



Cornell University Program on Breast Cancer and Environmental Risk Factors in New York State (BCERF)

Pesticides and Breast Cancer Risk, An Evaluation of DDT and DDE

The majority of the well-controlled studies on white North American or European women have not been able to show that higher blood or fat levels of the persistent insecticide DDT or its breakdown product DDE are associated with an increased risk of breast cancer. Whether race, ethnicity, or recent history of DDT exposure influence breast cancer risk are questions researchers need to answer more fully.

Why study DDT and the risk of breast cancer?

There has been great interest in whether certain organochlorine pesticides can affect the risk of breast cancer. Most organochlorine pesticides, including the insecticide DDT, have not been used in the US, Canada or Western Europe since the early 1970s. DDT and its breakdown product DDE, can stay in the environment for long periods of time. DDT and DDE can accumulate and are stored in fatty tissues of animals and humans. Levels are greater in species higher on the food chain, including fish and large marine mammals (whales and seals), certain birds (eagles, hawks and gulls), and in people.

One component of the DDT that was sprayed, called "o,p'-DDT" was identified as an environmental estrogen 30 years ago. Estrogens are hormones that are chemical messengers. Estrogens can signal breast cells to grow and divide. This increased rate of cell division can play a role in the development of breast cancer. Over half of all breast tumors also depend on estrogen for growth. Estrogenic forms of DDT can support the growth of estrogen-dependent breast tumors in rats. However,

there is little evidence that the breakdown product of DDT, called DDE, is estrogenic. DDE is resistant to being broken down, and stays in the environment and in human tissues for long periods of time.

Does DDT or DDE affect the risk of breast cancer?

While earlier studies suggested that white women with higher levels of DDE in their blood or fat were at higher risk for breast cancer, the majority of the more recently conducted, well-controlled studies of North American and European white women have not supported this association. Studies of women of color and women from developing countries have been less consistent.

Several small studies published in the late 1980s and early 1990s reported a higher level of DDE in the fat of women with breast cancer compared to women without breast cancer. Yet, the small scale of these studies and the lack of control of other factors that may have affected breast cancer risk made the significance of these studies questionable. These studies were followed by a larger, well-controlled study of New York City women published



in 1993. Researchers reported a four-fold higher risk of breast cancer in women with the highest levels of DDE in their blood compared to the women with the lowest levels.

However, the majority of the North American and Western European studies published since 1994 have not been able to show that white women with elevated blood or fat levels of DDE had a significantly higher risk of breast cancer. A few reported a slight to moderate reduction in breast cancer risk in the women with the highest DDE levels. The majority of these studies were well designed, and carefully conducted. Most considered other factors that may affect breast cancer risk (confounding factors, like lactation history), and followed careful laboratory procedures for analyzing the blood or fat samples.

The results of studies conducted in developing countries that continue to use DDT are less consistent. Studies of women from Mexico City, Brazil and North Vietnam did not report a higher risk of breast cancer in women with higher blood or fat levels of DDE. However, a recent study conducted in Mexico City, and a study conducted in Colombia, South America, did find a moderately higher risk of breast cancer in women with higher blood levels of DDE. A study of breast cancer risk and blood DDE levels in white and African American women was recently conducted in North Carolina. Researchers did not find a higher breast cancer risk in white women who had high levels of DDE in their blood. But breast cancer risk tended to be higher in a sub-group of African American women who were thin (a body mass index of less than 25) and had higher blood DDE levels. There was no relationship between body size, blood DDE levels and breast cancer risk in white women. More studies are needed to determine if particular ethnic or racial sub-groups of women exposed to DDT have a higher risk of breast cancer.

Does DDT cause other types of cancer?

In 1991, the International Agency for Research on Cancer classified DDT as a *possible human carcinogen*, based on the strong evidence of cancer in laboratory

animals and the limited evidence of higher cancer risk in humans exposed to DDT. The US Environmental Protection Agency (EPA) classified DDT as a Class B2 *probable human carcinogen*.

DDT can cause cancer in laboratory animals including rats, mice or hamsters. Tumors have been found in the liver, adrenal glands, lung, or lymphatic tissues of laboratory animals fed or injected with DDT. In contrast, there is little evidence that DDT can cause cancer in monkeys or dogs.

There is some evidence of a higher risk of lung or pancreatic cancer in men who worked in DDT manufacturing plants. A greater number of deaths from liver cancer and multiple myeloma (cancer of cells in the bone marrow) was seen in a study of male pesticide applicators who had sprayed DDT for mosquito control. A higher risk of non-Hodgkin's lymphoma, multiple myeloma, or leukemia has been reported in some, but not all studies of male farm workers exposed to DDT. However, when researchers took into account the exposure to other types of pesticides, there was no longer a higher risk of non-Hodgkin's lymphoma in farmers who used DDT on crops or livestock. In another study, researchers saw more liver cancer in the white men and women who had higher levels of DDE in their fat. But, unlike other studies, they did not observe higher levels of pancreatic cancer or multiple myeloma. Hence, while there is suggestive evidence of higher cancer risk in DDT-exposed populations, the strength of the evidence is weakened by the lack of consistent results. More studies are needed to evaluate cancer risk of those who were exposed to DDT through their occupations.

