanes have been found in all studies of breast milk in North America. Thus, children may experience transplacental transfer of chlordanes as well as breast milk exposure.

Adverse Health Effects of HCE

The following are adverse health effects of HCE.

- ▶ HCE is a powerful pro-oxidant and difficult to clear through normal phase 2 detoxification.
- ▶ High maternal levels of HCE lead to increased rates of cryptorchidism in male offspring.²²
- ▶ HCE has demonstrated the ability to be an initiator, promoter, and progressor of breast cancer.^{23,24}
- ▶ Higher HCE blood levels increase risk of non-Hodgkins lymphoma (third quartile levels [OR 1.82]; fourth quartile levels [OR 3.41]).²⁵
- ▶ HCE is neurotoxic to the dopaminergic system and may lead to increased risk for parkinsonism.²⁶
- ▶ HCE presence, along with other chlorinated compounds, can lead to increased risk of atherosclerosis.²⁷

Oxychlordane

Oxychlordane is the major metabolite of the various chlordane and nonachlor compounds used agriculturally until 1974 and residentially until 1988. It is found commonly in all persons living in North America when serum levels are tested. As a human metabolite, it has not been found in food, since it is produced by the liver after exposure to chlordanes and nonachlors from air, water, or food. This metabolite is eight times more toxic than its parent compounds and is more bioaccumulative.²⁸

Exposure Sources of Oxychlordane

See the above list for HCE, as all the chlordanes were often present together in whatever mixture was used agriculturally or residentially.

Adverse Health Effects of Oxychlordane

Adverse health effects of oxychlordane include the following.

- ▶ Increased risk of diabetes (OR 14.7) with high levels of oxychlordane.²⁹
- ▶ Increased risk of non-Hodgkin's lymphoma (OR 2.68).³⁰
- ▶ Increased risk for seminoma (testicular germ cell tumor) (OR 1.63).³¹
- ▶ Increased risk of prostate cancer.³²
- ▶ *In vitro* evidence of immunosuppression of cell-mediated immune response to pathogens.³³
- ▶ Decreased natural-killer cell ability to lyse tumor cells.³⁴

Trans-Nonachlor

Trans-nonachlor is another of the major chlordane compounds used agriculturally from 1953-1974 and as a termiticide until 1988.

Exposure Sources of Trans-Nonachlor

The same exposure sources listed for the other chlordanes also apply here. The most likely source is exposure to dirt or dust that is already contaminated with it, especially in homes built before 1988 that have the original ductwork still intact. Unfortunately, there were a number of individual homeowners who chose to apply chlordane themselves, and, in these cases, chlordane may be inside the home itself.

Dietary sources of trans-nonachlor were surprisingly few in the Total Diet Survey, with the most frequent finding being in sweet cucumber pickles (25% of samples), rather than dairy and beef like the other chlordanes. Trans-nonachlor is found in most persons tested for it.

Adverse Health Effects

The following are adverse effects associated with trans-nonachlor exposure.

- ▶ The adverse effects of the chlordanes are all very similar. Many of the cancer and other adverse health associations are found with trans-nonachlor as well, but at differing levels of risk (different odds ratios).
- ▶ Exposure to trans-nonachlor results in increased risk of obesity and diabetes, with the highest odds ratio for diabetes of the organochlorine compounds (OR 37.7).²⁹

DDE

DDE, a DDT metabolite, is the most ubiquitous and abundant of the chlorinated pesticides.⁶ When DDT is produced, it consists of a combination of both DDT and DDE. The rate of breakdown in the environment (in temperate climates the half-life of DDT in soil is 20-30 years) is measured by the changing ratios of DDE to DDT. Once in the human body, DDT is broken down to DDE within about six months. Many published research articles use the term DDT or total DDT to include DDT, DDE, and DDD. Since DDE is the most commonly found DDT compound, the main aspects of the DDTs will be reviewed under DDE.

DDT was first synthesized in 1874. Its pesticide ability was discovered in 1939 and used in wartime to control typhus and malaria. It was put into agricultural use in the United States in 1945 and banned from use in 1972. Production in this country, however, was not banned so it was still manufactured and sent to other countries, often as part of U.S. agricultural aid. The DDTs are found throughout the globe, including the Arctic and Antarctic, because they have been carried on the winds across the planet.

