Environmental Medicine, Part 4: Pesticides – Biologically Persistent and Ubiquitous Toxins.

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Abstract
Although the use of pesticides has doubled every ten years since 1945, pest damage to crops is more prevalent now than it was then. Many pests are now pesticide resistant due to the ubiquitous presence of pesticides in our environment. Chlorinated pesticide residues are present in the air, soil, and water, with a concomitant presence in humans. Organophosphate and carbamate pesticides—the compounds comprising the bulk of current pesticide use—are carried around the globe on air currents. Municipalities, schools, churches, business offices, apartment buildings, grocery stores, and homeowners use pesticides on a regular basis. Pesticides are neurotoxins that can cause acute symptoms as well as chronic effects from repeated low-dose exposure. These compounds can also adversely affect the immune system, causing cell-mediated immune deficiency, allergy, and autoimmune states. Certain cancers are also associated with pesticide exposure. Multiple endocrine effects, which can alter reproduction and stress-handling capacity, can also be found. Limited testing is available to assess the toxic overload of these compounds, including serum pesticide levels and immune system parameters. Treatment for acute or chronic effects of these toxins includes avoidance, supplementation, and possibly cleansing.
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Introduction

The objective of pesticide use to prevent crop loss from insects remains unachieved. K. Ausubel in his book, *Seeds of Change, The Living Treasure*, notes that since 1945 overall pesticide use has risen 3,300 percent, while overall crop loss due to insects has risen 20 percent in the same time period.[1] Ausubel reminds us about Martin Borlaug and the "Green Revolution," which introduced F1 hybrid seeds that provided exceptional crop yield when augmented by utilizing high nitrogen fertilizer. To protect plant growth, herbicides were needed to prevent weeds from competing for nutrients and space, as well as pesticides to prevent pest-induced crop damage.

The killing of primary pests with pesticides has paved the way for secondary pests to come to the fore. Where previously there were 10 primary pest insects – defined as causing greater than one million dollars of crop damage per year – there are now 300. Of the 25 most serious pests, 24 were previously secondary pests and 72 percent of these are now pesticide resistant.[1]

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Non-Occupational Pesticide Exposure

Of the 2.5 million tons of pesticides used worldwide each year, less than 0.1 percent reaches the target pest.[2,3] Thus, 99+ percent of currently applied pesticides are being released indiscriminately into the environment, many of which will persist for years and travel far from the point of application.

Chlorinated pesticides found in the soil can persist for decades. Dichlorodiphenyltrichloroethane (DDT- see Figure 1) has been shown to accumulate in soils where it was used agriculturally.[4] Soil-based DDT is incorporated into grasses growing in the soil, into cattle consuming the grass, and eventually into the milk and fat tissue of the cows.[5] While the half-life of DDT had been thought to range between 4-30 years, evidence from the Yakima River drainage in Washington State, and in other areas, points to a much more extended half-life.[6] The studies in this area show increased levels of p,p'-DDT in the soil and the persistence of ratios of p,p'-DDT: o,p'-DDT found in the parent compound applied 25 or more years earlier. This indicates that in certain soils DDT degradation is not occurring as rapidly as previously thought.

Such persistence has been found in other areas of the United States, such as soil in Texas and New Mexico.[7] When soil previously used agriculturally is excavated to accommodate housing sites, DDT finds its way into nearby streams and rivers via erosive runoff. The study of the Yakima River drainage found DDT in 100 percent of the fish sampled from that river. Other rivers, such as the South Platte, show a multitude of organochlorine pesticides in both sediment and fish.[8]

When houses are built on previously contaminated land, pesticides can easily be brought from the soil (from residents merely being "outside," from working in the garden, etc.) into the house, where they contaminate the home as house dust, as previously shown to do.[9] Pesticide exposure via house dust has been shown to cause higher serum levels of pesticides than what is incurred by eating contaminated foods.[10] There is also the possibility of pesticide contamination of vegetables grown in the home garden.

