Systems Studies of DDT Transport

A systems analysis provides new insights for predicting long-term impacts of DDT in ecosystems.

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During the recent hearings conducted by the State Department of Natural Resources considering a petition to ban the use of DDT (*1*) in Wisconsin (*2*, *3*), both the petitioners and the defendants produced witnesses who gave testimony from a wide range of scientific disciplines. The evidence presented included a description of the transport mechanisms of DDT, its chemical properties, its physiological effects on individual organisms, and its effects on whole populations. In this article we describe a model for DDT transport which was presented as testimony to integrate the range of evidence. The resulting systems analysis yields testable hypotheses, demonstrates gaps in our understanding of the impact of DDT, and indicates possible future consequences of its use.

First, a mechanistic model is developed to describe the movement of DDT and its breakdown product DDE (*1*) in an inland ecosystem. The analysis is based upon the trophic-level concept (*4*) which allows a simplified quantitative examination of complex "food web" processes in ecosystems. Trophic levels in their basic form may be represented by a pyramid of energy, or a pyramid of biomass, such that there is more energy or biomass in green plants than in herbivores, and more in herbivores than in their predators. These concepts allow use of mathematical formulations for the flows and storages of DDT in the ecosystem. The mechanisms leading to selective concentration of DDT in specific living organisms are then discussed and a mathematical model is formulated to describe how DDT or DDE concentration varies with time and with trophic level. Finally, a mathematical model is derived to indicate the dependence of population size in any trophic level upon the populations in adjacent levels, and to provide a basis for predicting population changes attributable to DDT.

The results suggest, first, that even if no more DDT is ever added to the biosphere, its concentration in certain species at or near the top of the trophic structure could continue to rise for some years. In the light of the known broad range of DDT and DDE toxicity, additional species may decline or disappear. The population analysis indicates that secondary changes in prey populations would occur in response to direct effects on predator numbers. The methods described in this article should be viewed as an example of the approach that will have to be followed in many future studies of persistent contaminants in the environment if we are to make satisfactory estimates of long-term effects.

Descriptive Models for the Transport of DDT

The inputs, outputs, and storages of DDT in a Wisconsin regional ecosystem are shown schematically in Fig. 1. The ecosystem is divided into three levels—atmosphere, terrestrial biomass, and substrate water with its associated aquatic organisms. The major sources of DDT entering Wisconsin are listed as *Inputs*. DDT may be introduced into any one of the three levels. Examples are transport into the Wisconsin atmosphere by means of DDT attached to particulate matter carried by wind, commercial application of DDT to the land surface, and DDT brought into the Wisconsin water system by lake and river currents. DDT and its breakdown products leave the Wisconsin regional ecosystem by similar transport processes. There do not appear to be any mechanisms that will completely degrade these toxic breakdown products at rates comparable to the present commercial application of DDT.

All available evidence indicates that the total output of DDT and its breakdown products is considerably less than the total input, the remainder being stored (*3*, *5*). First, there is buildup of DDT in the lipid portions of living organisms and this DDT is subsequently retained in dead tissue for varying periods of time. Second, there is long-term accumulation in the soil, in deep bodies of water, and in deep organic deposits in marshes and lake borders. A complete determination of DDT inputs and their redistribution requires a detailed examination of the mechanisms controlling redistribution of DDT in the natural environment. Research at the University of Wisconsin on the movement of nutrients through lake and stream systems has provided a means of examining in detail these inputs, storages, transformations, and losses of DDT from both terrestrial and aquatic environments (*6*).

A complete listing of the DDT inputs, transports, and outputs in air, in water, and in living organisms, as well as the potential transformation to breakdown products in the air and in the organisms, was prepared as a foundation to the generalized ecosystem trophic structure shown in Fig. 2. The three basic carrier systems—atmosphere, water, and living biomass—provide the basis for a series of differential equations which, taken together, would permit mathematical simulation of the flow of any transported material such as DDT or its breakdown products in an ecosystem (*6*). As represented in Fig. 2, all of the exchanges of DDT from one carrier variable to another within the system are included,

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and also the sites at which transformations of DDT to degradation products take place. All information available to date indicates that a degradation product such as DDE also will be transported and redistributed by the same mechanisms. The general analysis of transports showed that many of the exchanges of DDT and DDE occurred in the subsurface physical environment where degradation cannot take place. In addition, it showed the sites in both the physical environment and in living and dead biomass where significant storage of DDT and DDE can be expected because of either the slow exchange of the host material or a slow breakdown process.

