APPENDICES

The Ruling

DEPARTMENT OF NATURAL RESOURCES
STATE OF WISCONSIN

Petition of
CITIZENS NATURAL RESOURCES ASSOCIATION, INC.,
WISCONSIN DIVISION, IZAAK WALTON LEAGUE
OF AMERICA, INC.

For a declaratory ruling on the use of dichloro-diphenyl-trichloro-ethane, commonly known as DDT, in the state of Wisconsin

Docket 3-DR

EXAMINER'S SUMMARY OF EVIDENCE AND PROPOSED RULING

On October 28, 1968, the Citizens Natural Resources Association, Inc. by Frederick L. Ott, Wauwatosa, Wisconsin, and on November 1, 1968, the Izaak Walton League of America, Inc., Wisconsin Division, by J. Michael Borden, Elm Grove, Wisconsin, filed petitions with the Department of Natural Resources requesting a declaratory ruling in respect to the use of Dichloro-Diphenyl-Trichloro-Ethane, commonly known as DDT, in the State of Wisconsin.

The Department issued its Notice of Hearing on November 5, 1968 and held hearing on the matter December 2, 1968 and on days thereafter at Madison, Wisconsin, before Examiner Maurice H. Van Susteren.*

*The list of Appearances which precedes the Examiner's Summary of Evidence has been deleted. (Eds.)

Examiner's Summary of Evidence

I. Statutes, Rules, Issues.

A. The petitioners seek a declaratory ruling under Section 227.06, Wisconsin Statutes, which provides that any interested person may petition for a declaratory ruling with respect to the applicability to any person, property or state of facts of any rule or statute enforced by it. A ruling is sought declaring DDT to be a highly toxic persistent chemical, that its use be restricted in such way that it cannot enter the biosphere and that its existence in the biosphere constitutes pollution.

> **Section 144.01 (11), Wis. Stats.,** defines pollution:
> Pollution includes contaminating or rendering unclean or impure the waters of the state, or making the same injurious to public health, harmful for commercial or recreational use, or deleterious to fish, bird, animal or plant life.
> **Section 144.30 (9), Wis. Stats.,** defines environmental pollution:
> Environmental pollution means the contaminating or rendering unclean or impure the air, land or waters of the state, or making the same injurious to public health, harmful for commercial or recreational use, or deleterious to fish, bird, animal or plant life.

B. Rules

Under the provisions of Section 144.025 (2) (b), the Department is authorized to adopt rules setting standards of water quality, to protect the public interest which includes the protection of the public health and welfare and the use of the waters for public and private water supplies, the propagation of fish and aquatic life and wildlife and other uses.

In compliance with the above, the Department adopted minimum standards of water quality in Wisconsin Administrative Code section RD 2.02 (1) (d):

> Substances in concentrations or combinations which are toxic or harmful to humans shall not be present in amounts found to be of public health significance, nor shall substances be present in amounts, which by bio-assay and other appropriate tests, indicate acute or chronic levels harmful to animal, plant or aquatic life.

C. Issues.

1. DDT in what concentrations or combinations is toxic or harmful to humans and its presence in water in what amounts can be found to be of public health significance?
2. What amounts of DDT in water, which by bio-assay and other appropriate tests, indicates acute or chronic levels harmful to animals, plant or aquatic life?
3. Is DDT a pollutant within the statutory definitions of "pollution" as found in Sections 144.01 (11) and 144.30 (9) of the Wisconsin Statutes?

II. DDT—Chemical Structure

A. Chemical Structure

DDT, a chlorinated hydrocarbon, is chemically described as 1, 1, 1-trichloro-2, 2-*bis* (para-chlorophenyl) ethane.* It is common practice to refer to DDT and to include in the term its various isomers—DDE and DDD, and lesser isomers DDA and DDMU. Each of the isomers is further broken down into the ortho, para derivative and the para, para derivative. Isomers have the same number and kind of atoms but in either different configuration or different locations within the molecule. What is known as technical DDT is composed of 75% of para, para DDT, 20% ortho, para prime DDT, and 5% is the other isomers. DDT is manufactured in the reaction of chloral and monochlorobenzene in the presence of sulfuric acid, the spent sulfuric acid carrying away any monochlorobenzene that remains.

DDT has been known since 1870, developed in Switzerland as an insecticide and first used in United States armed forces in 1943. DDT in its pure state is not insecticidal.

B. Properties, Uses, and Tests

1. Physical Properties

DDT is unique in that it has broad biological activity as an insecticide, it has great chemical stability, high mobility, is relatively insoluble in water and soluble in fat or lipid tissue. It has a strong tendency to form suspensions more than solutions. Ability to absorb particulate matter can cause a concentration in water particulates which is thousands of times greater than the concentration in water itself. Although it has a finite vapor pressure, and is relatively non-volatile, it will evaporate and recrystallize in air and travel in airborne dust. It also has the power of co-distillation. Half-life is estimated at ten years, there being no standards of determination.

However stable DDT is, it does degrade in sunshine and oxygen, first to DDE and then to bis (chlorophenyl) ketone and parachlorobenzoic acid. Since the DDT molecule is a very efficient light absorber, it absorbs the shorter wave lengths of the solar spectrum. Being a diphenyl methane type compound, it readily dissociates to a free radical leading to the formation of DDE. The degrading mechanism of DDE is not clear, but it does degrade faster than DDT. Under sunlight irradiation in sealed Petrie dishes, DDT will degrade approximately 50% in 12 days and it can be expected to be faster in a vapor state in the atmosphere.