Those compounds not trapped in soil, tree bark, sediment, animals, humans, or other stable material begin a wind-driven leapfrogging around the globe.[11] Volatile chemicals move more frequently whenever the ambient temperature is sufficient to volatilize them. Less volatile compounds, like DDT, stay in place longer before being volatilized again.

Residues of DDT and other halogenated compounds have been found in medicinal herbs picked in the forests of Poland and Germany, where no spraying of DDT in these areas has been reported.[12,13] In a Polish study, herbs from all regions of the country were found to contain pesticide residues. The authors concluded, "Pesticide contents in most of the herbal raw materials should be attributed to the global contamination of the environment."

When these compounds reach upper latitudes and colder temperatures, they precipitate from the air and tend to stay trapped in whatever material they settle in. This has led to high amounts being found in the mountains of Western Canada,[14] the Arctic Ocean,[16] and recently even Aleutian eagles' eggs.[16] It likely accounts for the high amounts of toxins found in breast milk of indigenous Inuit mothers subsisting on traditional diets.[17] Decades of precipitation of airborne chlorinated pesticides in the Arctic have resulted in fat accumulation of these residues throughout the food chain, ultimately being transferred to Inuit infants through breast milk.

Fortunately, 120 countries currently participating in the United Nations Environmental Programme are negotiating agreements for global action on the movement of pesticides and other chemical pollutants from one

country to another.[18] It is hoped such agreements will lead to lower levels of airborne traveling toxins. Unfortunately, this effort does not address current pesticide loads found in every part of the planet.

Pesticides of every category have been found in groundwater throughout the United States. The U.S. Geological Survey's analysis of groundwater found pesticides in the water from agricultural areas,[19] and large amounts of pesticides have also been found in urban waterways, primarily due to frequent overapplication by homeowners. The Mercer Slough, a protected wetlands area in Bellevue, Washington, has been found to contain 18 different types of pesticides.[20] Pesticide residues are also found in foods; the 12 most contaminated fruits and vegetables being strawberries, bell peppers, spinach, cherries (U.S.), peaches, cantaloupe (Mexican), celery, apples, apricots, green beans, grapes (Chilean), and cucumbers.

The choice exists, of course, to either find alternative, less-contaminated fruits and vegetables, or purchase organic varieties of these items; however, given existing global pollution levels, food labeled "organically raised" does not necessarily mean "pesticide-free." Foods grown without pesticides can absorb pesticides from the soil, or be exposed to them in the air while growing, or during packing and transportation. Some foods, such as squash and carrots, may actually concentrate chlorinated compounds from the soil while growing.

While adults can make an informed choice about what foods to eat, infants cannot. While there are numerous beneficial aspects of breastfeeding, numerous studies have shown persistent chlorinated pesticide residues in breast milk. This startling fact has been recorded in tests of breast milk around the globe, including South Africa,[21,22] Kenya,[23] Saudi Arabia,[24] Jordan,[25] Sweden,[26] Finland,[27] Turkey,[28] Poland,[29] Ukraine,[30] Germany,[31] Hong Kong,[32] Australia,[33] New Guinea,[34] Mexico,[35-37] Brazil,[38] Canada,[39,40] and Arkansas[41] and New York[42] in the United States. DDT residues (including DDT, DDE [Dichlorodiphenyldichloroethylene] and DDD [dichlorodiphenyldichloroethane]) have been found in 93-100 percent of the milk samples in these studies.

High levels of polychlorinated biphenyls (PCB) and hexachlorocyclohexanes (HCH) – also referred to as BHC – are also frequently found. The majority of these studies also found numerous other chlorinated compounds, including hexachlorobenzene (HCB), endrin, dieldrin, and various chlordanes.

Most contaminated breast milk samples have a combination of many such compounds. All studies had samples that exceeded the World Health Organization's acceptable daily intake of 0.005 mg/kg/day. Levels of these compounds in breast milk were shown to correlate directly with the level of such compounds in maternal adipose tissue. Furthermore, infant serum levels of pesticides were shown to correlate with maternal breast milk levels, and not with airborne exposure. Pesticide levels in the milk and adipose tissue of mothers decreased with each breast-fed child.