Do we know how DDT may cause other types of cancer?

Scientists are continuing to explore and identify the ways DDT may affect cancer risk. Here are some examples. DDT can help activate certain cancer genes, called oncogenes. However, DDT and DDE do not appear to be mutagens. Mutagens are substances that can cause small changes in our genetic code (DNA) that may lead to cancer. Clastogens are substances that can physically



break strands of DNA. These changes in DNA may increase the risk of developing a cancerous tumor. Most studies show that DDT and DDE are not clastogenic, though a few studies have shown some evidence of clastogenicity.

The immune system defends the body against harmful cancer-causing substances. Chemicals that suppress the immune system may increase susceptibility to cancer. There is evidence that DDT and DDE can suppress the immune system. However, researchers have not fully evaluated whether the suppression of the immune system leads to increased cancer risk. Researchers are interested in determining if leukemia or lymphomas observed in workers exposed to DDT may be related to a suppression of the immune system.

What are some additional avenues of research?

Researchers are interested in whether body levels of DDE and other organochlorine pesticides affect the characteristics of breast tumors, or breast cancer survival rates. Other researchers are looking at differences in genes that control the ability to detoxify chemicals in the body. Some of these differences in genes, called polymorphisms, may affect the ability of a person to make or break down estrogen, or handle chemicals from the environment.

What is the history of DDT's use?

DDT was developed as an insecticide against mosquitoes and lice during World War II to prevent diseases such as malaria and typhus. After the war, DDT was used against a variety of insect pests in forests, swamps and in agriculture. DDT was used extensively in the southeastern US on cotton, peanut and soybean crops. Usage in the US dropped dramatically by the late 1960s as harmful health effects of DDT exposure were identified in wildlife and humans. The EPA banned use of DDT in the US in 1972. However, American companies continued to produce DDT for export into the late 1980s. During the late 1980s and 1990s, use on agricultural crops was reduced or banned in India and many Latin and South American countries. DDT is still

used in many developing countries worldwide against mosquitoes to control malaria outbreaks. DDT can be carried in the atmosphere, and can be deposited in remote areas, especially in very cold northern regions. Large sea animals (whales and seals) and indigenous people living in the Arctic Circle have some of the highest body levels of DDT and DDE.

Is DDT or DDE still found in food?

Though DDT has not been used in the US for nearly 30 years, DDT and its breakdown products are still detected at low levels in a wide variety of foods eaten by Americans. Residues of DDT and DDE, the persistent breakdown product of DDT, are still found in the soil of fields and forests treated years ago, and in the sediment of lakes and rivers. Crops grown in contaminated soil can still come in contact with these residues. DDE concentrates up the food chain, and livestock and fish can accumulate DDE in their fat.

As a part of its monitoring program, the Food and Drug Administration (FDA) determines the levels of pesticide residues in a wide variety of foods typically consumed by Americans. DDE or DDT were detected in 22% of the 1,040 food items analyzed in the 1999 FDA Total Diet Study (see Table 6 in <http://vm.cfsan.fda.gov/~acrob/pdf/pes99rep.pdf>). Over the past ten years, these surveys have detected DDE in a variety of foods. This includes meat, fish and shell fish products, eggs, root vegetables, legumes (beans, peas, peanuts), some fruits, and leafy greens. (See <http://vm.cfsan.fda.gov/~acrob/TDS1byps.pdf> for a complete list of foods and levels of DDT and its breakdown products. Also see BCERF Fact Sheet #25 on *Pesticide Residue Monitoring and Food Safety*.)

Are there limits put on the levels of DDT and DDE allowed in food?

The FDA and the US Department of Agriculture (USDA) monitor the levels of DDT and DDE in domestically produced and imported food, and in animal feed. Because of the past widespread use of DDT, the FDA set limits on the “unavoidable” residue levels of DDE and DDT



in food (called “Action Levels”). These levels do not represent permissible levels, but when these Action Levels are exceeded the FDA takes action to remove the food item and make it unavailable to the consumer. The complete list of Action Levels for DDT and its breakdown products is available at <http://vm.cfsan.fda.gov/~lrd/fdaact.html> and single copies can be obtained by calling the FDA at (202) 205-5251.

Is DDE or DDT found in drinking water or in sediments?

Both DDE and DDT do not dissolve easily in water. While they are frequently found in the sediments at the bottom of contaminated rivers and lakes, the levels in drinking water have seldom been a concern. When detected in water supplies, levels have usually been very low. One of the surveys that has detected DDE in well water was conducted in the Midwest by the US Geological Survey. About 6% of the wells tested had positive detections of DDE. A statewide survey of DDE levels in surface water of New York State also found 6% of the samples with detectable levels of DDE.

However, bottom-feeding fish, which eat plants and other aquatic life living in the sediment, can accumulate DDT or DDE in their bodies. Nationally, levels of DDT and DDE have been declining in fish in the US since the mid-1970s, when agricultural uses of DDT were banned. DDT and DDE have been detected in the sediment of the Hudson River Basin of New York State by the US Geological Survey. The New York State Department of Health and the EPA issue fish advisories. These advisories can help individuals limit their exposure to sport fish caught from contaminated bodies of water.