The DDTs are highly lipid-soluble and are stored in the lipid-rich tissues of the body, including adipose tissue, the liver, and the brain. It has been

estimated that 1 ppb DDT in serum means 5-10 ppb in brain, 47 ppb in liver, and 100-300 ppb in fat cells. DDE has been found in all samples of breast milk around the world. Unfortunately, the level of DDE in some of these samples exceeds what is allowed to be sold commercially in any other milk product.³⁵

Exposure Sources of DDE

The diet is the major source of exposure to DDE/DDT. From 1986-1991, adults in the United States consumed an average of 0.8 mcg of DDT daily. The largest amount of dietary DDT comes from meat, poultry, dairy products, and fish, including sport fish. A number of fresh-water fish advisories in certain lakes and rivers in the United States are posted because of DDT contamination of trout and other fish. Leafy vegetables often contain more DDT than other vegetables, possibly because DDT in the air is deposited on the leaves. Infants can be exposed by drinking breast milk.

The most recent Total Diet Survey assessment of DDE in food reveals that DDE is one of the most ubiquitous toxins in foods.¹⁰ Table 3 shows those foods in which it was found commonly in this survey. In each percentage grouping, the most contaminated foods are listed first.

A listing of the 10 foods with the highest DDE content according to this survey are:

- ▶ Catfish (farm-raised)
- ▶ Butter (non-organic)
- ▶ Spinach (non-organic)
- ▶ Atlantic salmon (farm-raised)
- ▶ American cheese
- ▶ Lamb chops
- ▶ Collard greens (non-organic)
- ▶ Cream cheese (non-organic)
- ▶ Quarter-pound cheeseburger
- ▶ Cheddar cheese (non-organic)

The other main source is DDE-contaminated dust or dirt in a home. This could occur in an older dwelling where DDT was used or in newer housing developments built on or around previously contaminated soil. Toxic levels can also come from airborne contamination when DDE/DDT is used agriculturally in other parts of the world and makes its way across the globe on the winds.

Table 3. Common Food Sources of DDE¹⁰

Food	Percentage of samples positive for DDE	Mean concentration of DDE in ppm
Catfish (farm raised)	100%	0.032
Butter (non-organic)	100%	0.02
Spinach (non-organic)	100%	0.01
Salmon steaks (farmed Atlantic)	100%	0.008
American cheese, processed	100%	0.006
Cream cheese	100%	0.004
Candy bar, chocolate, nougat, nuts	100%	0.001
Quarter pound cheeseburger	98%	0.004
Meatloaf (homemade)	98%	0.002
Quarter pound burger	98%	0.002
Lamb chops	95%	0.005
Sour cream	93%	0.0028
Fast food egg, ham, biscuit breakfast	93%	0.0024
Pizza, pepperoni	93%	0.0012
Ground beef	91%	0.002
Pizza, cheese	90%	0.0015
Collard greens, non-organic	89%	0.0045
Hot dogs	89%	0.002
Half-and-half	82%	0.002
Vanilla ice cream	80%	0.0012
Lasagna, beef	80%	0.0008
Cheddar cheese	77%	0.003
Peanut butter, creamy	75%	0.0016
Baked potato, with peel (non-organic)	73%	0.0013

Sport fishing is another potential source of DDE exposure for persons who consume their catch. State regulatory agencies periodically issue advisories for toxic fish present in their waterways. Anyone planning on fishing in a particular state, including coastal waterways, should avail themselves of this information before consuming the catch.

All children are exposed *in utero* and via breast milk if breast fed.

Adverse Health Effects of DDE

In general, DDE causes ongoing neurological problems (including cognitive difficulties, headaches, and depression), along with immune and endocrine problems. Various cancers are also associated with DDE presence. Breast cancer is not listed below because there have been, and continue to be, published articles that do show a correlation and others that do not. One intriguing study reported that when DDT exposure occurred before age 14, those women have higher rates of breast cancer.³⁶ Other health risks include the following:

- ▶ Prenatal exposure to DDE causes hyporeflexia³⁷ and attention problems³⁸ in infants. Exposure while breastfeeding can cause delays in mental and psychomotor development identifiable at the age of 13 months.³⁹ *In utero* exposure can also cause impairment of cognitive skills still evident when the infants become preschoolers.⁴⁰
- ▶ Prenatal exposure to DDE, HCB, and dieldrin increases the risk of otitis media, and higher levels increase the risk of recurrent otitis media.⁴¹
- ▶ Prenatal DDE exposure increases the rate of asthma in children (relative risk of 2.6 for the highest levels of DDE).⁴²
- ▶ DDE increases the rate of mast-cell degranulation and increases risk of allergy and asthma.^{17,43,44}
- ▶ Elevated serum DDE significantly reduces mitogen-induced lymphocyte proliferation response,⁴⁵ resulting in cell-mediated immune deficiency and possible increased risk of herpes zoster.⁴⁶
- ▶ DDE (and other chlorinated pesticides) are found in higher levels in the substantia nigra in persons with Parkinson's disease,⁴⁷ and *in vitro* have

been found to disrupt the transport of dopamine in the brain.⁴⁸

- ▶ DDE and HCB are associated with chronic fatigue syndrome.²⁰
- ▶ DDE is associated with higher rates of type 2 diabetes.^{11,49}
- ▶ DDE is associated with a 71-percent increased risk of developing testicular germ cell tumors.³¹
- ▶ Persons with above-median levels of DDE (and other chlorinated compounds) may have increased risk of developing pancreatic cancer,⁵⁰ and are prone to significantly shorter survival times should they develop the disease.⁵¹
- ▶ High DDE levels almost double the risk (OR 1.9) for endometrial cancer.⁵²
- ▶ DDE and other environmental endocrine disruptors have been associated with increased rates of precocious puberty.⁵³
- ▶ High concentration of DDE is associated with preterm births and small-for-gestational-age babies.⁵⁴
- ▶ DDE was the most frequently found chlorinated compound in a study of infertile females and their male partners, with the highest residue levels being negatively associated with fertilization.⁵⁵
- ▶ DDE levels are associated with multiple abnormalities in semen indices and sperm count, motility, and quality.⁵⁶
- ▶ DDE toxicity can lead to early menopause.⁵⁷
- ▶ DDE is associated with greater risk of endometriosis and reduced functioning of natural-killer cells.⁵⁸
- ▶ DDE exposure *in utero* can lead to altered levels of thyroid hormones.⁵⁹

DDT

Refer to the above section on DDE. The sources of DDT are similar, except DDT is present in much smaller quantities. High DDT levels are associated with a greatly elevated risk for liver cancer.⁶⁰ The health problems of DDT exposure, on the whole, are virtually identical to those of DDE. The main difference is the frequency with which DDE and DDT are found and their respective levels. While a small amount of DDT

is found in the adipose tissue of almost everybody, it is rarely found in the serum. Due to the time it takes for DDT to be metabolized to DDE in the body, detection of DDT in the serum typically indicates current exposure (probably from food sources) within the preceding six months. In the Total Diet Survey the greatest source of DDT was non-organic spinach.¹⁰

Dieldrin

From the 1950s until 1970, dieldrin and a similar compound aldrin were used extensively as insecticides on crops such as corn and cotton. They were both approved by the U.S. Environmental Protection Agency in 1972 for killing termites and were used as such until 1987. Aldrin is metabolized into dieldrin after entering the body or the environment (where sunlight and bacteria bring about the production of dieldrin). Dieldrin does not break down in water and is not easily volatilized to release into the air. It attaches very strongly to soil, sediment, and dust particles, and can be taken up by plants and stored in leaves and roots. Fish or animals that eat dieldrin-contaminated materials store a large amount of it in fat tissue. Animals or fish that eat other animals have fat levels of dieldrin many times higher because of bioaccumulation. Humans, at the top of the food chain, are the final repository for dieldrin.

Exposure Sources of Dieldrin

The most common exposure to aldrin and dieldrin occurs from contaminated foods, including fish or shellfish from contaminated lakes or streams, root crops, dairy products, and meats. Exposure to aldrin and dieldrin also occurs from water, air, or contact with contaminated soil at hazardous waste sites (check for these at www.epa.gov). Individuals with the greatest potential for exposure include those who live in homes previously treated for termites using aldrin or dieldrin, because exposure to these toxins can occur years after they were applied.

Squash, cucumber, and zucchini plants have the ability to take up aldrin and dieldrin from the soil, where it accumulates in the portion we typically eat.⁶¹ This is reflected in the 10 foods listed in the Total Diet Survey¹⁰ as having the highest levels of dieldrin. Even organic vegetables will contain high levels of these toxins if grown in soil contaminated decades earlier by aldrin or dieldrin. While these plants can be used to

remove such pesticides from the soil, it is not recommended to eat or compost them. The following are the 10 foods with the highest levels of dieldrin according to the Total Diet Survey.