Maternal pesticide exposures from diet or airborne sources have been associated with maternal load. However, a study in Papua, New Guinea, was conducted in an area where there had been no DDT use, yet all lactating women had DDT in their breast milk. This was most likely due to the previously discussed movement of DDT on global air currents. Since pesticides can bioaccumulate over decades, and can be passed to the next generation through both cord blood and breast milk,[23] the implication is that each succeeding generation begins life with a pesticide load it took their parents decades to develop.

Children can also be exposed to organophosphate pesticides (OP), such as chlorpyrifos, from home use of this compound. It was demonstrated that after a single broad-

cast in apartment rooms of this pesticide by certified applicators (not the typical homeowner), chlorpyrifos continued to accumulate on children's toys and hard surfaces for two weeks after spraying.[43] Based on this and similar studies it was estimated that after indoor spraying, children were exposed to levels from 21-119 times the current reference dose of 3 µg/kg/day.[44]

From these and others studies that found birth defects associated with chlorpyrifos,[45] the U.S. Environmental Protection Agency recently banned home use of this compound and imposed tighter restrictions on the pesticide's use on some agricultural products, specifically apples and grapes, and banned its use on tomatoes. These restrictions are designed to eliminate the chemical's residues on foods often consumed by children. The pesticide may still be used on a variety of grains and other crops; however, retail sale will be stopped after December 31, 2001. Despite overwhelming scientific evidence, no recall of existing stock was issued.

Pesticide Presence in Human Adipose Samples

Studies examining general populations for chlorinated pesticide residues have found the presence of multiple chlorinated hydrocarbons (DDT, DDE, DDD, aldrin, dieldrin, heptachlor, heptachlor epoxide, and PCBs) in the adipose tissue of residents of Greenland and Denmark.[46] Adipose samples from women in Germany, The Netherlands, Northern Ireland, Spain, and Switzerland, as part of the European Study on Antioxidants, Myocardial Infarction and Cancer of the Breast, showed the consistent presence of DDE.[47]

An adipose study of 40 autopsied trauma victims in Israel revealed DDE in all 40, HCB in 34, beta hexachlorocyclohexane in 27, DDT in seven, and gamma HCH in two. Three or more chlorinated residues were found in 80 percent of all studied.[48]

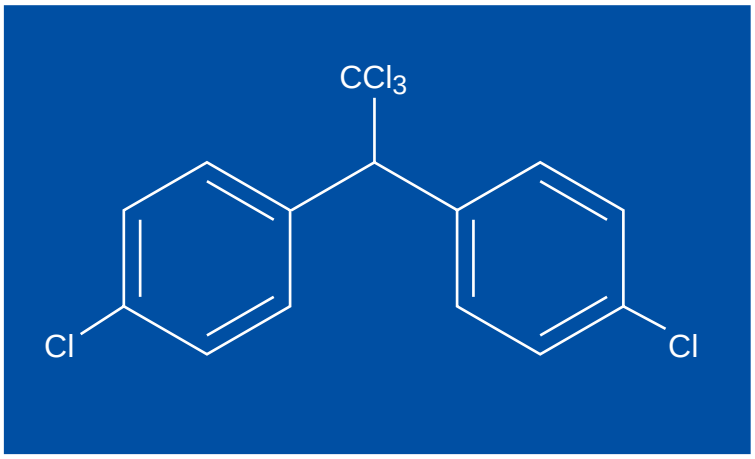
Samples of adipose tissue and other fluids taken from 17 caesarean section deliveries in Germany and Tanzania revealed chlorinated residues in all women. Those from Germany had higher levels of HCB and PCBs, while those from Tanzania had higher levels of DDT and DDE.[49] This study found maternal adipose tissue contained a 10 to 100-fold increase in accumulation of chlorinated hydrocarbons compared to other tissues and fluids tested. The concentration of certain toxins was higher in fetal cord blood and the placenta than in the maternal serum. These persistent chlorinated pesticide residues have also been found in adipose tissue throughout North and South America.[50-54]

