# CHAPTER 8

# Terrestrial Animals*

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## 8.1. INTRODUCTION

Pollutants matter because of their biological effects, and the type and magnitude of these effects depend on the degree of exposure that individual organisms receive. Terrestrial animals will be used to illustrate four related themes: the intake, distribution, and loss of pollutants by organisms; the causes of death; the effects of sublethal exposures; and the effects on populations. Most of the examples come from insects, birds, and mammals, and refer to the effects of insecticides, polychlorinated biphenyls (PCBs) and metals, but the ideas that these examples illustrate should apply to a much wider range of habitats, organisms, and pollutants. Some aspects of pollutants in terrestrial ecosystems, especially the effects of organochlorines on avian reproduction, have recently been reviewed by Stickel (1975).

## 8.2. AMOUNTS OF POLLUTANTS WITHIN ORGANISMS

There is an enormous amount of data published on results from chemical analyses of plants and animals, but much is of doubtful scientific value. Although the details of chemical analysis for pollutants can be complicated and difficult, it is too easy to collect many biological specimens and have them analysed for a range of pollutants. The precise questions to be answered need to be clearly formulated at the beginning (Holden, 1975).

Some pollutants, such as ozone, are so transitory that it is not possible to detect them in animal tissues — one can only detect the effects. However, for pollutants that are more persistent it is useful to know the amounts that are present within animals: it gives some measure of past exposure, and it can indicate the likelihood of biological effects.

The compartmental model (Atkins, 1969), developed extensively in physiology and pharmacology, appears to be the most useful approach developed so far for the quantitative study of pollutants within organisms (Moriarty, 1975a). A compartment is defined as a quantity of pollutant that has uniform kinetics of transformation and transport, and whose kinetics are different from those of all other compartments. A whole animal is envisaged as consisting entirely of compartments, which are linked in a mamillary system: the peripheral compartments are all linked to the central compartment, but not with each other (Figure 8.1).

The central compartment, compartment 1, always represents the blood, haemolymph, extracellular fluid or transport system. This is the only compartment that receives pollutant from, or returns it to, the exterior. This makes reasonable physiological sense. A fish, for example, is likely to take up (indicated by $R$) most of any pollutant into its bloodstream either through its gills or from food in its gut. Excretion, via the kidney (indicated by $k_{01}$), occurs from the blood. Compartment 2 might represent the liver, the principal site of metabolism. Other tissues of the body may be represented by additional compartments.

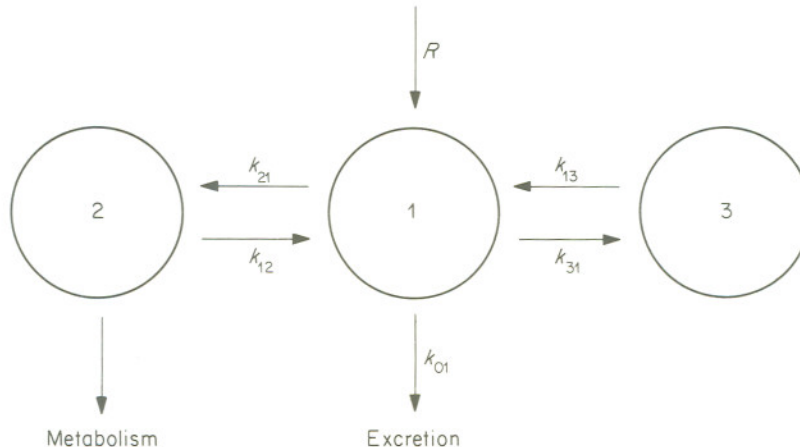The model assumes that the rate at which pollutant leaves any compartment is



Figure 8.1    A three-compartment model for the distribution of a pollutant within a vertebrate. Pollutant is absorbed into the blood (compartment 1) at a rate $R$, most metabolism occurs in the liver (compartment 2), and rates of transfer between compartments are indicated by the rate constants ($k$)

directly proportional to the amount of pollutant in that compartment. The rate constants (fraction of pollutant transferred per unit of time) are indicated by $k$, with suitable subscripts.