Woodwell and others (*7*, *8*) have described the role of the trophic structure in transporting and concentrating DDT in the ecosystem. The listing of the transport variables and flows of DDT in the environment through each successive level of the trophic structure can be summarized schematically as in Fig. 2, and more precisely in the equations that follow. Storage of DDT in each trophic level, transport from one trophic level to another, and transformation of DDT into DDE or DDD (*1*) by metabolism are examined in view of the processes involved within each trophic level and between each pair of trophic levels (*6*). Thus, all available information on the transport, accumulation, and transformation of DDT in natural biological systems was drawn together as a foundation for the mathematical analyses that follow.

DDT-Concentrating Mechanisms in Natural Systems

DDT has been described (*3*) as a chemical that "combines in a single molecule the properties of broad biological activity, chemical stability, mobility, and solubility characteristics that cause it to be accumulated by living organisms. . . ." DDT has a solubility in water of only 1.2 parts per billion (*9*) and a low vapor pressure (*10*). However, the vast amounts of air and water moving in the atmosphere and oceans transport significant quantities of DDT in relatively short periods of time. The result is that DDT has become ubiquitous.

In contrast to its near insolubility in water, the solubility of DDT in lipids and other organic materials is very high (*5*). These properties account for DDT accumulation in the lipids of plants and animals. DDT can be taken up actively with water and nutrients, or simply absorbed when an organism is exposed to water or air containing the pesticide. Uptake by exposure can occur, for example, through the gills of fish or the skin of terrestrial animals, or directly into the cells of aquatic plants.

The chlorinated hydrocarbons are chemically stable; DDT stored in the lipids of the organisms in a given trophic level often undergoes little degradation (*5*). The combination of high solubility and high stability allows "magnification" of DDT concentrations from lower to higher trophic levels within an ecosystem. Individuals in each trophic level feed on those in the levels below, and the proportion of food that an individual in a particular level converts into biomass of its own species is usually much less than 50 percent (*7*). The rest is excreted after the organism removes and respires much of the energy stored in it. Furthermore, organisms that grow to full size early in their life spans and others that give relatively little biomass to the egg or fetal stages of their young will tend to respire an even greater percentage of their food intake. Therefore, a substance like DDT, which is stored in the lipids and breaks down slowly, can accumulate to high concentrations in a trophic level.

In Table 1 are summarized available data on DDT concentration in a Lake Michigan ecosystem. Because of the concentrating mechanism described above, the concentration of DDT in the herring gull is some 7000 times that in the bottom muds. This increase in concentration may be even higher in other ecosystems; Woodwell *et al.* (*8*) found that concentrations in individuals from the top trophic level were some 10^6 times higher than those in the environment.

Data on the toxicity of DDT for the species in the Lake Michigan ecosystem are limited, but other data indicate that the species vary considerably in sensitivity to DDT. Some organisms in low trophic levels show adverse effects from concentrations much lower than those that are known to affect animals higher in the structure (*3*, *5*). American kestrels fed DDT and dieldrin (*1*) in food (6 to 18 parts per million) underwent reproductive failure (*11*). Field observations of falcons and eagles show similar effects at relatively high concentrations in food and body tissues (*12*). The brain tissue of robins killed in an elm-spraying program showed high concentrations (50 parts per million) of DDT (*13*). Lower in the trophic structure, 39 percent of adult brine shrimp died within 3 weeks when placed in a solution of 1 part DDT in 10^{12} parts water, and all died within 5 days in concentrations of 1 part in 10^{10} (*14*). Finally, only a few parts DDT in 10^9 parts water are necessary to reduce photosynthesis in a number of species of phytoplankton (*15*).