- ▶ Summer squash (0.007 ppm)
- ▶ Hubbard squash (0.005 ppm)
- ▶ Dill cucumber pickles (0.004 ppm)
- ▶ Sweet cucumber pickles (0.003 ppm)
- ▶ Raw, peeled cucumbers (0.003 ppm)
- ▶ Pumpkin pie (0.0024 ppm)
- ▶ Atlantic salmon (0.002 ppm)
- ▶ Cream cheese (0.0008 ppm)
- ▶ Creamy peanut butter (0.00076 ppm)
- ▶ Cheddar cheese (0.0007 ppm)

Adverse Health Effects of Dieldrin

The following are the most significant adverse health effects of dieldrin exposure.

- ▶ Dieldrin disrupts dopamine transport in the brain.⁴⁸
- ▶ Dieldrin increases oxidative damage in the brain (nigrostriatal pathway),⁶² and may lead to increased rates of parkinsonism.⁶³ A significant association has also been observed between dieldrin toxicity and already-diagnosed Parkinson's disease.⁶⁴
- ▶ Dieldrin is associated with increased rates of hypothyroidism.⁶⁵
- ▶ Dieldrin adversely affects the Leydig cells, reducing their production of testosterone, which could contribute to infertility.⁶⁶
- ▶ Dieldrin is associated with increased rates of lung cancer,⁶⁷ breast cancer,⁶⁸ and non-Hodgkin's lymphoma.²⁵
- ▶ Dieldrin may increase risk of pancreatic cancer mortality.⁶⁹
- ▶ Dieldrin increases superoxide production and causes neutrophil inflammation.⁷⁰

Mirex

Mirex was used as a pesticide to control fire ants, mostly in the southeastern United States until 1978. It was used as a flame retardant additive under the trade name Dechlorane® in plastics, rubber, paint,

paper, and electrical goods from 1959 to 1972. Mirex is found attached to soil and dust, like many of these other compounds.

Once in the bloodstream, mirex is carried to many parts of the body where it is stored, mainly in fat. Mirex is not broken down in the body by biotransformation processes.

Exposure Sources of Mirex

The most likely way for the general population to be exposed to mirex is by eating food, particularly fish, taken from contaminated waters. Currently, three states (Ohio, New York, and Pennsylvania) have issued warnings that fish (primarily caught in Lake Ontario) may contain mirex. The FDA's Total Diet Survey has not measured other foods for mirex; thus, exposure sources are not clearly delineated.

Adverse Health Effects of Mirex

In rodents, mirex causes liver, adrenal, and blood cancer. In humans it causes trembling, tiredness, weakness, increased oxidative damage, and neurological and immunological problems.⁷² Most of the published studies examining adverse health effects of mirex were conducted while studying a number of chlorinated compounds. As a single agent, it has not been well studied so no specific articles are noted.

The Presence of Multiple Chlorinated Pesticides

Most of the studies referenced in this article reviewed multiple chlorinated compounds. Some studies demonstrate synergism and some show adverse health effects due to the total toxic load. The studies that have not shown one compound as more damaging than the others have not been referenced here, but are readily available on PubMed (<http://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed>).

Action Steps

The first step is to identify exposure sources and remediate them. Cleansing protocols can be implemented to enhance the clearance of these persistent toxins from the body. Sauna therapy and colonic irrigations have been used to reduce the presence of PCBs and chlorinated pesticides.⁷³ Various dietary measures have been shown to increase normal bowel excretion of

these fat-soluble toxins. The daily use of rice bran fiber has been documented in animal and human studies in Japan to increase the clearance of PCBs.⁷³⁻⁷⁵

Chlorophyll and chlorophyll-containing foods have been shown to increase excretion of these fat-soluble persistent toxins via the feces.⁷⁶⁻⁷⁸ Increasing chlorophyll-containing foods or daily supplementation with chlorophyll can slowly increase the excretion of these compounds. In addition to chlorophyll-containing agents, polyphenols in white and green teas have been shown to increase the excretion of fecal fat that carries fat-soluble toxins with it.⁷⁹ In addition to increasing the excretion of these toxins from the body, supplementing with nutrient and botanical antioxidants to protect the tissues and cells targeted by these toxic compounds should be considered.