When applied to crops, it has a period of rapid decline believed to be evaporation, and then degrades at a rate that is logarithmic

*References to specific pages in the hearing transcript have been deleted throughout this document. (Eds.)

in time. Exact mechanism of "disappearance" is not known. Half-lives of DDT and isomers are reported in crop materials from as little as two days to 40 days—the longer time being the more appropriate. DDT on crops or plants degrades to DDD and DDE and when harvested, the residues go with the plant. DDT very tightly sorbs to soil particles which may be moved deeper into soil or washed away. "Disappearance" of DDT from soil is logarithmic in time and on the average 20% of the amount present at any time "disappears" each year. DDT "degradation" in soil is due to microorganisms amounting to as much as two-thirds of the DDT in a period of two weeks to DDE, DDD, DDA, DBM, DBP and chlorobenzoic acid. Degradation is more rapid under *anaerobic* conditions. DDT is extremely insoluble in water, sorbs to particulate matter which collects on bottoms of streams, etc. and tends to degrade under the anaerobic conditions existing. It will degrade in the gut of fish, insects, mammals and birds because of microorganisms found there.

2. Uses

   DDT is and has been primarily used as an agricultural pesticide, for the control of Dutch elm disease and for the control of mosquitoes. Slightly over 100,000 pounds were shipped into Wisconsin in 1968 with approximately 58,000 pounds used for Dutch elm disease. Recommendation for mosquito control was withdrawn (in Wisconsin), as was registration for use on dairy cattle and around dairy barns since a zero tolerance was established for milk in 1954 in the Miller amendment. It is also used as a rodenticide in the control of mice and bats.

3. Toxicity

   DDT has a wide range toxic effect on agricultural pests, on the entire phylum Arthropoda, and is toxic to a degree to fish, birds, mammals, amphibians and reptiles and crustaceans. It is a Class III pesticide under the Federal Economic Poisons Law. Technical DDT is somewhat more toxic than metabolites DDE and DDD and toxicity varies between species. Pure undiluted DDT is not an insecticide. It was stipulated that DDT has an effect on the nervous system. Effects occurring at very low concentrations are variable but in the case of cockroaches consist of repetitive firing in nerves, resulting in disorientation, running about, tremor, overwork, and death due to exhaustion.

   The effect on crustacean nerve is the failure of the nerve axon to transmit impulses. The effect on robins is similar to that of the cockroach—quivering, tremor, and death due to nerve effect. DDT, at acute toxic levels, affects the central nervous system of fish causing instability, difficulty in respiration, and sluggishness. In humans, the first clinical signs of toxicity are a sensation of burning or itching of the tongue, lips, and face, tingling of fingers and toes. With larger doses tremor appears, a sensation of alarm,

of fear, marked uneasiness, and convulsions with accidental doses.

In rats clinical signs of poisoning are directly correlated with the concentration of DDT in the nervous system, as measured by the concentration in the brain. All parts of the nervous system are affected. In both animals and man poisoned by DDT it is the nervous system that is affected primarily.

4. Translocation

DDT, because of its chemical/physical properties, once applied to crops or placed in the atmosphere, moves throughout the environment in water, air, soil, and food. A minor transport mechanism is in organisms such as birds and fish. It has been found in filtered air, untreated forest soil, and the fish in untreated watersheds had in some instances 2.4 ppm DDE. DDT and metabolites have been found in oceanic food chains from zooplankton to gulls, osprey, cormorants, petrels, pelicans and peregrine falcons with a corresponding biological concentration at each level of the chain. It was not present in plankton gathered in the 1920's and studied, nor in the body of a penguin of 1911. It was not present in ten samples of human fat collected before the advent of DDT. DDT is found in bodies of fresh water (Lakes Michigan/Superior) from the muds up through all aquatic organisms.

5. Storage/Accumulation in Organisms

While relatively insoluble in water, DDT is soluble in lipid or fatty tissue, and accumulates in such tissue.

Unlike birds, DDE levels in mammals appear to reach a plateau with the same amount of intake and remain at approximately 10-12 ppm in the body fat of the general population. A study of 35 men with 11 to 19 years of exposure in a DDT manufacturing plant shows the overall range of storage of isomers and metabolites in body fat ranged from 38 to 647 ppm, with an average of eight ppm for the general population. There is no evidence of progression of storage of DDT in the general population since 1950–1951.

Abstainers from meat stored on the average about half as much DDT and half as much DDE as persons in the general population who stored an average of 4.9 ppm and 6.1 ppm of the two compounds respectively. Agricultural occupational exposure caused marked increase in storage at an average concentration of 17.1 ppm. In a 1958 study applicators stored less than formulators but stored about three times as much DDT/DDE as neighbors with only environmental exposure. A 1961–1962 study showed no difference in levels of DDT/DDE between the general population and persons living in or near areas of extensive agricultural use. Differences in the two studies may be due to decreased use by more than 50% in the test area. One of the general

population samples came from a six-month-old bottle-fed boy whose extractible lipid levels for DDT (6.2 ppm) and DDE (16.5 ppm) were well within range of the general population. In six months he had stored at least the average concentration for the general population.