Taken together, the chemical and physical properties of DDT allow it to "flow" to and concentrate in living tissue, especially in organisms in high

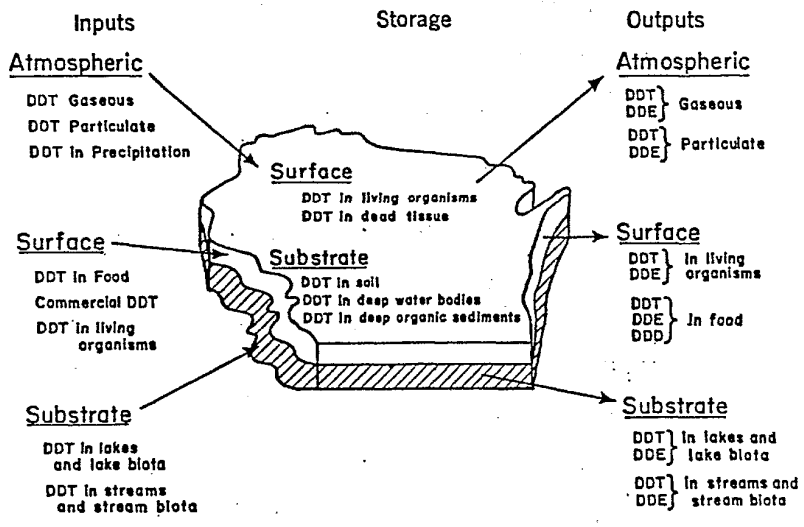

Fig. 1. Transport of DDT in the Wisconsin regional ecosystem. The major pathways by which DDT and its by-products flow throughout the ecosystem are shown.

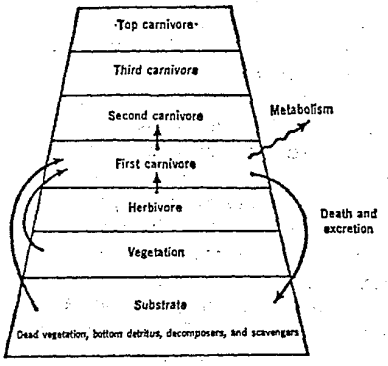

Fig. 2. Schematic representation of the flows of DDT in the ecosystem. The flows of DDT are shown for the first carnivore level only. Pathways similar to those indicated by the arrows exist between any other level and all other levels but are not shown for simplicity. Storage of DDT also occurs in each level.

trophic levels. However, low concentrations in individuals in low trophic levels may be equally alarming owing to the apparent greater sensitivity of some organisms in those levels.

Mathematical Analysis of DDT Concentration

A mathematical model of the movement of DDT from one trophic level to another in an ecosystem based on established information on the transporting and concentrating processes of DDT has been developed. The objectives of this model are: (i) to state quantitative relationships that show how DDT and its metabolites are concentrated in the various trophic levels, and (ii) to indicate the dynamic nature of the transport and concentrating processes. The transport of DDT is considered, but the analysis also applies to DDE and other products of similar chemical and biological properties.

The transports and storage of DDT identified in Fig. 1 are depicted schematically in trophic level form in Fig. 2. The possible pathways by which DDT may enter or leave are shown for the first carnivore level only.

DDT is carried by the flows of matter. The flows of mass into and out of a given trophic level i are related by the conservation of mass principle, where $\dot{m}$ is used to denote a mass flow rate (for example, in kilograms per day) entering or leaving a level.

$$\dot{m}_{\text{in},i} = \dot{m}_{\text{out},i} \tag{1}$$

The total mass of each trophic level is assumed to be constant. We recognize that population fluctuations exist, and in this analysis the time-average mass of the level is used. The inflow of mass is the sum of the rates of exposure to environment and of the ingestion of organisms in the lower levels, $\dot{m}_{j,i}$

$$\dot{m}_{\text{in},i} = \sum_{j=1}^{i-1} \dot{m}_{j,i} \tag{2}$$

The outflow of mass from the level is due to death, $\dot{m}_{d,i}$, and excretion, $\dot{m}_{ex,i}$

$$\dot{m}_{\text{out},i} = \dot{m}_{d,i} + \dot{m}_{ex,i} \tag{3}$$

Equations 2 and 3 can be combined with Eq. 1 to yield an expression for the flow of biomass through any trophic level