In theory, different cell constituents should probably be represented by distinct compartments. In most cases two or three compartments have sufficed to describe the distribution of pollutants within entire organisms. There are at least two reasons for this:

(1) Analytical results usually refer to, at least, whole tissues, so that smaller compartments become meaningless.

(2) Even if there is reason to suppose that there are many compartments and there are analytical data with which to test this supposition, in practice the residual variation in even very good data makes it difficult to estimate parameters on retention or excretion for a model with more than two or three compartments.

In spite of these severe practical limitations, the compartmental model has proved a very useful way of describing experimental results. Equations derived from these models consist of a series of exponential terms. Figure 8.2 shows as an example the loss of dieldrin from blood (after cessation of uptake), which has been described by a two-term exponential equation. The two exponential constants (0.535 and 0.0529) are derived, theoretically, from the three rate constants for the two compartments. In practice of course they are estimated directly by fitting an equation to the data points with a least squares procedure. In practical terms these data show that, for the first 71 days after exposure, the dieldrin in rats' blood could be considered in two parts. That part that is lost more rapidly has a half-life of 1.3 days ($\log_e 2/0.535$) while the part lost more slowly has a half-life of 13.1 days. The exponential coefficients indicate that, when exposure stopped, the rapid component was almost twice as large as the slow component (542 : 298, or about 9 : 5). The higher this ratio, the more rapidly most of the HEOD disappears. It should be noted that, unless the simplest one-compartment model is used, it is meaningless to talk of a pollutant's half-life in organisms – the proportion lost from the organism in unit time is not constant.

Similar equations can be derived for the amounts of pollutant found within a compartment during chronic exposure (Figure 8.3). This example shows clearly that the equation for a one-compartment model gives a poor fit to the data for amounts of dieldrin in the blood, whereas a two-compartment model gives a good fit. The compartment model assumes that the rate constants for transfer of pollutant between compartments have the same values during and after exposure. There is at present little experimental evidence either way on this point.

This model implies that, if exposure continues for long enough, then a steady state will be reached, in which the amounts of pollutant within each compartment
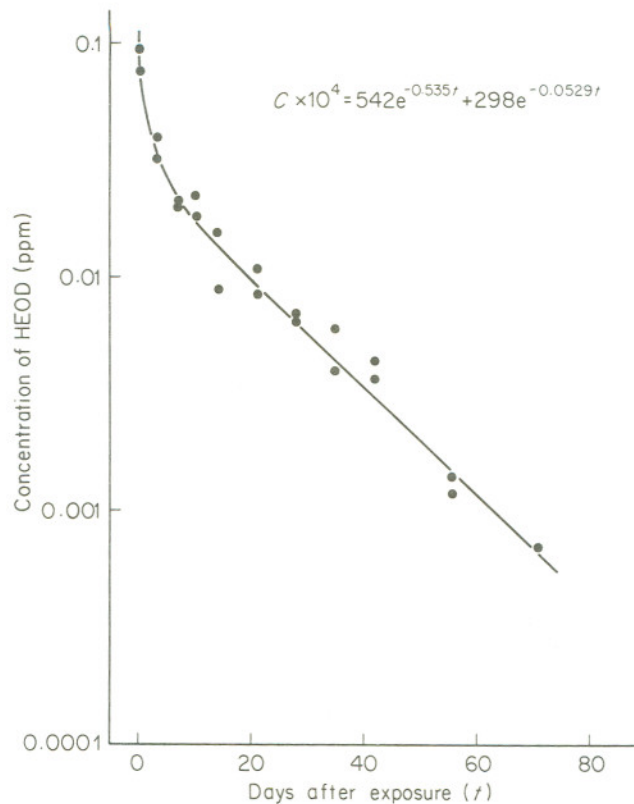
The plotted equation reads:

$$C \times 10^4 = 542e^{-0.535t} + 298e^{-0.0529t}$$

with the y-axis labelled "Concentration of HEOD (ppm)" (values 0.0001, 0.001, 0.01, 0.1) and the x-axis labelled "Days after exposure (*t*)".