Is DDT or DDE found in breast milk?

Human breast milk is the preferred sole source of nutrients during early infancy. Breast feeding can help strengthen the infant immune system, and promotes mother-child bonding. However, breast milk can be a source of exposure to organochlorine pesticides in the breast fed infant. Organochlorine pesticides such as DDT and its breakdown products accumulate and are stored over time in a woman’s body fat, and can appear in human breast milk. The average levels of DDE or

DDT in US breast milk ranged from 0.2 to 4.3 ppm (parts per million) prior to 1986. The levels of DDE in human breast milk in developed countries like the US appears to be decreasing over time. A more recent study conducted in Arkansas reported average levels of DDT in breast milk to be 0.04 to 1.0 ppm. However, there are some groups of women who have higher levels of DDE in their breast milk. This includes women who eat large amounts of contaminated sports fish from the Great Lakes in the US, women from the former Soviet Republic of Kazakhstan, and Native American Inuit women who rely on fish as a major food source. Levels of DDT and DDE in breast milk can also be high in developing countries where DDT is still used in agriculture or for mosquito control.

Does exposure to DDE in breast milk affect breast development or growth?

A study of children in North Carolina looked at whether their exposure to DDE in breast milk during infancy affected the age at which their breasts developed during puberty. No significant effect of DDE was found. There was also no effect on the age-adjusted height of daughters or sons exposed as infants to higher levels of DDE in breast milk. However, other studies have found that while the health of the children did not appear to be affected, mothers with higher levels of DDE in their breast milk breast fed their infants for shorter periods of time. It is possible that DDE can affect a mother’s ability to lactate.

What are current efforts to help reduce exposure to DDT?

On August 24, 2000, the EPA released a National Action Plan to address the risks of “persistent bioaccumulative toxins” (called PBTs) including DDT and other persistent pesticides. (see <http://www.epa.gov/pbt/pestaaction.pdf>) Goals of the plan included: 1) facilitate local government efforts to collect and properly dispose of any DDT that is still being stored, 2) facilitate efforts to remove DDT from contaminated sites, 3) facilitate reduced exposures through education and outreach efforts, 4) eliminate risk from long-range transport by working internationally to phase out production and use in other countries, and to encourage



proper disposal and destruction of stockpiles, and 5) continue to monitor residues in fish, wildlife and humans to help measure progress toward reduced exposure.

The passing of the international “POP treaty” in November of 2000 was a major landmark. This is the first global agreement that will ban or phase out most uses of twelve persistent organochlorine pesticides (POPs), including DDT. DDT will still be allowed in developing countries for use against mosquitoes to control malaria.

How can I minimize my exposure to DDT and DDE?

- If you still have containers of DDT stored in your home, garage or barn, ask your regional environmental authorities how to dispose of these products properly.
- Check with your local fish and game or health authorities before eating sport fish caught from local streams and rivers. (In New York call 1-800-458-1158 extension 27815, or call EPA at 1-513-489-8190, for information on fish advisories.)
- Wash all fresh produce thoroughly in water, especially leafy greens, beans, root vegetables, and fruits and berries, to remove soil and any residues of DDT or DDE on the surface of the food.
- Choose lean cuts of meat, poultry and fish. Trim excess fat from meat and poultry, since DDT and DDE concentrate in animal and fish fat.

Where is more research needed?

- Studies are needed to develop more precise and inexpensive methods to estimate past exposures to DDT and DDE.
- Studies are needed in developing countries that continue to use DDT to determine if recent use of DDT affects breast cancer risk in exposed women.

- Studies are needed to determine the breast cancer risk in groups of women that have relied on sports fish as a major food source. This includes indigenous people living in North America and Greenland that have had a historically high consumption of fish potentially contaminated with DDT and DDE.
- Studies are needed to determine the breast cancer risk of persons of color with high body levels of DDT or DDE.
- Studies are needed to see whether the higher rates of leukemia or lymphomas observed in DDT-exposed agricultural workers may be related to a suppression of the immune system.
- Studies are needed to see if exposure to DDT or DDE affects how long a woman is able to breast feed, or the age when she starts to menstruate or enters menopause.

What research is underway?

There are numerous studies around the world evaluating how organochlorine pesticides, including DDT, may affect the risk of breast cancer and other cancers. Here are a few examples:

- Mt. Sinai School of Medicine researchers are evaluating the estrogenic (hormonal) activity of persistent pesticides found in New York City harbor sediments.
- University of Wisconsin researchers are exploring whether regional differences in breast cancer rates are due to differences in environmental exposures, including DDT.
- University of California at Berkeley researchers are investigating whether there are any disturbances in the monthly menstrual cycles of Laotian women who have eaten DDT-contaminated shell fish from San Francisco Bay.



An extensive bibliography on DDT, DDE and Breast Cancer Risk is available on the BCERF web site at <http://www.cfe.cornell.edu/bcerf/>

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