$$\sum_{j=1}^{i-1} \dot{m}_{j,i} = \dot{m}_{d,i} + \dot{m}_{ex,i} \tag{4}$$

For a given trophic level i, DDT is carried into and out of the level with these mass flows, destroyed by metabolic processes, and stored in the lipid biomass of the individuals in the trophic level. The mass flows are related by the conservation of mass principle, where $\dot{p}$ denotes the flow rate of the pesticide (DDT) (for example, in kilograms per day) and $\dot{p}_{\text{met},i}$ is the rate of metabolism of DDT in level i

$$\dot{p}_{\text{in},i} = \dot{p}_{\text{out},i} + \dot{p}_{\text{met},i} + \left(\frac{dp}{dt}\right)_i \tag{5}$$

where dp/dt is the rate of storage of DDT in the trophic level.

The flow of the pesticide into the i level is expressed as the product of the rate of ingestion or exposure to the species in lower levels $\dot{m}_{j,i}$ and the instantaneous average concentration c_j of DDT in that lower level. The inflow is the sum of these terms for all such lower levels

$$\dot{p}_{\text{in},i} = \sum_{j=1}^{i-1} c_j \dot{m}_{j,i} \tag{6}$$

For application to DDE, or similar byproducts of DDT metabolism, the inflow term would also include creation of the substance from DDT.

The flow of DDT from the level by death, predation, or other causes is the product of the instantaneous average concentration c_i in the level and the death rate $\dot{m}_{d,i}$. In addition, DDT leaves by excretion in amount $c_{ex}\dot{m}_{ex,i}$ where c_{ex} is the DDT concentration.

$$\dot{p}_{\text{out},i} = c_i \dot{m}_{d,i} + c_{ex}\dot{m}_{ex,i} \tag{7}$$

Table 1. Concentration of DDT in a Lake Michigan ecosystem (27).

Trophic level	Concentration (parts per 10^9)
Bottom muds	14
Amphipods	410
Fish	3,000–6,000
Herring gulls	99,000

The rate of storage of DDT in the i level is the product of the total mass in the level m_i and the rate of change of the average concentration with respect to time

$$\frac{dp}{dt} = m_i \frac{dc_i}{dt} \tag{8}$$

Combining Eqs. 6, 7, and 8 with Eq. 5 and rearranging yields an expression for the DDT concentration in a given level as a function of time

$$\left(\frac{\dot{m}_i}{\dot{m}_{d,i}}\right)\frac{dc_i}{dt} + c_i = \frac{1}{\dot{m}_{d,i}}\left[\sum_{j=1}^{i-1} \dot{m}_{j,i}c_j - \dot{p}_{\text{met},i} - c_{ex}\dot{m}_{ex,i}\right] \tag{9}$$

The expression for the substrate, level 1 of Fig. 2, has additional inflow terms due to man, $\dot{p}_{\text{man}}$, excretion from the upper levels, and the fraction f_i of those individuals in the upper levels that die naturally. DDT may also leave the substrate in amounts $\dot{p}_{\text{out}}$ through the mechanisms described in Fig. 1. Therefore

$$\frac{dc_1}{dt} + \left(\frac{\sum_{i=2}^{I} \dot{m}_{1,i}}{m_1}\right) c_1 = \frac{1}{m_1}\left[\dot{p}_{\text{man}} - \dot{p}_{\text{out}} + \sum_{i=2}^{I} (c_{ex}\dot{m}_{ex,i} + c_i f_i \dot{m}_{d,i})\right] \tag{10}$$

where I is the total number of trophic levels.

Equations 9 and 10 are general equations that describe the rate of change of DDT or any other pesticide in the various levels of the ecosystem. At present, there are insufficient data to allow an evaluation of all the terms. Nevertheless, a qualitative study of these equations can provide valuable insight into pesticide transport through an ecosystem.