Figure 8.2    Decrease in the concentration ($C$) of dieldrin in rats' blood during the first 71 days after exposure. Data fitted to an equation with two exponential terms (data and equation from Robinson *et al.*, 1969). (Reproduced by permission of Academic Press, London, from Moriarty, 1975a)

stay constant. At this stage the rate of intake equals the rate of loss. There is considerable evidence to support this. In theory, the amount of pollutant in a compartment, once the steady state has been reached, should be proportional to the degree of exposure and inversely proportional to the rate of loss from the compartment. This does seem to be true as a first approximation (see Figure 8.4). This is potentially of great value for any monitoring system, but several points should be noted:

(1) In some of the few experiments where exposure has continued for long enough, the steady state, apparently reached, does not continue and the amounts in the compartment have increased again (see Figure 8.5). This
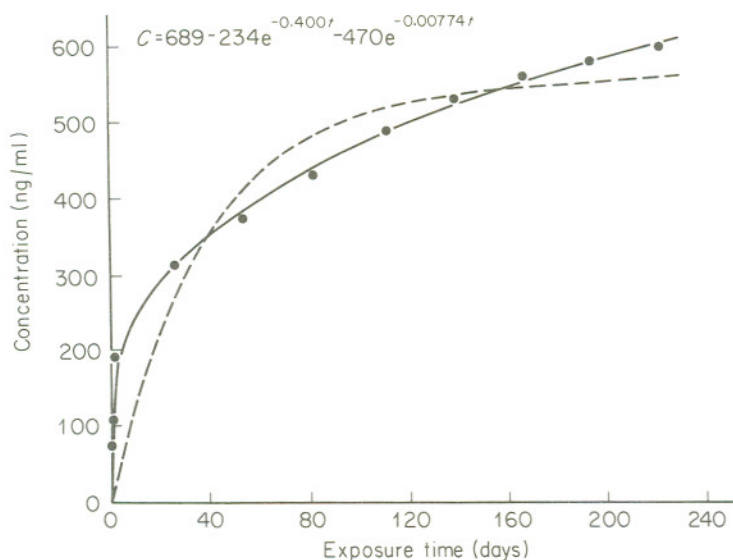
Figure 8.3    Increase in the concentration ($C$) of dieldrin in sheeps' blood while ingesting 2 mg dieldrin/kg body weight/day. ———, line derived from equation with two exponential terms; ———, line derived from equation with one exponential term (data from Davison, 1970). (Reproduced by permission of Academic Press, London, from Moriarty, 1975a)

may result from some change in metabolism possibly due to the presence of the dieldrin or to some assumption in the model which is false, but at present we have no information on possible explanations.

(2) Specimens from the field will not have experienced a constant or standard physical environment, nor, in many instances, will exposure have been constant. Many physiological events, such as breeding activity (Anderson and Hickey, 1976) and hibernation (Jefferies, 1972), quite apart from catastrophes and inclement conditions (Clark, 1975), may well affect pollutant levels.

The crux of the compartmental model is the use of rate constants to quantify the movement of pollutants into, within, and out of an organism. Future work will doubtless modify this model, but it seems most unlikely that this central feature can be discarded.

Another discussion of retention equations and the changes in body and organ content with time is to be found in Chapter 5.