Human storage of DDT in England, West Germany, France, and Canada is lower than in the United States. Only in Hungary did the level of DDT storage equal or exceed that of the U.S. Sixty-five per cent of the material stored is DDE. Loss of DDT from storage, although always slow—less than 0.3% per day in man—is always more rapid when the storage is high than when it is low. Rate of excretion of DDT changes with concentration. In general, doubling the dosage will double the storage. A total of 282 autopsy samples of human abdominal fat tissue obtained at random from patients who had died of a variety of causes, regularly showed concentrations of 0.1 ppm or higher. The sum of DDE plus DDT averaged 10.3 ppm with standard deviation of 7.2 ppm. Nearly 96% of the values ranged from 0.1 to 22.3 ppm. About 72% of the DDT was present as DDE, the rest as DDT itself. Other studies of volunteers receiving 35 mg per man per day of technical DDT resulted in average storage in fat of 234 ppm and 281 ppm respectively. Other studies of 1961 to date give ranges of storage at between 2.3 and 4.0 ppm body fat, but the witness was uncertain. Concentration or storage depends on the dosage and the nature of the tissue in which stored and the rate of metabolism depends on the concentration. A point or plateau is reached where concentration/storage and rate of metabolism matches ingestion or dosage.

All organisms store DDT in varying amounts in fat, muscle, and internal organs.

6. Detection and Measurement

The laboratory equipment now most commonly used for the detection and measurement of chlorinated hydrocarbons is what is known as the gas chromatograph. Long U-shaped tubes filled with a powder-like material coated with resin are in a heat cabinet with temperatures around 200° C. A specific amount of a mixture of various chlorinated hydrocarbons in a solvent such as hexane is injected through the system at one end of the column. Some of the compounds come out earlier than others because of their physical-chemical properties and pass through a mechanism known as a detector, the most commonly used being known as an electron capture detector. The detector operates a recorder pen which marks a moving sheet of paper showing the times and amounts of gas which spin off the basic hydrocarbon ring. Peaks are made on the moving paper corresponding to the compound coming off. Because of chemical properties it occasionally happens that two of the compounds may come

out at or about the same time, creating one peak or confusing the type of peak. This may happen with a nonpolar column known as a DC-200 or a SE30 where compounds known as polychlorinated-biphenyls may come off at the same time as *p,p'* DDT. The interference is avoided by also using a QF-1 polar column where the retention times of the compounds will be differentiated. Laboratory procedures such as saponification can also be utilized. The chlorinated hydrocarbons are extracted from the sample provided, no matter what it may be, through certain laboratory procedures.

Almost all analytical techniques are based on pesticide manuals of the Food and Drug Administration listing techniques and how to verify conclusions. There are several techniques and procedures used for confirmation or verification.

Chlorinated hydrocarbon residues in organisms or samples of other material can be detected and measured in parts per million (ppm) up to .10 ppm in the Schecter-Haller colorimetric method and with the more sensitive gas chromatographs up to parts per billion (ppb).

7. Chlorinated hydrocarbons are tested for effects, storage, and residues by feeding tests, bio-assay, in "vitro" and in "vivo."

III. Enzyme Induction

A. Enzymes—Function and Body System Effects

1. Enzymes and Liver Function

Enzymes are catalysts, protein in nature, manifested morphologically by an increase in the smooth *endoplasmic reticulum* of the liver. The production of nonspecific hydroxylating enzymes by the *microsomal* fragment liver cells is a normal function of the liver. The existence of nonspecific enzymes and their production in the liver became evident in 1958. Two major functions of the liver are digestion and detoxification. Enzymes are produced in the liver to *hydrolyze* or *oxidize* non-polar substances such as fat and resins to a polar condition, capable of excretion by the kidney. They have not been isolated in purified form. They will also hydroxylate both endogenous and exogenous steroids which is a natural physiological process and will themselves disappear when no longer needed. The enzyme substrate affinity for steroids is much greater than it is for drugs and it has been shown that *o,p'* DDD stimulates the metabolism of hydrocortisone to 6-Beta hydroxy-cortisone in humans. Enhanced steroid metabolism causes compensatory synthesis of more steroid by organisms and raises problems of drug interaction.

Many drugs, compounds, and poisons induce detoxifying enzymes. For an example, while phenobarbital induces enzymes and is metabolized, a high level of enzymes induced by chlorinated hydrocarbons will decrease its physiological effect. Levels

of enzymes induced are directly proportional to the dose and rate.

An overall body concentration of one ppm and ten ppm of DDT in the fat—levels slightly below levels found in the human body—causes a significant increase in liver enzymes. Five ppm of DDT given to rats for three months caused an increase in enzyme activity in the rat liver and as little as 40 micrograms of DDT given to rats for four weeks caused an increase in the metabolism of several clinically useful drugs, an effect which extended for twelve weeks after the DDT feeding was stopped. Liver detoxification mechanisms are less highly developed in human infants than in adults and this is also true of rat infants.

2. Clinical Effects

There are no overt pathological changes in body organs at tissue levels of ten ppm. There are no overt pathological *histological* changes in rats fed DDT in very low amounts. This does not mean, however, that there are no bio-chemical or pharmacologic effects. Histologically, no effect is evident except a proliferation of the smooth endoplasmic reticulum of the liver cell following the administration of enzyme-inducing agents. Up to this time this effect has not been considered a pathological one and therefore has no pathological significance. The biochemical significance is a higher enzyme level and the metabolism at a faster rate of steroids and drugs.