The term $(m_i/\dot{m}_{d,i})$ in Eq. 9 is related both to the time it takes the trophic level to respond and to the equilibrium concentrations of DDT in

the level. In order to show this more clearly, the average values for the mass and life span of the individuals in a trophic level will be used. The total mass in a level is the product of the number of individuals N_i and the average mass of each member M_i, or

$$m_i = N_i M_i$$

The death rate is the product of the average mass of the individuals and the number dying per unit time $N_{d,i}$, or

$$\dot{m}_{d,i} = M_i N_{d,i}$$

The average life span of the members is denoted T_i. The number dying per unit time is then $(1/T_i)$ times the total number of individuals, or

$$N_{d,i} = N_i / T_i$$

Combining these relations yields

$$\frac{m_i}{\dot{m}_{d,i}} = \frac{N_i M_i}{(M_i N i / T_i)} = T_i \qquad (11)$$

It is now possible to deduce the equilibrium levels of DDT once the ecosystem reaches a steady state. With the simplifying assumption that there is no further addition of DDT to the system, and that the term representing the metabolism of DDT is small relative to the other terms (*16*), the equilibrium concentration C_i from Eq. 9 is

$$C_i = \frac{T_i}{m_i}\left[\sum_{j=1}^{i-1} \dot{m}_{j,i} C_j - C_{ex} \dot{m}_{ex,i}\right] \qquad (12)$$

Equation 12 states that the equilibrium concentration of DDT in a level is: (i) directly proportional to the average life span T_i of its members; (ii) inversely proportional to the total mass m_i of the level; and (iii) proportional to the net retention of DDT in the level. The net retention in a level depends on the concentration of DDT in the lower levels, the rate at which organisms of the lower levels are ingested, and the amount of DDT excreted. Equation 12 is based on the assumption that the metabolism of DDT in the trophic structure is negligible; metabolism would serve to reduce the net retention in a level. Metabolism would also prevent the attainment of a true equilibrium condition. However, Eq. 12, modified to account for metabolism, would still provide an estimate of the concentration in a level under these conditions. The effects of life span and mass would be unchanged. In general, as one moves up the ecosystem (Fig. 2), the average life spans increase and the mass in the trophic level decreases. Equation 12 provides an explanation for the observed increased concentration of DDT in the higher levels of the ecosystem.

The dynamic nature of pesticide flows in an ecosystem are determined from the solution of Eqs. 9 and 10. However, there are virtually no quantitative data on the flow of DDT into and out of natural populations and on the movement of biomass through predator-prey interactions. To indicate the dynamic nature of the concentrating process, we consider a simplified, but representative, situation. For this approximation, we assume that organisms in all consumer levels feed only on the organisms in the level immediately below, retain all DDT ingested, and neither metabolize nor excrete DDT. Equation 9, when combined with Eqs. 4 and 11, can then be simplified to

$$T_i \frac{dc_i}{dt} + c_i = \left(\frac{\dot{m}_{i-1}}{\dot{m}_{i-1} - \dot{m}_{ex,i}}\right) c_{i-1} \qquad (13)$$

The equilibrium concentration is then

$$C_i = \left(\frac{\dot{m}_{i-1}}{\dot{m}_{i-1} - \dot{m}_{ex,i}}\right) C_{i-1} \qquad (14)$$

The coefficient of C_{i-1} in Eq. 14 is the ratio of the rate of mass ingested to the difference between the rates of ingestion and excretion. For an individual member, the difference between ingestion and excretion over the life span is the body weight. Thus, the coefficient of C_{i-1} can be written as the ratio of mass ingested over a lifetime to body weight. This coefficient is always greater than unity and probably ranges between 10 and 10,000. Thus, the equilibrium concentrations increase as one moves from lower to higher levels in the ecosystem.

The coefficient T_i in Eq. 13 is the time constant (*17*) of the level, and indicates the ecosystem response times. For example, a sudden sustained increase in concentration in the $i-1$ level yields an exponential increase in the concentration c_i

$$c_i = \left(\frac{\dot{m}_{i-1}}{\dot{m}_{i-1} - \dot{m}_{ex,i}}\right)(1 - e^{-t/T_i})\, c_{i-1} \qquad (15)$$

Calculations made with Eq. 15 show that the concentration in the i level reaches 98.2 percent of its equilibrium value in a time equal to 4 T_i. Each trophic level requires about four average life spans to reach equilibrium in response to changes in DDT concentration in the level below it.

This conclusion regarding response times is based on the simple system described by Eq. 13. Feedback loops, represented by the additional terms on the right side of Eq. 9, provide a more accurate model of an ecosystem and these increase the time required to respond (*17*). Thus the prediction that the ecosystem cannot reach equilibrium until about four times the longest average life span is conservative.