The application of this model to calculation of the amounts of a persistent pollutant in each level of a food chain, provides an important advance over earlier
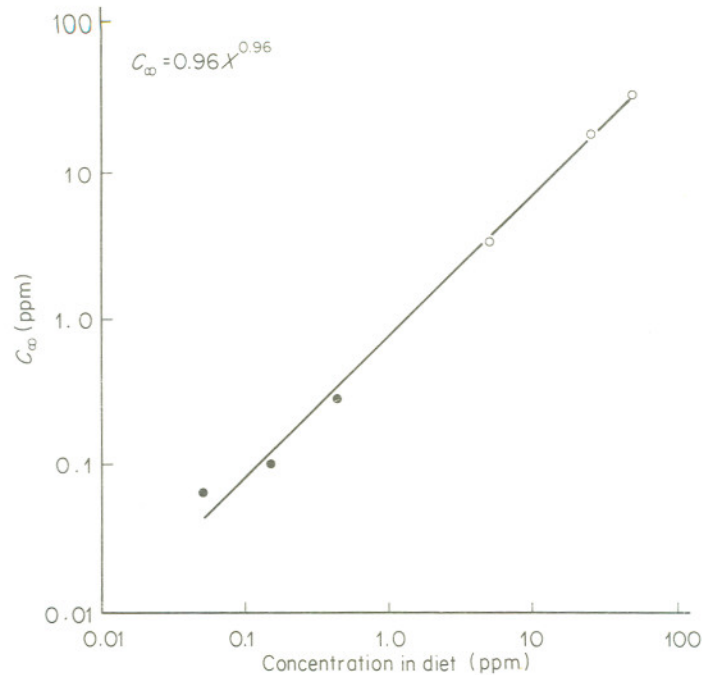
Figure 8.4    Linear regression for the steady-state concentration
($C_\infty$) of $p,p'$-DDT in eggs of white leghorn hens on the
concentration of $p,p'$-DDT in the diet ($X$). Both scales are
logarithmic. ●, values calculated from data of Cummings *et al.*
(1966); ○, values calculated from data of Cecil *et al.* (1972).
(Reproduced by permission of Academic Press, London, from
Moriarty, 1975a)

concepts. One of these, that persistent pesticides accumulate and concentrate along
food chains, is too simple and does not take account of the physiological processes
operating at each trophic level. The compartmental model approach is clearly to be
preferred (Moriarty a, in press) over that of Hamelink *et al.* (1971) and Neely *et al.*
(1974) who have proposed that for pesticides in aquatic habitats, partition
coefficients will indicate the amounts to be found in animals. The uptake and
retention of molecules depends not only on passive diffusion but also on processes
of active transport, metabolism, and excretion, which vary with different species
(Walker, 1975).

The great potential value of the compartmental model is that, by estimating rate
constants or parameters derived from them, it should enable us to measure the
differences between species in the way they take in, accumulate, distribute, and get
rid of pollutants. It should also help us assess the more intractable problems of
interactions between pollutants and the effects of other variables. There are reviews
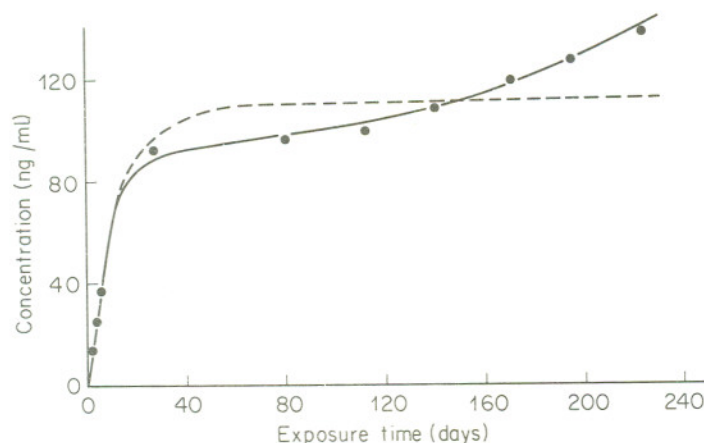
Figure 8.5   Changes in the concentration of dieldrin in sheeps'
blood while ingesting 0.5 mg dieldrin/kg body weight/day,
————, line derived from equation with two exponential terms;
— — — —, line derived from equation with one exponential term
(data from Davison, 1970). (Reproduced by permission of
Academic Press, London, from Moriarty, 1975a)

of the available data for metals (Task Group, 1973) and for organochlorine
insecticides (Moriarty, 1975a).

## (i) Absorption

Intake from the physical environment deserves special mention. Intake, be it
through the epidermis, respiratory organs, or gut, depends very much on the precise
form of the pollutant — both its chemical 'species' and the degree and type of
physical aggregation. Much of our information relates to man.

Aerial pollutants can occur either as gases or associated with particulate matter.
The ease with which gases reach the lungs' alveoli depends greatly on their water
solubility: those that are highly water-soluble will dissolve readily in the mucous
membranes before the alveoli are reached. For those gases which do reach the
alveoli, lipid solubility probably aids penetration through the membrane into the
lung tissue and thence into the blood. For example, mercury vapour is non-polar, is
not dissolved in the nasopharyngeal or tracheobronchial tracts of man, but about
80% of that inhaled is absorbed through the alveoli (Kudsk, 1965).