Repeated oral doses of DDT to volunteers of approximately 200 times what the general population gets in their diet caused no clinical signs of illness. Dosages of 3.5 and 35 mg, depending on the exact weight of each man, averaged .05 and .5 mg/kg per day and did not produce any clinical illness. It did result, however, in increased storage. Storage in fat of 35 mg per man per day dosages of technical DDT was an average of 234 ppm in one study and 281 in another study. Average person's diet intake of DDT is .028 mg per man per day on values established for 1964 through 1967. Ten mg/kg produces illness in many people and sixteen mg/kg has frequently produced convulsions. Routine clinical examinations and simple laboratory tests of workers with great exposure to DDT failed to show any illness and disorders were looked for which had been produced by moderately larger dosages of DDT in man and in animals. There were no clinical or sub-clinical effects found. Clinical effects were primarily sought. No procedures were utilized to determine sub-clinical effects of enzyme induction. Not only the dosage that ordinary people get but the higher dosages workers get produce no detectable clinical effects. People who have not ingested enormous amounts of DDT have evidenced no manifest clinical symptoms.

3. Sub-clinical Effects

The principal sub-clinical metabolic effect of DDT is the ac-

celerated degradation of drugs, hormones, and *endocrines* as listed in preceding paragraphs. Hormones such as testosterone, estrogen, and progesterone in the human body function in a ratio of parts per billion (ppb) and are broken down by liver enzymes. Other chlorinated hydrocarbons such as polychlorinated biphenyls (PCB) also induce hepatic enzymes with the capacity to hydroxylate steroids. The PCB-induced enzyme produces a different polar metabolite. PCB is found in almost all organisms throughout the world.

The degradation of hormones without observable effect on the organism itself by DDT-induced hepatic enzymes is evidenced by what is known as "the thin eggshell phenomenon" in birds. High levels of hepatic enzymes cause a rapid metabolism of the avian estrogen necessary for the laying down of calcium in the medullary bone of birds. An upset in the calcium metabolism of the bird itself results in abnormal brooding behavior and the eating of its own eggs. Lack of available calcium carbonate results in thin eggshells.

In a controlled feeding study at the Patuxent Wildlife Research Center, Department of the Interior, mallards were fed DDT or metabolites DDD or DDE each separately at 10 ppm or 40 ppm on a dry weight basis. Mallards fed DDE cracked or broke 24% of their eggs, controls 4%, and good eggs produced had shells 13½% thinner than controls. Incubated eggs laid by ducks fed DDE produced less than one-half (½) as many healthy ducklings as did the controls. Both amounts of DDE produced effects of similar magnitude. A "no effect level" was not found and must be lower than the dosages given. Ducks fed *p,p'* DDT minus the *o,p* fraction at 25 ppm showed results similar to those produced by DDE at 10 or 40 ppm. DDD fed to ducks at 10 or 40 ppm produced normal eggs and healthy ducklings.

In another experiment, kestrels (sparrow hawks) were fed 2 ppm of DDT plus ⅓ ppm of dieldrin and also 5 ppm DDT plus 1 ppm of dieldrin. Shells of dosed birds were 15% thinner than the controls as were second generations fed diet of the parents.

Another experiment with the Japanese quail, was designed to determine what effect toxic levels of DDT fed in the growing period might have on reproduction efficiency later on, and whether resistance to DDT could be developed. The quail were fed 200 ppm until a 50% mortality was reached which was approximately 30 days. Survivors were bred for four generations. There was no effect on reproduction or shell thickness. No residue levels were determined. The Japanese quail is related physiologically to chickens, pheasants, and turkeys.

What role, if any, DDT plays in fish physiological processes is unknown. It is unknown whether the principle of hepatic enzyme induction is involved in fish reproduction.

B. Environmental Effects

1. Birds

a. Sea or Waterfowl

The effects shown in the Patuxent studies are similar to those experienced in the environment, resulting in declining raptor and water bird populations. The pelagic Bermuda petrel, the brown pelican, the peregrine falcon, the forked-tail petrel, the bald eagle, the osprey, the coopers hawk, the double-crested cormorant, and mallards are affected.

b. Upland Birds

The population trend on a nationwide basis so far as small game and upland game birds are concerned, taking into account anticipated population fluctuations, shows a generally healthy upward trend. This is true of cottontails, snowshoe hare and squirrels and birds such as pheasant, quail, grouse, and woodcock.

c. Omnivores

Golden eagles, red-tailed hawks, and the great horned owl are omnivores, feeding on both birds and mammals, and representative of reasonably stationary populations showing no statistically significant fluctuations in eggshell weight. The golden eagle in the British Isles is also declining and it is shown that in feeding on sheep carrion and fleece is ingesting DDT in combination with dieldrin, both being used as sheep-dips.

2. Fish

The University of New Hampshire studies on effects of DDT on reproduction of brook trout and their resistance to stress show that mortality where one gamete came from fish exposed to DDT was in every case higher than instances where neither of the gametes came from a fish exposed to DDT, and in five out of six cases the differences were statistically significant. The stress imposed was starvation and lowered water temperatures and the approach of spawning activity.

The findings in the New Hampshire study are consistent with the study and field investigation of the New York State Conservation Department, which showed a relatively close relationship between the amount of DDT in lake trout eggs and observed mortality. Canadian studies establish that when DDT and its metabolites occur in concentrations slightly above 400 ppb in eggs, mortality in the resulting fry ranged from 30% to 90% in the 60-day period following the swim-up stage. The Wisconsin studies, however, are inconclusive. Data indicated no relationship between pesticide residue levels in eggs and median life span of fry nor pesticide levels in fry and median life span of the fry. Difficulties in the experiment occurred and the experiment design differed also from the New York and New Hampshire studies.