Summary

Chlorinated pesticides were first used in the 1940s. Although they have now been mostly banned from use in the United States, their typically long half-life contributes to ongoing risk of exposure. They are fat-soluble compounds difficult to eliminate from the body and they consequently accumulate in adipose tissue of mammalian species. Because adipose tissue goes through daily lipolysis, a certain amount of these xenobiotics are regularly released back into circulation. These compounds can be measured in the serum and reported both as ppb in the serum and as lipid-adjusted amounts. The lipid-adjusted values reflect the amount of these toxins stored in the adipose tissue and therefore reflect total body burden. The ppb measurements reflect the amount in circulation, either from current exposure or daily release from adipose tissue. The CDC has recently provided basic reference values to help clinicians interpret the reported values of these xenobiotics. Of the chlorinated pesticides, heptachlor epoxide, oxychlorodane, trans-nonachlor, DDE (p,p-1), DDT (p,p-1), dieldrin, and mirex are the most commonly found and are associated with the greatest documented detrimental health effects. Hexachlorobenzene has numerous documented adverse health effects, although it is not commonly found in the serum.

Many of these chlorinated pesticides are still present in the listed food sources and can be easily avoided. In addition to avoiding ongoing exposure to these compounds, the addition of brown rice, rice bran

fiber, chlorophyll, and green tea to the diet can enhance the excretion of these toxic compounds and help reduce the total toxic burden.

Testing for these compounds permits a clinician to identify persons who may need such treatments to help prevent exacerbation of their health problems or to reverse their health complaints.

References

1. <http://archive.ewg.org/reports/bodyburden1/> [Accessed October 7, 2009]
2. <http://archive.ewg.org/reports/bodyburden2/> [Accessed October 7, 2009]
3. <http://www.cdc.gov/exposurereport/report.htm> [Accessed October 7, 2009]
4. Noren K, Westrand C, Karpe F. Distribution of PCB congeners, DDE, hexachlorobenzene, and methylsulfonyl metabolites of PCB and DDE among various fractions of human blood plasma. *Arch Environ Contam Toxicol* 1999;37:408-414.
5. Patterson DG Jr, Needham LL, Pirkle JL, et al. Correlation between serum and adipose tissue levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin in 50 persons from Missouri. *Arch Environ Contam Toxicol* 1988;17:139-143.
6. Stehr-Green PA. Demographic and seasonal influences on human serum pesticide residue levels. *J Toxicol Environ Health* 1989;27:405-421.
7. Rea WJ. Personal communication.
8. <http://www.atsdr.cdc.gov/toxprofiles/tp35.html> [Accessed October 7, 2009]
9. Dehn PF, Allen-Mocherie S, Karek J, Thenappan A. Organochlorine insecticides: impacts on human HepG2 cytochrome P4501A, 2B activities and glutathione levels. *Toxicol In Vitro* 2005;19:261-273.
10. <http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/Pesticides/ResidueMonitoringReports/ucm125183.htm> [Accessed October 9, 2009]
