Pollutants and food-chains

On careful scrutiny, the belief that organochlorine insecticides and other pollutants concentrate along food chains proves to be suspect. The amounts of such chemicals retained by different organisms probably depends more on differing rates of metabolism and excretion

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Pollutants will, if present in sufficient quantity, have adverse effects on animals and plants. Many of these compounds do, of course, break down so rapidly that there is little risk of toxic concentrations. But compounds such as the organochlorine insecticides and polychlorinated biphenyls (PCBs) can persist long enough for living organisms to acquire considerable concentrations within their bodies. It is commonly believed that such persistent substances accumulate or concentrate as they pass along or up the food-chain. This idea of concentration developed from field observations on the occurrence of organochlorine insecticides in wildlife specimens, and most of our evidence still relates to these insecticides. There is, however, little good evidence that pollutants in general, or organochlorine insecticides in particular, do concentrate along the food-chain.

It is common knowledge that animals are selective in what they eat—one man's meat is another man's poison. The energy that animals obtain from their food is derived ultimately from the sun's radiant energy, some of which is utilised by green plants for photosynthesis. The first link in the food-chain, or food-web as it is now usually thought of, is the herbivores. These may be preyed on by carnivores, which may themselves be eaten by other predators. It is then easy to assume that persistent compounds will accumulate along the food-chain. If 10 herbivores all have, and retain, equal amounts of a persistent substance, then the carnivore that eats these 10 herbivores will contain 10 times as much of this substance as did any one of the herbivores.

But this is not a real situation, and the confusion comes possibly from the unappreciated difference between persistence in the physical environment and persistence within organisms. Organochlorine insecticides last a long time in soils—given suitable conditions 50 per cent may remain for well over 10 years. And elemental pollutants such as the heavy metals obviously persist indefinitely, either in the elemental form or in a variety of compounds. But we do not yet know of any pollutants that remain indefinitely within a living organism.

In general terms, there are two processes by which animals can remove foreign substances from their bodies: metabolism and excretion. Metabolism, or chemical change, can by itself remove some compounds from the body. It is sometimes a necessary preliminary too before a substance can be excreted. This is particularly true of [illegible] as the organochlorine insecticides, that are [illegible] in water. They are often first [illegible] water-soluble compounds, [illegible]. The amount remaining the residue left unaffected by metabolism and excretion.

Considerable studies have been made of the quantitative relationships between rate of intake and rate of loss of drugs, and we can probably use the same mathematical models for studies on the kinetics of pollutants. Such analyses have been made for organochlorine insecticides. It is appropriate to use the compartmental model—so called because the body is considered as a series of compartments, each defined as a quantity of insecticide with uniform and distinguishable kinetics of metabolism and of transport to the other compartments. In biological terms these compartments usually correspond to specific tissues or organs. The data available are mostly for mammals, but the little information we have for other groups, principally birds and fish, suggests that they are similar.

These quantitative relationships can usefully be summarised in four simple statements. They are illustrated in Figure 1, but need some qualifications:

1 The concentration of insecticide in a tissue depends on the rate of intake. The higher the rate of intake, the higher the concentration in the body.

2 Eventually, if the rate of intake is constant, and continues for long enough, the concentration of insecticide in a tissue reaches a plateau level. Sometimes, months after a plateau concentration has been reached with constant rates of insecticide intake, the tissue concentrations may change again, either up or down. For example, research workers at Shell's Sittingbourne laboratories fed beagle hounds for two years with a constant daily dose of dieldrin. The dieldrin reached a steady concentration in the blood within about three months, but after a further 17 months or so the concentrations rose again, about two-fold, and had not stopped rising by the end of the experiment. There was no indication at any time during the experiment that the dieldrin was affecting the dogs, and we do not know the reason for this increase.