The above-mentioned studies all investigated biologically persistent chlorinated hydrocarbons. Such tests for determining the presence of the non-biologically-persistent organophosphate, carbamate, and pyrethroid pesticides are not available, although the metabolite of chlorpyrifos, one of the most common organophosphates, was found in the urine of 82 percent of U.S. adults.[55] However, these studies provide a clear indication that more than the toxic effect of a single pesticide must be considered. Based on the above-mentioned studies, the average person, wherever they live in the world, most likely has more than one chlorinated hydrocarbon residue in their adipose and serum components. To this load can be added any of the organophosphate, carbamate, pyrethroids, or arsenical pesticides that may be in the air, food, or water, as well as solvents, heavy metals, polycyclic aromatic hydrocarbons (from combustion of fossil fuels, wood, cigarettes), terpenes, molds, etc.

Neurotoxicity of Pesticides

Pesticides kill insects by disrupting the nervous system. The primary action of chlorinated pesticides – which includes endrin, aldrin, toxaphene, benzenehexachloride (BHC), HCH, DDT, heptachlor, heptachlor epoxide, chlordane, trans-nonachlor, polychlorinates,

Figure 1: Dichlorophenyltrichloroethane (DDT)



dicofol, chlorobenzilate, mirex, HCB, methoxychlor and ethylan – is interfering with axonal transmission by disrupting ion flux, leading to over-stimulation of the nerves and uncontrolled neuronal discharge. Symptoms of acute human toxicity include headache, nausea, vomiting, hyperesthesias, irritability, confusion, convulsions, respiratory depression, cardiac arrhythmia, aplastic anemia, and porphyria cutanea tarda.

DDT, the best known of this class, was synthesized in 1874. Its pesticide activity was found in 1939, and it was used extensively by the U.S. military in World War II to control typhus, malaria, and lice. It was put into use in U.S. agriculture in 1945, and subsequently banned in 1974 after the uproar caused by Rachel Carson's publication of *Silent Spring*. In certain individuals DDT has been shown to cause changes in electromyographic potential and symptoms of fatigue, poor cognition, withdrawal from reality, blurred vision, headache, and ataxia.[56,57]

Levels of gamma HCH and dieldrin have been found to be higher in the brain tissue of persons with Parkinson's disease than in controls.[58] Elevated levels of DDE were also found in the substantia nigra in these Parkinson's patients. Lindane (gamma HCH) blocks the chloride ion channel by antagonizing GABA stimulation of chloride ion uptake through benzodiazepine receptors. Lindane can dramatically reduce the time needed for establishing CNS kindling, lower the convulsive threshold, and prolong the sensitivity to convulsive stimuli even after its clearance from the blood.[59] Having such potential for increasing seizure activity, it is surprising Lindane is allowed in shampoo treatments for head lice in children and adults.

The OP pesticides were first synthesized in 1820. These compounds were developed by Germany as nerve gases for military use during World War II. Their present-day nerve gas relatives include Sarin, recently used in a Japanese terrorist attack in the Tokyo subway system.[60] They were first used as pesticides in 1941-1944.

OP pesticides are rapidly absorbed following inhalation or ingestion. Dermal absorption is slower but prolonged exposure can result in severe poisoning. Once absorbed, OP compounds accumulate in fat, liver, kidneys, and salivary glands.[61] Instead of affecting axonal transmission, as chlorinated hydrocarbons do, they are acetylcholinesterase (AChE) inhibitors via phosphorylation. This leads to accumulation of acetylcholine, which binds to and stimulates muscarinic receptors (found in autonomic ganglia, CNS, heart, salivary glands, and smooth muscles) and nicotinic receptors (autonomic ganglia, skeletal muscle, and CNS). The brain initially over-stimulates; later there is paralysis of neural transmission. Antibodies to the cytochrome P450 hepatic detoxification system are also generated.