The mathematical development is based on populations containing individuals at all ages, with deaths occurring in equal proportions in all age groups. This assumption is an oversimplification for natural populations when all ages are included, although adult birds tend to have a linearly decreasing expectation of further life as a function of time (*18*).

An alternate model for the age distribution in a population is that all deaths occur at an age corresponding to the average life span. With this model it can be shown that pesticide concentration in an ecosystem described by Eq. 13 cannot reach equilibrium until a time equal to the sum of the average life spans in all trophic levels. Concentrations in a more complex ecosystem described by Eqs. 9 and 10 would take longer to reach equilibrium because of the complex feedback loops.

Our estimates for the length of time necessary to reach equilibrium after the introduction of DDT, or any similar pesticide, depend upon the life spans in the trophic structure and the age distributions. We estimate that this time lies between four times the average life span of the longest-lived species and the sum of the life spans for all trophic levels.

We are not certain what value of average life span most closely approximates what occurs in nature, but it is most likely between the maximum attainable life and the expected life at birth (mean death age) for any species. In ecosystems with long-lived members such as the herring gull with a life span between 2.8 years (*19*) and 40 to 50 years (*20*), and the osprey, eagle, and falcon with life spans of as long as 60 to 100 years (*20*), it is quite apparent that the full effects of today's use of DDT will not be completely felt for many years to come. Furthermore, it is easily possible that ecosystems with such long-lived constituents have not yet felt the full impact of the original use of DDT in the late 1940's.

The model result, Eq. 15, describes the redistribution of an initial step input of DDT to the ecosystem. All inputs can be synthesized as a series of steps, and thus the time constant represents the time response of the system to all inputs. Therefore, the concentra-

tion of DDT or DDE in any species or trophic level at the present time reflects the addition of the responses to all of the step inputs of DDT to the present. In view of the continuing worldwide inputs of DDT, it is readily apparent that the Wisconsin ecosystem is not yet in equilibrium.

Population Response to Declining Predator Control

In the previous section, mathematical expressions relating the dynamic nature of pesticide flows were developed. In an earlier section, the impact of these DDT levels on major carnivores such as falcons, eagles, and ospreys was discussed. In this section we shall consider the population responses in the lower trophic levels (prey species) to variations in predator populations. The objective is to explore the overall impact on the system resulting from elimination or decline of certain species as a result of DDT.

The use of mathematical models to predict population responses is well established (*18, 21*). Lotka (*22*) and Volterra (*23*) formulated the first mathematical population model which assumes that population growth rate is proportional to population size. Predator-prey interactions, in which predator eats prey, are modeled by the product of the predator and prey populations (*23, 24*). The solutions for these models show that the prey populations react unstably to fluctuations in predator levels in a manner similar to some natural population fluctuations (*25*). It is the unstable behavior of these models, which does not correspond to the present behavior of the Wisconsin regional ecosystem, that has led to the development of the present population model.

To explore the possible effects of predator fluctuations in stable systems (*17, 18*) we considered a three-level model with a prey population n feeding on a food population f and being preyed upon by a predator population g. The system is assumed to be in equilibrium initially with population values N, F, and G. Deviations from equilibrium δf, δn, and δg, are assumed which are positive when a population exceeds its equilibrium value and negative when the population is less than that value. This approach is commonly used in control systems analysis (*17*).

For the food-prey interaction, the impact on population n of a change in food supply is a function of the difference between the food deviation and an equivalent population deviation, and is assumed to be proportional to the difference. Thus, in some period of time Δt, the change in population Δn can be expressed as

$$\Delta n = K_1 (\delta f - W_1 \delta n) \qquad (16)$$

where K_1 is a proportionality factor and W_1 is an equivalence factor.

Equation 16 states, for example, that if the food supply is great ($\delta f > 0$) at a time when the population is at its equilibrium value ($\delta n = 0$), the population will increase. Further, if decreased food ($\delta f < 0$) coincides with increased population ($\delta n > 0$), there would be a marked reduction in population.

