

## **No Spray Research Paper:**

# **How do Pesticides Affect People**

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Dear No Spray Coalition,

I am a retired insecticide toxicologist and a several year reader of your efforts regarding ending spraying of insecticides for control of West Nile Virus. I support your efforts.

For several years now, I have been doing literature reviews on why it is that exposures to pesticides cause human health problems. My conclusion is that organophosphate and carbamate insecticides cause problems because they poison the esterase(s) that activate Vitamin A, retinol, from retinyl ester precursors. Retinol yields vitamin A hormone and the presence of this hormone is required for synthesis of immune system proteins. The result is that those with normally less active immune systems and those exposed to insecticides are more sensitive to WNV than the rest of the population.

Exposures to pyrethroids may produce similar effects since it is likely that pyrethroids compete with retinyl esters for the enzymatic sites that metabolize them.

For your information, below is a brief statement outlining some of my ideas in regard to your interests. What to do? Blocking breeding with *B. israelensis* (larvicide) and/or use of *Gambusia* seem the most effective approaches.

Good luck with your efforts.

## **ENVIRONMENTAL CHEMICALS AND ENVIRONMENTAL ILLNESS: A MODEL THAT MAY EXPLAIN THE RELATIONSHIP**

Exposure to a wide range of environmental chemicals (ECs) is known to cause environmental illness (EI) in both humans and wildlife. ECs include pesticides such as insecticides and herbicides, and non-pesticides such as PCBs and dioxin. EIs include myalgia, chronic fatigue syndrome, multiple chemical sensitivity, and Gulf War Illness. Observed effects of exposure to ECs include interference with the ability of the immune system to protect our bodies against viruses, reproductive problems, increased cancer rates, and decreased ability to make enzymes to protect against naturally occurring dietary poisons.

Deficiencies in retinoic acid, the hormone derived from vitamin A, characterize most human autoimmune diseases. In addition to the conditions listed above, rheumatoid arthritis, type 1 (juvenile) diabetes, multiple sclerosis, systemic lupus, and asthma are well known autoimmune diseases that are related to vitamin A hormone deficiencies. Exposure to ECs may trigger any of these diseases. Certainly, their occurrence has increased dramatically in the age of ECs. Laboratory test animals exposed to ECs show the same declines in retinol and retinoic acid as occur in humans with autoimmune diseases. This suggests all these ailments are (a) autoimmune diseases (b) may be triggered by exposure to ECs and (c) are characterized by a deficiency in the critically important hormone retinoic acid.

Retinoic acid must be present in vertebrate cells for protein synthesis to occur. Where levels are low, the ability to make antibodies and other immune system products is decreased, ability to repress synthesis of potential cancers is lost, reproduction is blocked, and ability to make enzymes to protect organisms from dietary poisons is decreased.

Most autoimmune diseases occur more often in females than in males. The major reason for this is the greater need for vitamin A in activating the reproductive cycles in females as compared to males. This is why lack of vitamin A may interfere with reproduction in males and why females are more sensitive to autoimmune diseases resulting from vitamin A deficiency.

Suspect ECs have one or both of two characteristics in common. Many of them mimic the structure of thyroid hormones and react with transthyretin, the protein that transports both thyroid hormones and vitamin A from storage sites to sites where they are needed. Examples are dioxin, PCBs, DDT and other chlorinated insecticides and the newer synthetic pyrethroids, all of which contain a phenoxybenzyl chemical structure similar to that of dioxin. Other ECs, particularly organophosphate, carbamate, and pyrethroid insecticides, are probable poisons of the enzymes that activate retinoic acid from its fat-soluble precursors, retinyl acetate and retinyl palmitate.

Exposure to these chemicals, whether used for pest control in either agricultural or urban situations, or as clothing treatments to protect against insect bites and disease transmission, may lead to the health problems described above.

It is possible to determine if ECs cause these problems, and if so, which ECs are most dangerous. One way to do this is to measure poisoning of transthyretin in animals fed or otherwise exposed to ECs. A second way is to measure concentrations of vitamin A (retinol) and vitamin A hormone (retinoic acid) in the liver and urine of animals fed or exposed to ECs. Lastly, poisoning of the enzymes that convert retinol to retinoic acid can also be measured.

Some humans are more sensitive to ECs than others and therefore, more likely to have EIs. Sensitive individuals may be those with lower than usual levels of retinol and retinoic acid. This may show up as high sensitivity to sunlight and also, night blindness. Low levels may also occur in individuals on diets deficient in yellow vegetables and other sources of retinoic acid precursors. Lastly and most importantly, exposure to ECs represents a potential health risk for all of us.

The concepts described above can tell us why ECs are dangerous and which ECs are most dangerous. Restrictions on the use of the most dangerous chemicals should decrease the occurrence of EIs.