OP toxicity is heightened by the presence of the solvents toluene and xylene, which are found in some commercial OP products as "inert ingredients." Synergistic toxicity is also found with OP compounds and polycyclic aromatic hydrocarbons from auto exhaust.[62] The combination of neurotoxicity of solvents and

organophosphates can cause axonal and myelin degeneration in distal fibers. Their neurotoxic effect may be more pronounced in older individuals, partly due to the normal decline of AChE with aging.[63] Neonatal exposure to both OP and chlorinated pesticides appears to potentiate the neurotoxic effects if re-exposure occurs later in life.[64]

Symptoms of acute organophosphate poisoning include (1) nausea, vomiting, abdominal cramps, and diarrhea; (2) salivation and rhinorrhea; (3) headache and vertigo; (4) fixed pinpoint pupils, blurred vision, and ocular pain; (5) muscle twitches (face, tongue, and neck); (6) difficulty breathing (from excess secretions); and (7) respiratory paralysis and death.

The acronym "SLUDGE" – for salivation, lacrimation, urination, defecation, gastrointestinal disturbances, and emesis – is often used for the toxic picture of these compounds. If the antidote pralidoxime (2-PAM) is not given within 24-48 hours, the AChE-phosphate bond becomes so strong that physiologic recovery depends on new synthesis of AChE. AChE is restored to the affected area in about two weeks, but in the whole body it can take up to three months to recover. Serum testing for red blood cell AChE levels is often inconclusive except in the most serious poisonings. One study looked at both central nervous system effects via SPECT scans and red blood cell cholinesterase levels. Patients were differentiated into three groups, latent poisoning, mild and moderate poisoning, and severe poisoning. While all groups showed CNS damage on SPECT scans only the severe poisoning group had AChE levels below normal.[65]

Ishikawa has studied organophosphate poisoning in Japan, and found that in addition to being neurotoxic, OPs cause severe oxidative damage and stress, resulting in decreased selenium concentration in the brain and kidneys within 14-21 days of exposure.[66] He also found docosahexanoic acid (DHA) (animals 15 mg/kg/day, humans 5mg/kg/day), but not eicosapentaenoic acid (EPA), crossed the blood-brain barrier and prevented a rise in superoxide radicals secondary to OP exposure. The areas of greatest oxidative damage were the eyes (including optic nerve atrophy, neuroretina, and the destruction of retinal pigment), brain, and heart. Neurological changes unrelated to AChE inhibition include behavioral abnormalities, anxiety, irritability, confusion, depression, and fatigue.

Carbamates are also AChE-inhibiting pesticides, but they accomplish this by reversible carbamylation, not phosphorylation. SLUDGE symptoms can still be present, as carbamates primarily affect muscarinic and nicotinic receptors.

Chronic CNS symptoms have been frequently reported after repeated exposure to OP and carbamate compounds. Exposed greenhouse workers exhibited longer reaction times and reduced motor steadiness as well as increased tension, depression, and fatigue.[67] When matched with controls, workers in a factory making OP pesticides showed no difference in AChE levels but did show greater problems with memory, learning, and vigilance.[68] Sheep ranchers exposed to OP compounds in the course of sheep dipping exhibited poorer sustained attention and speed of information processing than non-exposed controls. The ranchers also showed greater vulnerability to psychiatric disorders than controls.[69]

When compared with controls, workers applying termiticide demonstrated poorer performance on pegboard turning tests and postural sway tests. They also displayed significantly more symptoms of memory disturbances, altered emotional states, fatigue, and loss of muscle strength.[70] Such neurotoxic effects can be found years after a single OP poisoning episode. Thirty-six such individuals were re-evaluated two years after a single episode of unintentional OP intoxication. On re-examination the poisoned group did worse than the control group on all neuropsychological subtests, as well as other tests for verbal

and visual attention, visual memory, visuomotor speed, sequencing, problem solving, motor steadiness, and dexterity.[71]

Animal studies have shown that in-utero exposure to OP compounds result in impairment on maze performance, locomotion, and balance in neonates.[72]