The predator-prey interaction is developed similarly:

$$\Delta n = -K_2 (\delta g + W_2 \delta n) \qquad (17)$$

Equations 16 and 17 are combined to yield a defining equation for population n:

$$d(\delta n)/dt = -(K_1 W_1 + K_2 W_2)\delta n + K_1 \delta f - K_2 \delta g \qquad (18)$$

Equation 18 is inherently stable as a result of the negative coefficient of the δn term. The population will not "blow up" because of the increased possibility of its members either being eaten or starving when the population increases ($\delta n > 0$).

The three-level population model is

$$d(\delta f)/dt = -K_1 W_1 \delta f - K_1 \delta n$$
$$d(\delta n)/dt = K_2 \delta f - (K_2 W_2 + K_3 W_3)\delta n - K_3 \delta g$$
$$d(\delta g)/dt = K_4 \delta n - K_4 W_4 \delta g \qquad (19)$$

Equation 19 produces oscillatory responses that qualitatively represent actual population fluctuations (*18*). For example, an abrupt reduction of predator population leads to an increase of population n followed by a decrease of food population. Ultimately, the values return to their equilibrium values. The continual oscillations observed in natural population numbers may simply be the result of random changes in external factors such as weather.

The increase in prey population due to a decrease in predator numbers (for example, due to DDT) has the potential of becoming a public nuisance. In addition, the resulting decrease in prey food supply is potentially damaging in that a particular food species may be eliminated entirely. Alternate food sources (for example, crops) may be sought by the prey in order to nourish the increased population.

The explosive nature of populations deprived of their natural predators is well documented (*25*). The classical example is the removal of wolves, coyotes, and mountain lions from the Kaibab Plateau in Arizona, which resulted in an explosive increase in the mule deer populations until the deer decimated their own food supply. Starvation and disease resulted with a tremendous mule deer population crash (*26*).

We recognize that the population models considered above represent a somewhat oversimplified view of actual population response. Nevertheless, these models are sufficiently descriptive to yield the general nature of population response, and to make possible the prediction that a significant variation of a predator population would cause upsets throughout the entire system, some of which might be of sufficient magnitude to create "out-of-control" conditions.

Discussion and Conclusion

Some predictions of the consequence of adding DDT to the environment based on the DDT transport, accumulation, and concentration mechanisms and on the evidence of the impacts of DDT in ecosystems are now possible. DDT concentrates in the higher trophic levels, and, depending on unknown rates of metabolic breakdown, the concentrations in long-lived species in higher levels can be expected to continue to increase long after the addition of DDT to the environment has ceased.

The presence of DDT in any trophic level can have three major consequences:

1) The concentration may be high enough to kill the members in that level. If this occurs, the entire ecosystem will move toward a new equilibrium no longer influenced by the removed trophic level.

2) The concentration may not be lethal, but may cause adverse sublethal effects such as reproductive failure (*12*). The affected trophic level will disappear just as if the dosage were lethal.

3) The concentration may have no apparent effect on the trophic level. The DDT in this level will then pass on to the next higher trophic level, which will then be subject to the same three consequences of DDT concentration.

The analysis of DDT diffusion through the trophic structure of the ecosystem indicates that the equilibrium

concentrations will be a function of the "life spans" of the organisms in the system. Thus, the top carnivores, which play an important role in stabilizing the system, may take a long time to respond to the input of DDT. Since DDT is reducing predator numbers in present ecosystems, new population explosions may result.

The ecosystems making up the world biosphere might restabilize after the loss of a species, but with different population levels of the remaining species. Radical changes in population levels could have serious economic and public nuisance consequences. Further, the DDT once present in the obliterated populations will then be concentrated into fewer remaining species. Whether or not this process could be repeated in a series of systematic obliterations of the species in upper trophic levels with a consequent concentration of DDT into remaining species cannot be predicted at this time. However, with the models presented here, it can be predicted that the consequence of the present worldwide inputs of DDT in the environment will not become apparent for many years.