11. Codru N, Schymura MJ, Negoita S, et al. Diabetes in relation to serum levels of polychlorinated biphenyls and chlorinated pesticides in adult Native Americans. *Environ Health Perspect* 2007;115:1442-1447.
12. Smink A, Ribas-Fito N, Garcia R, et al. Exposure to hexachlorobenzene during pregnancy increases the risk of overweight in children aged 6 years. *Acta Paediatr* 2008;97:1465-1469.
13. Hardell L, van Bavel B, Lindstrom G, et al. Increased concentrations of polychlorinated biphenyls, hexachlorobenzene, and chlordanes in mothers of men with testicular cancer. *Environ Health Perspect* 2003;111:930-934.
14. Sala M, Sunyer J, Herrero C, et al. Association between serum concentrations of hexachlorobenzene and polychlorobiphenyls with thyroid hormone and liver enzymes in a sample of the general population. *Occup Environ Med* 2001;58:172-177.
15. Grimalt JO, Sunyer J, Moreno V, et al. Risk excess of soft-tissue sarcoma and thyroid cancer in a community exposed to airborne organochlorinated compound mixtures with a high hexachlorobenzene content. *Int J Cancer* 1994;56:200-203.
16. Peters HA, Cripps DJ, Gocmen A, et al. Neurotoxicity of hexachlorobenzene-induced prophyria turcica. *IARC Sci Publ* 1986;77:575-579.
17. Karmaus W, Kuehr J, Kruse H. Infections and atopic disorders in childhood and organochlorine exposure. *Arch Environ Health* 2001;56:485-492.
18. Hardell E, Eriksson M, Lindstrom G, et al. Case-control study on concentrations of organohalogen compounds and titers of antibodies to Epstein-Barr virus antigens in the etiology of non-Hodgkin lymphoma. *Leuk Lymphoma* 2001;42:619-629.
19. Michielsen CC, van Loveren H, Vos JG. The role of the immune system in hexachlorobenzene-induced toxicity. *Environ Health Perspect* 1999;107:783-792.
20. Dunstan RH, Donohoe M, Taylor W, et al. A preliminary investigation of chlorinated hydrocarbons and chronic fatigue syndrome. *Med J Aust* 1995;163:294-297.
21. Daniel V, Huber W, Bauer K, et al. Associations of blood levels of PCB, HCHS, and HCB with numbers of lymphocyte subpopulations, *in vitro* lymphocyte response, plasma cytokine levels, and immunoglobulin autoantibodies. *Environ Health Perspect* 2001;109:173-178.
22. Pierik FH, Klebanoff MA, Brock JW, Longnecker MP. Maternal pregnancy serum level of heptachlor epoxide, hexachlorobenzene, and beta hexachlorocyclohexane and risk of cryptorchidism in offspring. *Environ Res* 2007;105:364-369.
23. Cassidy RA, Natarajan S, Vaughan GM. The link between the insecticide heptachlor epoxide, estradiol, and breast cancer. *Breast Cancer Res Treat* 2005;90:55-64.
24. Khanjani N, English DR, Sim MR. An ecological study of organochlorine pesticides and breast cancer in rural Victoria, Australia. *Arch Environ Contam Toxicol* 2006;50:452-461.
25. Quintana PJ, Delfino RJ, Korrick S, et al. Adipose tissue levels of organochlorine pesticides and polychlorinated biphenyls and risk of non-Hodgkin's lymphoma. *Environ Health Perspect* 2004;112:854-861.
26. Richardson JR, Caudle WM, Wang MZ, et al. Developmental heptachlor exposure increases susceptibility of dopamine neurons to N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) in a gender-specific manner. *Neurotoxicology* 2008;29:855-863.