Peripheral neuropathy is also a common sequelae of OP exposure. South African farm workers exposed to OP pesticides who reported significantly more problems with dizziness, sleepiness, and headache were also found to have reduced vibratory sense and increase in hand tremor.[73] Flower bulb farmers were noted to have decreased conduction velocity of fast and slow motor fibers of the median and peroneal nerves as well as sensory fibers of the median and sural nerves.[74]

Ecuadorian pesticide applicators exhibited a significantly greater incidence of poor coordination, abnormal deep tendon reflexes, and reduced strength than non-exposed local controls.[75] Of 217 chlorpyrifos poisoning incidents reported by DowElanco, 21 cases had some evidence of peripheral neuropathy.

Symptoms of delayed neuropathy typically show up several days to four weeks after acute organophosphate exposure. Early signs are paresthesias, weakness and ataxia, gait changes, or flaccid paralysis. Some of the chronic neurotoxic effects of OP pesticides may be due to their ability to induce the formation of antibodies to neuronal tissues. Antibodies to myelin basic protein, neurofilament triplet protein, and glial fibrillary acidic proteins have all been exhibited after OP exposure.[76] Other antibodies to smooth muscle, parietal cells, brush borders, and thyroid have been demonstrated, as well as antinuclear antibodies.[77]

Immunotoxicity of Pesticides

The patient with chronic environmental pesticide overload generally presents to the clinician with either the above-mentioned neurotoxic manifestations or those of immunotoxicity. Different pesticides can cause varying effects on the immune system of any given individual. However, in viewing the overall effect, toxin exposure causes a specific immunologic imbalance unseen by other causative agents, including a general decrease in cell-mediated immunity (CMI) and an increase in humoral immune response.[79-82] The reduction in CMI can include reductions or elevations of T cell counts, including natural killer cells, depending on the specific compound.[83] In general, chemotactic and phagocytic responses are significantly reduced. Many chlorinated compounds also cause reduction of thymic weight and function. Natural killer cell activity is universally reduced.

These changes result in the clinical picture of decreased resistance, and an increase in allergies and certain cancers. The elevation of humoral immunity often results in production of antibodies to various tissues, as previously mentioned,[77] and is also seen in exposure to chlorinated pesticides.[84] The immunotoxic effects can be modulated by several factors, including level of exposure, nutritional status (low protein), concurrent pathologic conditions, biotransformation and activity of metabolites, physical and emotional stress, and oxidative stress.[85] Both OP and chlorinated pesticide exposure have been associated with chronic fatigue syndrome.[86,87]

One of the most published and contested arenas of pesticide-induced immunotoxicity is the area of oncology. Some studies looking only at DDT exposure and serum levels have failed to show any significant increase in cancer mortality or long-term health effects.[88,89] However, when the large picture of pesticide use is viewed, a positive correlation with cancers is noted.[90] Pesticide exposure causes DNA damage and the formation of DNA adducts, which can ultimately lead to cancer formation.[91]

OP pesticide use has been associated with aplastic anemia and leukemia in exposed farmers,[92,93] and in children exposed from

having their homes treated.[94] The studies involving U.S. farmers and their exposed children revealed a positive association with both OP and pyrethroid pesticide exposures and these hematologic disorders. Increased rates of multiple myeloma have also been associated with OP exposure.[95] Chlorinated pesticides are positively associated with the incidence of non-Hodgkin's lymphoma,[96-98] aplastic anemia,[99] cancers of the liver, colon/rectum, and lung,[100,101] multiple myeloma,[102] pancreatic cancers,[103,104] blood dyscrasias and leukemia,[105] and acute myeloid leukemia (along with solvent exposure).[106,107]

The issue of the association of chlorinated products with breast cancer has been the subject of numerous studies, and is beyond the scope of this article. Many studies have found a positive association between breast cancer and chlorinated pesticides[108-116] and many have not.[117-120] In the positive studies, associations have been made between breast cancer and adipose levels of DDT, DDE, PCB, dieldrin, and HCH.