References and Notes

1. DDT, 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl) ethane; DDE, 1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethylene; DDD, 1,1-dichloro-2,2-bis-(*p*-chlorophenyl)ethane; dieldrin, 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-endo-exo-1, 4 : 5, 8-dimethanonaphthalene.
2. L. J. Carter, *Science* **163**, 548 (1969). On 22 May 1970, Chief Examiner Maurice Van Sustern of the Wisconsin Department of Natural Resources released his findings from the Wisconsin hearings. Van Sustern summarized the substantial evidence in a 25-page report and issued a one-page ruling which states in part: "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. . . . 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."
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Letters

Decay Time of DDT

In their article "Systems studies of DDT transport" (30 Oct., p. 503) Harrison *et al.* gave a fine description of a rather detailed mathematical model for predicting the ecological transport of DDT. However, due to lack of data, they make some assumptions on page 506 in order to arrive at estimates of the eventual decay time for DDT at various trophic levels. An important assumption in this analysis is that the organisms at each trophic level retain all DDT ingested. This assumption leads to rather long estimates of the decay times for organisms in the higher trophic levels.

We have arrived at a shorter estimate of the decay time, which we feel is somewhat more realistic, by using 1964 data on application rates of DDT and average levels of DDT in various foodstuffs. We have used the same type of mathematical formulation, that is, systems of simultaneous, ordinary differential equations) to develop a model for DDT transport. However, we have used existing data to develop our model rather than theoretically developing a model that requires data which do not exist. By assuming steady-state conditions (losses from the ecosystem balance the application rates) for 1964, our model shows that degradation of DDT in soil is the long-term factor controlling decay time. According to the data of Edwards and others (*1*), this decay time is on the order of 7 years, which corresponds to a 98.2 percent reduction of DDT (if all applications were halted) in about 27 years. On the basis of this steady-state assumption (which assumes ingested DDT is not indefinitely retained), all other decay times are faster and do not affect the long-term decay after approximately 5 years from the date of halting the DDT application.

The actual, long-term decay time for DDT may be somewhere between the 7 years we have estimated and the values of up to 100 years given by Harrison *et al.* Although metabolism of DDT is not extremely rapid, conversion of DDT into more polar metabolites and eventually into DDA, the excreted metabolite, does proceed in a number of species (*2*). Known intermediary metabolites of DDT such as DDMU [1-chloro-2,2-bis(*p*-chlorophenyl)ethylene], DDD, and DDE occur in all samples of environmentally acquired pesticide residues (*3*). Together these data support the hypothesis of an active metabolism and excretion of DDT in the environment by man, birds, insects, and fish (*2*). We would be more inclined to believe the 7-year decay time because it is based on actual data (although the interpretation may be questioned) and because the metabolism and excretion data conflict with the assumption which Harrison *et al.* make about complete retention of DDT.

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The approach we have taken in the development of our model has been based upon the physical principle of the "conservation of mass." The real importance of this type of approach is that the parameters significant to the system are decided by the theory associated with the principle. Models developed in this way can point to the absence of critical data and help to structure data acquisition programs.

Our "first-approximation" model (Eq. 13) assumed that DDT is a highly persistent substance and that it is neither metabolized nor excreted. However, an assumption that DDT is "lost" from any trophic level by *fixed* rates of metabolism or excretion would lead to a prediction of reduced equilibrium levels of DDT concentrations with no change in predicted response time. If any metabolism or excretion of DDT is functionally related to the concentration of the pesticide in a trophic level, then it is possible for the time constant, and thus the response time, of the system to be reduced.

There appears to be some misunderstanding as to our use of time constants for predictive purposes. We did not seek to estimate decay times for DDT at various trophic levels but, rather, the times required for a complete response (attainment of new equilibrium levels) to sustained inputs of DDT. In essence, we predicted that if all applications of DDT were to cease, concentrations of DDT in the higher trophic levels would continue to rise for some time due to the dynamics of the system.

It is true that different assumptions lead to different predictions and, as time passes, we would expect new information to become available which would permit more precise predictions of DDT flows and storages in ecosystems. However, with regard to assumptions, we would be interested in learning the reasons for Bloom's and Menzel's assumption that, in 1964, the losses of DDT from the ecosystem balanced the application rates.

For the benefit of those who may wish to make use of our equations, we would like to point out two errors in the equations as they were printed. In the first term of Eq. 9, $\dot{m}_i$ should be m_i. In Eqs. 13, 14, and 15, $\dot{m}_{i-1}$ should properly be $\dot{m}_{i-1,i}$.

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