IV. Effect on Nerves

A. Anatomy and Physiology

Exhibit 184 is a simplified diagram of a motor nerve cell showing receptor *dendrites*, the nerve soma or body, the axon and endplate. The axon or transmission part of the nerve cell is composed of a central core surrounded by a plasma membrane called the neurolemma. Next to the neurolemma is a Schwann cell which comprises the myelin sheath. The myelin sheath has openings or channels called the node of Ranvier.

The neurolemma is believed to be a laminated membrane with a lipid molecule interior sandwiched between layers of protein molecules with channels through the membrane. The term "channel" does not refer to an anatomical structure, but only refers to a conceptual pathway. Between the neurolemma and the cytoplasm is a concentration of potassium *ions* and on the outside of the membrane is a concentration of sodium ions. The membrane offers resistance to the passage of electrical currents and shows selective permeability to the exchange of the sodium and potassium ions. In the resting cell the inside of the membrane is negative to the outside.

In excitation of the cell, the nerve impulse received by the dendrites at a certain potential is passed on through the axon. With the decay of the potential due to resistance, sodium ions pass through the membrane to the inside and simultaneously the potassium ions flow outward. Both flows last but a thousandth of a second and at the moment the two effects balance one another, the inside of the membrane is slightly positive and the membrane restores ionic balance typical of the resting state. The excitation or nerve impulse passes down the lattice-type membrane by leaking currents through one node of Ranvier to the next node where sodium and potassium ions are exchanged through the neurolemma. The conductance is then not a smooth flow but a whole series of changes in potentials. (Examiner's Note: This is a gross oversimplification of an enormously complex, complicated neurophysiological reaction.)

DDT will increase membrane conductance to potassium or the inactivation of the nerve membrane conductance to sodium or both are inhibited, thereby increasing the negative after-potential and its prolongation. DDT does not simply reduce maximum sodium current, but instead gums open the channels or some of the channels for sodium, resulting in the prolonged action potential and the prolonged action current with an effect similar to *veratrine*, the alkaloid active ingredient of belladonna.

> . . . A prolongation of the active state of the nerve means—and coupled with no change in the inactivation process for sodium—that after one nerve impulse, when the channels become ready to conduct or to open again to conduct a second impulse, and examine or test the potential across the membrane, they will find

that according to the potential they should already go again, that is, no further impulse would be necessary to make it fire another signal. This could produce repetitive firing. On the other hand, in other nerves where sodium inactivation takes a longer time, one could simply find that the nerve on reactivating its sodium mechanism finds that the potential is already too high to fire an action potential, and instead would simply remain quiescent. This would produce a complete failure of the transmission line or at least intermittent failure. Either of these mechanisms quite clearly and very conclusively could cause tremors and could cause grave disturbances in terms of the ability of the animal to move or to make motions.

The effects of DDT on nerves involved in the experiments conducted did not wear off during the time course of the experiment. The effects are irreversible. There did not seem to be any concentration lower limit of DDT to create the effects shown. In cockroaches, insects, and crustaceans, effects occur at very low concentrations and are variable. Effect on the cockroach consisted of repetitive firing in nerves where one impulse applied to the nerve no longer evoked a single message but rather a large volley of messages. The behavior showed disorientation, running about, tremoring, kicking of legs in the air, and death. There is a similar effect on robins. How DDT reaches the nerves in the body is unknown.

It was stipulated that DDT has an effect on the nervous system. It is the nervous system that is primarily affected, and it does in fact stimulate all parts of the nervous system. The earliest subjective clinical sign of DDT poisoning is tingling of fingers and toes, and peculiar tingling about the mouth.

B. Environmental Effects

1. Fish

   At acutely toxic levels, the chlorinated hydrocarbons damage the central nervous system, causing instability, difficulty in respiration, and sluggishness in fish.

2. Birds

   In Hanover, New Hampshire, a study was made of effects of DDT on local birds after the town was sprayed for Dutch elm disease. Ninety-six dead birds had a concentration of about 30 ppm DDT. All birds with tremor had above 30 ppm in the whole bird.

   The study conducted at the East Lansing Campus at Michigan State University shows that birds, primarily robins, suffered from extensive tremor and death after the campus had been sprayed with DDT. More than 200 robins and 216 specimens of non-robins, representing about 50 different species, were analyzed. It was determined that the amount of DDT in the liver had no correlation with mortality. The brain was considered the best

criterion. DDT was found in 99.5% of birds from DDT-sprayed area. No robins with tremor were found that did not have 50 ppm or more DDT. All birds in tremor and dying birds had large concentrations of DDT in the brain.

3. Mammals

The giant squid *axon* fiber is like nerves of higher vertebrates, but unlike them has a very large diameter and is useful in nerve conductance experiments. Work done on the squid axon is applicable to nerves of both vertebrates and invertebrates. The effect of DDT on nerve conductance would not always be manifest in gross neuromuscular clinical signs and if manifested would not necessarily be the sole cause of the observed clinical signs.

The action of DDT in animals is manifested almost entirely through the nervous system with prominent signs of poisoning being muscle tremor, uncoordination, and convulsions. Concentration of DDT in the brain of rats fed *p,p'* DDT at 200 ppm for 90 days increased during a subsequent 10-day period of partial starvation. Increased concentration of DDT in the brain during starvation was correlated with clinical signs of poisoning. While all parts of the nervous system are affected by DDT the brain is of major importance.