While this question seems far from being settled it appears obvious that pesticide load in some women may be a factor in the development of breast cancer. A recent study showed that women with a genetic polymorphic variant of cytochrome P450-1B1 – which catalyzes the formation of 4-hydroxyestradiol that retains significant estrogenic activity and whose metabolites can generate potentially mutagenic free radicals that may damage DNA – have a greatly elevated risk of breast cancer.[121] It was noted that polycyclic aromatic hydrocarbons and chlorinated pesticides are all known inducers of CP450. Such an induction, associated with genetic polymorphism, might partly explain why pesticides are an apparent risk factor in some women but not others.

Endocrine Toxicity from Pesticides

After symptoms appear in the immunological and neurological realms, problems in endocrine function may also occur. Such hormonal imbalances are rarely the first to be noted when taking a chronological medical history. Chlorinated products are known to act as weak estrogens with potential for reproductive disruption[122] and to act as androgen antagonists.[123] These compounds have been associated with female infertility,[124] miscarriages,[125] and possibly male infertility.[126] OP pesticides have also been associated with male infertility, with increased LH production (possibly secondary to testicular damage),[127] and reduced numbers of morphologically normal and live spermatozoa.[128]

In addition to possibly affecting reproduction, pesticides can cause other endocrine problems. HCH, but not DDT, has been shown to modify pineal synthesis of melatonin.[129] DDE, the metabolite of DDT, can accumulate in the zona fasciculata in the adrenals[130] and lead to adrenal atrophy.[131] In animal models vacuolization and necrosis in the zona fasciculata secondary to DDE exposure appears most profound in fetal and neonatal animals, and less so in adults. The DDT metabolite also appears to be a tissue-specific toxicant to the zona fasciculata.[132] Reviews on these and other endocrine effects from environmental chemicals can be found in the literature.[133,134]

Other published health effects from pesticides include renal tubular toxicity from an OP compound accompanied with elevated hydrogen peroxide production and increased lipid peroxidation.[135] This again shows the extensive oxidative damage that OP compounds can cause. OP compounds have also been linked to reduced bone formation.[136] Agricultural workers exposed to OP compounds had significantly decreased bone formation than healthy controls.

Table 1: Chlorinated Pesticides in Serum and Adipose Samples (parts per billion). Courtesy of Accu Chem Laboratories.

Compound	Serum	Adipose
HCB	<0.3	135
Endrin	<0.3	168
Beta-BHC	<0.3	1657
Gamma-BHC	<0.3	121
Heptachlor	<0.3	63
Hep-Epoxide	<0.3	33
Oxychlordane	0.4	72
Trans-nonachlor	0.3	123
Dieldrin	<0.3	36
DDE	14.2	284
DDT	<0.3	222

Clinical Assessment

It should be recognized that all individuals are exposed to pesticides and that all carry some level of these toxins in their serum and adipose tissue. In addition, numerous variations in genetics, diet, lifestyle, and environment can interplay to either facilitate or conspire against clearance of these compounds from the body. Once a chronological medical history is obtained, the classic pattern of neurotoxicity and immunotoxicity, possibly followed by endocrine toxicity, may be seen. Once seen or suspected, testing may be warranted.

Because chlorinated compounds are fat soluble and bioaccumulative they can be easily measured in serum. This can be done either fasting or non-fasting, although non-fasting samples tend to show higher levels.[137] Most authors recommend the lipid content of the blood be looked at simultaneously so compounds can be rated as per gram of lipid. This provides the best correlation with adipose samples. However, serum and adipose samples can be vastly divergent.

Table 1 shows the variance between serum and adipose samples from one individual. If an adipose sample is to be taken, it is recommended that adipose tissue be taken from three different sites, as toxin distribution is uniform. Laboratories that specialize in testing these compounds do not give results in amount of toxin per gram of lipid as the literature suggests, but in ng/ml of blood. The laboratories also provide levels of their laboratory averages as a reference range. These are the averages of tests done by the specific laboratory, and do not necessarily represent "normal ranges" in the United States.