During such long-term experiments the experimental animals are inevitably ageing. The changes associated with age are little understood, and these may provide a sufficient explanation for deviations from a plateau level. Moreover, we do know that organochlorine insecticides often induce animals to produce more of the enzymes which metabolise these insecticides. Changes in plateau level could be associated with changes in the amount and/or activity of these enzymes. In [illegible] to the laboratory, environmental conditions will be much more variable, and often more adverse, and may well disturb these relatively simple relationships. Certainly we now know [illegible] insecticide, after

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a plateau level has been reached for the first, can alter plateau concentration severalfold.

3 One can deduce from the above two relationships that the concentrations in different tissues should be correlated. They are. In general terms, adipose (fat) tissue usually contains by far the highest concentrations. This is presumably related to the fat-soluble nature of the organochlorine insecticides.

4 The existence of a plateau level implies that the animal is excreting and/or metabolising the absorbed insecticide at the same rate as it is being absorbed. In other words, the concentrations of insecticide in the various compartments are in a steady state. One would expect, therefore, that if the rate of exposure to insecticide decreases or ends, the tissue concentrations will drop. They do. Sometimes when exposure ends there is a simple exponential decline. But sometimes, in some tissues, the rate of decline is more rapid at first than later on. These results appear to be consistent with a model in which insecticide can be excreted directly from the blood, whereas insecticide in other tissues has to pass into the blood first.

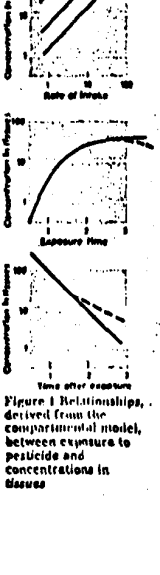


Figure 1 Relationships, derived from the compartmental model, between exposure to pesticide and concentrations in tissues

Concentration effect

It is an indisputable fact that living animals usually have far higher concentrations of organochlorine insecticides in their bodies than occur in their physical environment. Animals can, of course, concentrate many other substances besides synthetic compounds. The affinity of iodine for the thyroid gland is a homely example. But what relevance has an animal's position in the food-chain for the amounts of pollutants it may contain?

Obviously, as we have just seen (Figure 1), the amount in the body depends on the exposure. And pesticide can be acquired either directly from the physical environment, or indirectly from the food. This question arose when E. G. Hunt and A. I. Bischoff investigated the deaths of large numbers of the western grebe (Aechmophorus occidentalis) at Clear Lake, in California, US. They concluded that the birds had died from DDD poisoning. DDD is an insecticide, chemically similar to DDT, which had been used to control a gnat that bred abundantly there. Post-mortem analyses showed that fatty tissues of the grebe contained 1600 ppm of DDD. Subsequent analyses of various organisms during the year after the last application of DDD showed that some of the fish, which were apparently not killed by the insecticide, had even higher concentrations in their visceral fat than the grebes had.

Hunt and Bischoff suggested that the grebes had acquired their insecticide from the fish they ate, and that some of the fish species were more resistant than the grebes to the insecticide. These suggestions appear reasonable, but they went on to two further conclusions which were not so well supported. They said firstly that carnivorous fish accumulated more DDD than did the plankton-eating fish. In fact, Hunt and Bischoff's data showed that the concentration of DDD was higher in older fish. When this factor is allowed for, the data suggest that the carnivorous fish had higher concentrations in their tissues because their tissues had a higher fat content. They then said that this difference between types of fish was correlated with their positions in the food-chain.

Rachel Carson, in her well-known book Silent Spring, then confused the issue by stating that plankton in Clear Lake contained 5 ppm of DDD, which was about 25 times the maximum concentration ever reached in the water itself. This concentration factor is calculated incorrectly (it should be 250 times) and is based on the doubtful supposition that the DDD was dispersed uniformly throughout the water of the lake. More important, Hunt and Bischoff stated explicitly in their paper that, due to the size of their plankton sample, they had been unable to detect any DDD in the plankton. (Dr Bischoff tells me that they found 10·9 ppm in a plankton sample taken a year later.) Unfortunately, Rachel Carson's version is widely accepted, even by some who are professionally involved. Carson then inferred that animals contain residues at higher concentrations than their prey species, and obtain these higher concentrations by storing the accumulations from their prey.