V. Other Effects

A. Hormone Mimicry in Quail and Rats

An experiment designed to determine estrogenic effects resulted in a tripling of the wet weight of the coturnix quail oviduct when injected with 190 mg/kg of *o,p* DDT. No attempt was made to determine the amount that would cause the effect. The coturnix is a gallinaceous bird, related physiologically to chickens, pheasants and turkeys. Experiments at the University of Wisconsin designed to determine the effect toxic levels of DDT fed in the growing period would have on reproductive efficiency later on, and also whether DDT-resistant birds could be developed, showed no effect at all on coturnix eggshell thickness. The nationwide trend so far as small game and upland game birds are concerned shows "a generally healthy upward trend."

DDT also increases the uterine wet weight in immature female rats and in ovariectomized adult rats. The physiological significance of this effect is unknown.

B. DDT and Human Pathology

DDT concentrations were determined at autopsy in the fat and liver of 271 patients previously exhibiting pathological states of the liver, brain, and other tissues and compared with other random autopsy cases. There was a striking lack of correlation between concentrations in the liver and fat in all cases, but a significant correlation between levels in the brain and fat. There was no elevation of concentrations in the presence of brain tumors but a significant increase of the mean

p,p' DDE in encephalomalacia and cerebral hemorrhage. Significant concentrations of *p,p'* DDE were found in portal "cirrhosis" and highly significant concentrations in carcinoma. Fat concentrations of the various DDT analogs were consistently and significantly elevated in hypertension.

Individuals using pesticides extensively in the home had levels of *p,p'* DDT and *p,p'* DDE three to four times higher than those who had used minimal quantities. No conclusions can be drawn on the role of pesticide exposure in the production of the diseases without confirmatory studies to determine whether the diseases caused high DDT levels or vice versa.

Opinion

Clinically observable toxic effects of DDT in humans are obtained only with extremely large dosages by sudden extreme exposure, or of accidental origin. Clinically observable effects are evident injury, illness, loss of body function which directly inconveniences a person at work or play. Toxicity, as the word is ordinarily expressed, is related to dosage which in turn is related to storage.

DDT is ubiquitous. It is found in the atmosphere, soil, water, and in food in what might be considered minute amounts. The chemical property of being soluble in lipid or fat tissue results in storage primarily in the body fat and nervous systems of all organisms in all levels of food chains. It is therefore impossible to establish levels, tolerances or concentrations at which DDT is toxic or harmful to human, animal, or aquatic life.

The principle that DDT, being a chlorinated hydrocarbon, induces the production of non-specific detoxifying hydroxylating hepatic enzymes is well established. The induction of the enzymes is a normal adaptive hepatic process for the detoxification of substances and no definitive pathological effects are observed at present dosages. A high level of induced hepatic hydroxylating enzymes, however, causes a pharmacological biochemical effect in accelerating the metabolism of body steroids and drugs such as barbiturates and nonbarbiturate depressants.

While the exact physiological mechanisms are not known in enzyme induction, it is established by feeding studies that DDT and one or more of its metabolites will by themselves cause a thinning of eggshells in raptor, pelagic, or waterfowl birds. The effect explains the existence of the phenomena in the environment but does not exclude other causative factors, namely diet, illness, and other chlorinated hydrocarbons among them being polychlorinated biphenyls. The appearance of the phenomena, however, on two continents simultaneously, would seem to eliminate illness, diet, or predator interference as causative factors. Waterfowl and raptors on the top of water and

other food chains are suffering decline and insect/worm-eaters are affected whereas gallinaceous birds are not. The differences in dosage reactions can also be explained by well-known order differences in birds.

The effect of DDT in minute amounts on the extremely complex, complicated mammalian nerve system is unknown. Huge dosages of DDT bordering on the accidental will cause gross clinically observable neurological symptoms in humans. It is uncontroverted that DDT has an almost immediate nerve effect on the primitive nerve systems of insects and on the less well-developed nervous systems of other forms of life. It is also uncontroverted that nerve tissue of vertebrates and invertebrates is the same, that DDT has a harmful effect on nerve conductance as shown by experiments on the axon of crustaceans and amphibians, that the effects are irreversible during the duration of the experiments. Clinically observable signs of nerve effects in humans such as tremor disappear upon reduction of dosage. That there are sub-clinical residual effects can only be postulated on mathematical equations and principles worked out in conjunction with nerve conductance experiments on nerve axons of crustaceans and amphibians and shown to be valid in all cases. Taking into consideration the above experiments together with the fact that DDT is used as a rodenticide for mice and bats, the only valid permissible inference is that DDT in small dosages has a harmful residual effect on the mammalian nervous system.

While the physiological mechanism causing a reduced reproductive success in fish and a reduced resistance to stress when dosed with fairly high levels of DDT is unknown, the known effects themselves can only be considered harmful ones.

The record is replete with evidence of the economic benefits derived from use of DDT in the control of pests in agriculture and in the control of mosquitoes for both comfort and prevention of disease. Without doubt DDT has provided enormous economic benefits, but economic benefits are not an issue or part of any issue in this case.