These laboratories also perform urine analysis for metabolites of organophosphates and carbamates. These compounds are generally cleared from the urinary tract within 14 days of exposure and do not show exposure prior to that time. As mentioned earlier, testing for red blood cell acetylcholinesterase levels is generally not definitive except in cases of serious poisoning.

Some laboratories also offer testing for autoantibodies that can be formed from exposure to pesticides and solvents. Testing of immune parameters, including lymphocyte subpopulations and natural killer cell activity, may also give an indication of immunotoxicity.

Treatment for Chronic Pesticide Exposure

The first step in treating any toxic individual is avoidance of further exposure. Recognizing that pesticide use is ubiquitous, this may not be easy. Avoidance can include consuming organic foods, avoiding living in or traveling through agricultural areas during spraying seasons, avoidance of public buildings after spraying has taken place, finding out

Pesticide Protection and Detoxification.

- **Avoid Further Exposure**
- **Nutritional Supplementation**
  - Adequate protein (whey preferably)
  - Decrease sugar intake
  - Vitamin A
  - Vitamin B1 (Thiamine)
  - Magnesium
  - Vitamin B6 (Pyridoxal 5'-phosphate)
  - Selenium
  - Antioxidants
  - Silybum marianum (milk thistle)
  - Vitamin C
  - DHA
- **Adjunctive Therapies**
  - Low-temperature sauna
  - Hydrotherapy
  - Colonic irrigation

when neighbors or governmental agencies are planning to spray an area, etc.

In addition, it should be determined whether their present dwelling had been termite-treated (chlordane has a half-life of 20 years) or had other pesticide treatments. Carpeting from previous owners when the dwelling was treated would contain pesticide residues, as would the dust in furnace ducts, both of which would need to be remediated. The fewer pesticides personally used, and the fewer foods or other consumer products purchased that rely on pesticide use will ultimately reduce the amount of pesticides released into the atmosphere.

The second treatment step is supplementation of the nutrients needed to help clear pesticides from the body, restore common pesticide-induced deficiencies, and prevent tissue damage from these compounds. Dietarily, adequate protein and reduced sugar intake ensures proper liver clearance of xenobiotics from the blood. Whey protein increases glutathione levels in addition to providing complete protein to the body, which enhances liver function, making it the first choice for such cases. Chlorinated pesticides and other chlorinated compounds reduce the available vitamin A and thiamine to normal tissues, the deficiency of which tends to increase the toxic effect of pesticides.

In addition to these nutrients, a high quality multiple vitamin/mineral supplement with extra magnesium, pyridoxine, selenium, antioxidants, and milk thistle is recommended. Vitamin C should be taken in maximal doses to help clear toxins from the blood and to provide high antioxidant activity. Docosahexanoic acid (DHA) is necessary for any OP exposure to increase antioxidant activity in the brain and prevent OP-induced damage.

If elevated levels of chlorinated pesticides are found in serum or tissue, additional measures may be necessary, such as the frequent use of low temperature saunas, hydrotherapy, and colonic irrigation.[138]

Summary

Pesticides are ubiquitous in the environment. Residues of chlorinated pesticides are present in the air, soil, and water, as well as in most humans. Organophosphate and carbamate pesticides—the compounds comprising the bulk of current pesticide use—are carried around the globe on air currents. These pesticides are used in schools, churches, business offices, apartment buildings, grocery stores, and homes on a regular basis. Pesticides are primarily neurotoxins, causing both acute symptoms as well as chronic effects from repeated low-dose exposures. These compounds adversely affect the immune system, primarily causing cell-mediated immune deficiency, allergy, and autoimmunity. Multiple

endocrine effects can alter reproduction and stress-handling capacity. Laboratory testing to determine if a toxic pesticide overload exists is limited to serum pesticide levels and immune system parameters. Treatment for pesticide toxicity includes avoidance, nutritional/botanical supplementation, and detoxification/cleansing.

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