27. Pines A, Cucos S, Ever-Hadani P, et al. Levels of some organochlorine residues in blood of patients with arteriosclerotic disease. *Sci Total Environ* 1986;54:135-155.
28. Bondy G, Armstrong C, Coady L, et al. Toxicity of the chlordane metabolite oxychlordane in female rats: clinical and histopathological changes. *Food Chem Toxicol* 2003;41:291-301.
29. Lee DH, Lee IK, Song K, et al. A strong dose-response relation between serum concentrations of persistent organic pollutants and diabetes: results from the National Health and Examination Survey 1999-2002. *Diabetes Care* 2006;29:1638-1644.
30. Spinelli JJ, Ng CH, Weber JP, et al. Organochlorines and risk of non-Hodgkin lymphoma. *Int J Cancer* 2007;121:2767-2775.
31. McGlynn KA, Quraishi SM, Graubard BI, et al. Persistent organochlorine pesticides and risk of testicular germ cell tumors. *J Natl Cancer Inst* 2008;100:663-671.
32. Ritchie JM, Vial SL, Fuortes LJ, et al. Organochlorines and risk of prostate cancer. *J Occup Environ Med* 2003;45:692-702.
33. Johnson KW, Kaminski NE, Munson AE. Direct suppression of cultured spleen cell responses by chlordane and the basis for differential effects on *in vivo* and *in vitro* immunocompetence. *J Toxicol Environ Health* 1987;22:497-515.
34. Beach TM, Whalen MM. Effects of organochlorine pesticides on interleukin secretion from lymphocytes. *Hum Exp Toxicol* 2006;25:651-659.
35. Ennaceur S, Gandoura N, Driss MR. Distribution of polychlorinated biphenyls and organochlorine pesticides in human breast milk from various locations in Tunisia: levels of contamination, influencing factors, and infant risk assessment. *Environ Res* 2008;108:86-93.
36. Cohn BA, Wolff MS, Cirillo PM, Sholtz RI. DDT and breast cancer in young women: new data on the significance of age at exposure. *Environ Health Perspect* 2007;115:1406-1414.
37. Rogan WJ, Gladen BC, McKinney JD, et al. Neonatal effects of transplacental exposures to PCBs and DDE. *J Pediatr* 1986;109:335-341.
38. Sagiv SK, Nugent JK, Brazelton TB, et al. Prenatal organochlorine exposure and measures of behavior in infancy using the Neonatal Behavioral Assessment Scale (NBAS). *Environ Health Perspect* 2008;116:666-673.
39. Ribas-Fito N, Cardo E, Sala M, et al. Breastfeeding, exposure to organochlorine compounds, and neurodevelopment in infants. *Pediatrics* 2003;111:e580-e585.
40. Ribas-Fito N, Torrent M, Carrizo D, et al. *In utero* exposure to background concentrations of DDT and cognitive functioning among preschoolers. *Am J Epidemiol* 2006;164:955-962.
41. Dewailly E, Ayotte P, Burneau S, et al. Susceptibility to infections and immune status in Inuit infants exposed to organochlorines. *Environ Health Perspect* 2000;108:205-211.
42. Sunyer J, Torrent M, Garcia-Esteban R, et al. Early exposure to dichlorodiphenyldichloroethylene, breastfeeding and asthma at age six. *Clin Exp Allergy* 2006;36:1236-1241.
43. Narita S, Goldblum RM, Watson CS, et al. Environmental estrogens induce mast cell degranulation and enhance IgE-mediated release of allergic mediators. *Environ Health Perspect* 2007;115:48-52.
44. Sunyer J, Torrent M, Munoz-Ortiz L, et al. Prenatal dichlorodiphenyldichloroethylene (DDE) and asthma in children. *Environ Health Perspect* 2005;113:1787-1790.
45. Vine MF, Stein L, Weigle K, et al. Effects on the immune system associated with living near a pesticide dump site. *Environ Health Perspect* 2000;108:1113-1124.
46. Arndt V, Vine MF, Weigle K. Environmental chemical exposures and risk of herpes zoster. *Environ Health Perspect* 1999;107:835-841.
47. Corrigan FM, Wienburg CL, Shore RF, et al. Organochlorine insecticides in substantia nigra in Parkinson's disease. *J Toxicol Environ Health A* 2000;59:229-234.
48. Hatcher JM, Delea KC, Richardson JR, et al. Disruption of dopamine transport by DDT and its metabolites. *Neurotoxicology* 2008;29:682-690.
49. Rignell-Hydbom A, Rylander L, Hagmar L. Exposure to persistent organochlorine pollutants and type 2 diabetes mellitus. *Hum Exp Toxicol* 2007;26:447-452.
50. Porta M, de Barea MB, Benavides FG, et al. Differences in serum concentrations of organochlorine compounds by occupational social class in pancreatic cancer. *Environ Res* 2008;108:370-379.
51. Hardell L, Carlberg M, Hardell K, et al. Decreased survival in pancreatic cancer patients with high concentrations of organochlorines in adipose tissue. *Biomed Pharmacother* 2007;61:659-664.
52. Hardell L, van Bavel B, Lindstrom G, et al. Adipose tissue concentrations of p,p'-DDE and the risk for endometrial cancer. *Gynecol Oncol* 2004;95:706-711.
53. Lu JP, Zheng LX, Cai DP. Study on the level of environmental endocrine disruptors in serum of precocious puberty patients. *Zhonghua Yu Fang Yi Xue Za Zhi* 2006;40:88-92. [Article in Chinese]