Ruling

DDT, including one or more of its metabolites in any concentration or in combination with other chemicals at any level, within any tolerances, or in any amounts, is harmful to humans and found to be of public health significance. No concentrations, levels, tolerances, or amounts can be established. Chemical properties and characteristics of DDT enable it to be stored or accumulated in the human body and in each trophic level of various food chains, particularly the aquatic, which provides food for human consumption. Its ingestion and dosage therefore cannot be controlled and consequently its storage is uncontrolled. Minute amounts of the chemical, while not

producing observable clinical effects, do have biochemical, pharmacological, and neurophysiological effects of public health significance.

No acute or chronic levels of DDT which are harmful to animal or aquatic life can be established. For the reasons above set forth, a chronic level may become an acute level. Feeding tests, laboratory experiments, and environmental studies establish that DDT or one or more of its analogs is harmful to raptors and waterfowl by interfering with their reproductive process and in other birds by having a direct neurophysiological effect.

Feeding tests or experiments and environmental studies establish that DDT at chronic low levels is harmful to fish by reducing their resistance to stress.

DDT and its analogs are therefore environmental pollutants within the definitions of Sections 144.01 (11) and 144.30 (9), Wisconsin Statutes, by contaminating and rendering unclean and impure the air, land, and waters of the state and making the same injurious to public health and deleterious to fish, bird, and animal life.

Dated at Madison, Wisconsin, this 21st day of May, 1970.

STATE OF WISCONSIN

Model Pesticide Law

SECTION 1. Preamble

Since pesticides are useful in the control of certain insects, weeds, fungi and other forms of plant and animal life which have caused significant damage to man and his interests, but at the same time pesticides may contaminate the environment and have undesirable ecological effects, and therefore should be used to augment natural controls, it is deemed necessary for greater protection of the public health and welfare and to insure environmental quality consistent with the benefits derived from the safe and proper application of pesticides, to establish a pesticide control board that formulates pesticide policy in the .....(municipality)..... and administers and coordinates state efforts to control the use of pesticides in the (municipality)

SECTION 2. Definitions

In this act:

(1) *"Board"* means the pesticide control board.

(2) *"Control"* means maintaining pest population density at or below the economic threshold.

(3) *"Economic threshold"* means the pest population density above which there is significant damage to man or his interests.

(4) *"Non-target organism"* means any organism which the particular pesticide is not intended to control in a given application.

(5) *"Pest"* means any organism that is present at a population density above the economic threshold.

(6) *"Pesticide"* means any substance or mixture of substances intended to control pests and includes those substances commonly referred to as insecticides, fungicides, herbicides, and rodenticides.

(7) *"Target organism"* means any organism which the particular pesticide is intended to control in a given application.

SECTION 3. Pesticide Control Board

(1) Appointment, membership. There is hereby created a pesticide control board consisting of 7 members, to be appointed by the .....(municipal executive)..... with the approval of the .....(municipal legislature)..... The appointments shall be made in writing and filed in the office of the .....(municipal executive)..... The term of each member shall be the three years next following January 1 of the year in which his appointment is made and until the appointment of his successor except that the first six members shall be appointed respectively for such terms that on January 1 in each of the three years next following the year in which they are appointed the terms of two members will expire and except that a member appointed to replace a member who did not complete his full term shall be appointed for the balance of the term. Insofar as possible the board shall include appointed members representative of each of the following fields: aquatic or marine biology, agriculture, ecology, entomology, fish or game management, and pesticide application.

The following or their delegates shall serve as *ex officio* members of the Board: .....

.....

.....

(2) Organization

(a) The municipal executive shall call the first meeting of the board at the municipal hall without delay. The board shall elect from its membership a chairman and a secretary who shall serve for two years terms ending on January 1 next following a municipal general election. Meetings may be called by the chairman and shall be called on request of any two members, and may be held as often as necessary but not less than four times each year. Five members of the board constitute a quorum.

(b) The members of the board and of committees appointed by it shall receive no salary as members but shall be reimbursed their traveling and other expenses incurred in attending meetings of the board or committees or while in the performance of their duties as members.

(c) The secretary of the board shall be responsible for giving notice of meetings of the board and for the preparation of the agenda for meetings. He shall also be responsible for preparing and editing the minutes of the meetings of the board and the reports of the board for the municipal government and the general public.

(d) The board may appoint committees which may include employees and members of the several state, federal and local boards, commissions, departments, offices and agencies, having some competence in the matters under consideration, and may authorize such committees to make investigations and surveys and to report

to the board on such matters as may be necessary to enable the board to carry out the purposes of this act.

(e) The board shall make a report of its actions taken under this act and its recommendations to the municipal executive and municipal government. The board shall make reports of its actions and recommendations to the several state boards, commissions, departments, offices and agencies as it deems advisable.

(f) All data relating to the registration of a pesticide, licensing of a pesticide applicator or issuing of a permit for pesticide use shall be public records.

SECTION 4. Registration of Pesticides

Before any pesticide may be used or sold in this .....(municipality)..... it must be registered with the board. Registration will be granted only after an application has been filed with the secretary and only after the affirmative vote of five or more of the members present at a scheduled meeting. An application for registration must include the following:

(1) Evidence that the pesticide has been registered with the United States Department of Agriculture.

(2) Reliable scientific data showing:

(a) The amount of pesticide, determinable in units of treatment concentration for specific methods of application, required to reduce pest populations to or below the economic threshold.

(b) The ecological characteristics of the pesticide in the environment, particularly its:

1. chemical stability (persistence)
2. mobility
3. solubility characteristics
4. effect on non-target organisms

(3) Such other information as the board may require.