It is important, when considering these ideas, to distinguish clearly between aquatic and terrestrial animals. There have been two very clear experiments, for two species of fish, which show that fish acquire their insecticide directly from the water, not from their food. In one of these, G. G. Chadwick and R. W. Brocksen kept fish (Cottus perplexus) in tanks with $0{\cdot}5\times10^{-3}$ppm of HEOD (the active principle of dieldrin) for up to three weeks. They fed the fish on tubificid worms, some of which they exposed to the same concentration of dieldrin as the fish, whilst the rest of the worms were kept in uncontaminated water. Whole fish were analysed at intervals. Those fed on control worms reached a plateau concentration of about eight ppm within the first two weeks. Fish fed on contaminated worms had similar concentrations of HEOD to those fed on control worms. The total amount of HEOD contained in the contaminated worms fed to a fish for three weeks was only about 16 per cent of the total amount of HEOD found in these fish.

Obviously, the HEOD in the water was of much greater significance for the fish than that in their food. Fish in general do appear to acquire higher concentrations of organochlorine insecticides from the water than do invertebrates. Conceivably this is related to a higher rate of metabolism, with the consequent passage of much water through the gills for respiration, thus increasing the animals' effective exposure to insecticide. The available data suggest that for mercury compounds too, position in the food chain is irrelevant to the amounts acquired.

The situation is less clear cut for terrestrial animals. Presumably, predators do absorb most of their insecticide from food. Analyses of samples of breast muscle from British birds have shown that predators have higher concentrations than herbivores, and that insectivorous birds have intermediate con-

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centrations. Two experimental studies have been made. In both, earthworms were fed to birds. Dr W. H. Stickel, D. W. Hayne and L. F. Stickel fed worms which had been exposed to heptachlor, to American woodcock (Philohela minor) for 60 days. By that time the birds contained, on a dry-weight basis, three to four times as high a concentration of insecticide as the worms, and had retained about 18 per cent of the ingested insecticide.

Dr D. J. Jefferies and B. N. K. Davis fed four song thrushes (Turdus ericetorum) on worms exposed to dieldrin for 42 days. The birds were fed contaminated worms at different rates, but all had lower concentrations of dieldrin in their tissues by the end of the experiment than had the worms. This experiment showed clearly the importance of metabolism and excretion—only 0·8 to 4·4 per cent of the ingested insecticide was retained. We do not know whether the birds had reached a plateau concentration, but either way, the rate of elimination is important, and variable. The per cent retained varied by more than 20 fold.

So we are left with a contradiction between surveys of wildlife specimens, which often suggest that terrestrial predators contain higher concentrations of insecticide than their prey, and laboratory experiments, which suggest that all animals have a much higher affinity for organochlorine insecticides than does the physical environment, and that differences between species depend largely on specific differences in the rates of excretion and metabolism, which are presumably not related to position in the food chain.

Whether predators in the field have a greater exposure to pesticide than their prey species is a moot point that is difficult to answer. It is obvious that terrestrial predators do not retain all, or even most, of their ingested insecticide, but we do assume that the amounts in animals are indicative of their exposure. In one sense this must be true, but we do not know whether exposure is continuous or intermittent. There is some evidence to show that it can be intermittent. For example, the number of breeding pairs of the peregrine falcon (Falco peregrinus) in Great Britain declined 10 to 15 years ago. This is thought to have been caused by falcons eating a few pigeons immediately after these birds had acquired a heavy dose of dieldrin by eating dressed grain. The concentration of dieldrin in a random sample of pigeons would be lower than that in pigeons analysed after they had eaten dressed grain. It is often difficult to know how much insecticide or other pollutant a species is exposed to.

A second implicit assumption is that plateau concentrations have been reached. Possibly they have not. Predators tend to be long-lived, and might therefore reach higher concentrations.

These are speculations, and must be so until we have clear data. But it does look as though an animal's position in the food chain is less important than the rate at which it can eliminate the pollutant from its body.

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