54. Longnecker MP, Klebanoff MA, Zhou H, Brock JW. Association between maternal serum concentration of the DDT metabolite DDE and preterm and small-for-gestational-age babies at birth. *Lancet* 2001;358:110-114.
55. Younglai EV, Foster WG, Hughes EG, et al. Levels of environmental contaminants in human follicular fluid, serum, and seminal plasma of couples undergoing *in vitro* fertilization. *Arch Environ Contam Toxicol* 2002;43:121-126.
56. Aneck-Hahn NH, Schulenburg GW, Bornman MS, et al. Impaired semen quality associated with environmental DDT exposure in young men living in a malaria area in the Limpopo Province, South Africa. *J Androl* 2007;28:423-434.
57. Akkina J, Reif J, Keefe T, Bachand A. Age at natural menopause and exposure to organochlorine pesticides in Hispanic women. *J Toxicol Environ Health A* 2004;67:1407-1422.
58. Quaranta MG, Porpora MG, Mattioli B, et al. Impaired NK-cell-mediated cytotoxic activity and cytokine production in patients with endometriosis: a possible role for PCBs and DDE. *Life Sci* 2006;79:491-498.
59. Asawasinsopon R, Prapamontol T, Prakobvitayakit O, et al. The association between organochlorine and thyroid hormone levels in cord serum: a study from northern Thailand. *Environ Int* 2006;32:554-559.
60. McGlynn KA, Abnet CC, Zhang M, et al. Serum concentrations of 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT) and 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) and risk of primary liver cancer. *J Natl Cancer Inst* 2006;98:1005-1010.
61. Donnarumma L, Pompei V, Faraci A, Conte E. Uptake of organochlorine pesticides by zucchini cultivars grown in polluted soils. *Commun Agric Appl Biol Sci* 2008;73:853-859.
62. Hatcher JM, Richardson JR, Guillot TS, et al. Dieldrin exposure induces oxidative damage in the mouse nigrostriatal dopamine system. *Exp Neurol* 2007;204:619-630.
63. Richardson JR, Caudle WM, Wang M, et al. Developmental exposure to the pesticide dieldrin alters the dopamine system and increases neurotoxicity in an animal model of Parkinson's disease. *FASEB J* 2006;20:1695-1697.
64. Fleming L, Mann JB, Bean J, et al. Parkinson's disease and brain levels of organochlorine pesticides. *Ann Neurol* 1994;36:100-103.
65. Rathore M, Bhatnagar P, Mathur D, Saxena GN. Burden of organochlorine pesticides in blood and its effect on thyroid hormones in women. *Sci Total Environ* 2002;295:207-215.
66. Fowler PA, Abramovich DR, Haites NE, et al. Human fetal testis Leydig cell disruption by exposure to the pesticide dieldrin at low concentrations. *Hum Reprod* 2007;22:2919-2927.
67. Purdue MP, Hoppin JA, Blair A, et al. Occupational exposure to organochlorine insecticides and cancer incidence in the Agricultural Health Study. *Int J Cancer* 2007;120:642-649.
68. Hoyer AP, Grandjean P, Jorgensen T, et al. Organochlorine exposure and risk of breast cancer. *Lancet* 1998;352:1816-1820.
69. Clary T, Ritz B. Pancreatic cancer mortality and organochlorine pesticide exposure in California, 1989-1996. *Am J Ind Med* 2003;43:306-313.
70. Pelletier M, Girard D. Dieldrin induces human neutrophil superoxide production via protein kinases C and tyrosine kinases. *Hum Exp Toxicol* 2002;21:415-420.
71. <http://www.atsdr.cdc.gov/toxprofiles/tp66.html> [Accessed October 7, 2009]
72. Crinnion W. Unpublished research. Southwest College of Naturopathic Medicine.
73. Morita K, Hamamura K, Iida T. Binding of PCB by several types of dietary fiber *in vivo* and *in vitro*. *Fukuoka Igaku Zasshi* 1995;86:212-217. [Article in Japanese]
74. Morita K, Hirakawa H, Matsueda T, et al. Stimulating effect of dietary fiber on fecal excretion of polychlorinated dibenzofurans (PCDF) and polychlorinated dibenzo-p-dioxins (PCDD) in rats. *Fukuoka Igaku Zasshi* 1993;84:273-281. [Article in Japanese]
75. Nagayama J, Takasuga T, Tsuji H, et al. Active elimination of causative PCDFs/DDs congeners of Yusho by one year intake of FBRA in Japanese people. *Fukuoka Igaku Zasshi* 2003;94:118-125.
76. Morita K, Ogata M, Hasegawa T. Chlorophyll derived from chlorella inhibits dioxin absorption from the gastrointestinal tract and accelerates dioxin excretion in rats. *Environ Health Perspect* 2001;109:289-294.
77. Morita K, Matsueda T, Iida T. Effect of green vegetable on digestive tract absorption of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans in rats. *Fukuoka Igaku Zasshi* 1999;90:171-183. [Article in Japanese]
78. Morita K, Matsueda T, Iida T, Hasegawa T. Chlorella accelerates dioxin excretion in rats. *J Nutr* 1999;129:1731-1736.
79. Hsu TF, Kusumoto A, Abe K, et al. Polyphenol-enriched oolong tea increases fecal lipid excretion. *Eur J Clin Nutr* 2006;60:1330-1336.