SECTION 5. Use of Pesticides

As to each pesticide registered with the board, the board shall determine after public hearing upon notice the potential hazard, if any, to the natural resources of the .....(municipality)..... which might result from the use of such pesticide and set limitations on the proposed use that will prevent hazard to natural resources other than the target organism. If the potential hazard to the natural resources of the municipality other than the target organism is undeterminable after the hearing and the economic interests of the municipality justify cautious experimentation with the pesticide, the board may issue limited permits for use during one season in

selected areas and require that scientific data be gathered to enable the board to determine the hazard, if any, to the natural resources of the municipality other than the target organism. No pesticide shall be sold, distributed or used in the .....(municipality)..... until regulations regarding its use are issued by the board and all applications of the pesticide shall be made in accordance with those regulations.

SECTION 6. Licenses and Permits

(1) The board shall make regulations concerning and shall issue annual licenses to qualified pesticide applicators who apply for a license. The cost of the license shall be $..... . The board shall provide for the occasional inspection of work of the licensed applicators without notice to insure compliance with the statutes and regulations. Licenses of applicators who do not comply will be revoked. The board may make regulations concerning and may require and issue permits for particular types of pesticide application and for the application of particular pesticides.

(2) Any person aggrieved by any decision of the board, whether affirmative or negative in form, which relates to granting or revoking his license or permit is entitled to review thereof in accordance with the laws of the state of ..... .

SECTION 7. Hearing

The board shall hold with substantial adherence to the rules of evidence applicable to judicial proceedings, a public hearing relating to registration of a pesticide or to alleged or potential environmental degradation by pesticides upon the verified petition of six or more citizens filed with the board. The petition shall state the name and address of a person within the state authorized to receive service of answer and other papers in behalf of petitioners. The board shall serve a copy of the petition and notice of the hearing upon the applicant for registration or the person responsible for the alleged or potential degradation either personally or by registered mail directed to his last known post office address at least 20 days prior to the time set for the hearing which shall be held not later than 90 days from the filing of the petition. The respondent shall file his verified answer to the petition with the board and serve a copy on the person so designated by the petitioners not later than five days prior to the date set for the hearing, unless the time for answering is extended by the board for cause shown. For the purposes of any hearing under this act the secretary may issue subpoenas and administer oaths. Within 30 days after the closing of the hearing, the board shall make and file its findings of fact, conclusions of law and order, which shall be subject to review under Article ..... of the .....

Law and Rules of the state of ..... . If the board determines that any complaint has been filed maliciously or in bad faith, it shall so find and the person complained against shall be entitled to recover his expenses on the hearing in a civil action.

SECTION 8. Declaratory Ruling

(1) The board may, on petition filed with the board by any interested person, issue a declaratory ruling determining the applicability to any person, property or state of facts of any rule or statute enforced by it or promulgating new or amended administrative rules. Within a reasonable time after receipt of the petition, the board shall either deny the petition in writing or schedule the matter for hearing. If the board denies the petition, it shall promptly notify the person who filed the petition of its decision, including a brief statement of the reasons therefor. If the petition is granted, full opportunity for hearing conducted under the rules of evidence applicable to a judicial proceeding shall be afforded to interested parties. A declaratory ruling shall bind the board and all parties to the proceedings on the statement of facts alleged unless it is altered or set aside by a court. A ruling shall be subject to review in the ..... court of the state of ..... in the manner provided for the review of administrative decisions.

SECTION 9. Violations

(1) Any person who shall violate any of the provisions of this ordinance or any rule, regulation or specification promulgated thereunder, shall be guilty of an offense.

(2) Each and every day during which such violation shall continue shall constitute a separate violation and a separate offense.

SECTION 10. Penalties

Any person convicted of violating the provisions of this ordinance or the rules, regulations and specifications promulgated thereunder shall be fined in an amount not less than ..... dollars nor more than ..... for each separate offense.

SECTION 11. Existing Rights and Remedies Preserved

Nothing herein contained shall abridge or alter any rights of action or remedies now or hereinafter existing, nor shall this ordinance, nor any provision thereof, nor any rule or regulation promulgated thereunder, be construed as estopping the .....(municipality)..... ..... from exercising its rights and fulfilling its obligations to protect the public health and welfare.

SECTION 12. Conflicting Laws

This article shall be construed to be ancillary to and supplementing any laws now in force tending to effect the purposes set forth in Section 1 of this ordinance, excepting as they may be in conflict herewith, in which case provisions of this Title shall govern.

SECTION 13. Local Laws, Ordinances and Regulations

Any local laws, ordinances or regulations which are not inconsistent with this article or with any code, rule or regulation which shall be promulgated pursuant to this ordinance shall not be superseded by it, and nothing in this ordinance, or in any code, rule, or regulation which shall be promulgated hereunder, shall prevent the adoption of any local laws, ordinances or regulations which are not inconsistent with this Title or with any code, rule or regulation which shall be promulgated hereunder.

SECTION 14. Separability Clause

If any clause, sentence, paragraph, section or part of this article shall be adjudged by any court of competent jurisdiction to be invalid, the judgment shall not affect, impair or invalidate the remainder of this article, but shall be confined in its operation to the clause, sentence, paragraph, section or part of this article that shall be directly involved in the controversy in which such judgment shall have been rendered.*

*This model law was prepared by Victor John Yannacone, Jr. and presented to the 1969 Mid-Winter Republican Governor's Conference, Hot Springs, Arkansas.