Deposition and retention of aerial pollutants from particulate matter depend
greatly on the size of the particles. Particles may settle onto respiratory surfaces by
any of three distinct processes:

(1) Impaction, the most important process for particles larger than a few
microns, occurs where the airstream changes course abruptly.

(2) Settlement by gravitational forces, which of course depends on particle mass and shape. For particles of about unit density, this process is important for particles whose diameter is within the range of about 0.5–5.0 microns.

(3) Diffusion, which is important for the smaller particles.

The lung model for Reference Man described in Chapter 5 shows that inhaled particles may be deposited in the nasopharyngeal, the tracheobronchial, or the alveolar regions of the lung. In each of these regions the pollutant may be either absorbed into the extracellular fluids or transported up to the pharynx in mucus propelled by ciliary action. After reaching the pharynx the material is swallowed into the digestive tract. Material taken up from the alveoli (the most important site of absorption) may go to the bloodstream or be retained in the broncho-pulmonary lymph nodes.

Ingestion of pollutants can obviously occur with food and drink, but inhaled particulate matter may also be transferred to the gut in mucus. In qualitative terms, several pathways exist. Some may pass straight through the gut, unaltered and unabsorbed, some may be metabolized by the gut microflora, while some may enter the gut wall, by endocytosis or in a chemical form suitable for penetration of the gut wall. Absorption through the gut wall does not necessarily indicate transfer to the whole organism. Dichlorobiphenyl for example may pass to the liver, and then be excreted in the bile back into the gut (Iatrapoulos *et al.*, 1975).

## 8.3. TOXICITY

Pollutants matter because of their many possible effects on individual organisms (see Chapter 7). The most dramatic effect is of course death. Death is the end result of many disrupted bodily functions, but these are usually secondary effects from a single initial biochemical change. This 'site of action' may be dispersed throughout the body, and it can be important, if one wishes to relate amount of pollutant to degree of effect, to have precise knowledge of the site of action. There are two reasons for this.

Firstly, unless the critical organ is known it may prove difficult, or impossible, to relate degree and type of exposure, and amount of pollutant within the body, to the likelihood of adverse biological effects. For example, different compartments can approach their steady-state concentrations at very different speeds, so that the amounts of pollutant in one tissue or organ may give very little indication of the amount in another (Table 8.1).

Secondly, if an enzyme, or group of enzymes, that is widespread throughout the body is inhibited by a pollutant, results may appear contradictory unless the critical organ is known. The organophosphorus insecticides provide a good example. They have been known for a long time to inhibit esterases, and acute toxicity is due to acetylcholinesterase inhibition. It proved difficult to correlate degree of acetyl-

Table 8.1 Amounts of Dieldrin in the Blood, Liver, and Fat of Rats Fed on a Diet containing 50 ppm Dieldrin. Results for liver and fat also expressed as a ratio of that found in the blood (data from Deichmann *et al.*, 1968)

| Days fed | Concentration of dieldrin (ppm) | | | Ratio of concentration to that in blood | |
|---|---|---|---|---|---|
| | blood | liver | fat | liver | fat |
| 0 | 0.001 | 0.00 | 0.00 | | |
| 1 | 0.047 | 1.01 | 6.42 | 25 | 160 |
| 2 | 0.080 | 1.97 | 9.74 | 25 | 122 |
| 4 | 0.121 | 2.63 | 25.00 | 22 | 208 |
| 9 | 0.243 | 6.22 | 98.95 | 26 | 412 |
| 16 | 0.261 | 9.56 | 174.19 | 36 | 669 |
| 31 | 0.237 | 12.70 | 280.25 | 53 | 1182 |
| 45 | 0.255 | 8.55 | 196.10 | 33 | 754 |
| 60 | 0.262 | 7.44 | 148.22 | 29 | 570 |
| 95 | 0.193 | 4.77 | 130.77 | 25 | 688 |
| 183 | 0.268 | 8.15 | 184.75 | 30 | 684 |

cholinesterase (AChE) inhibition with the onset of death (e.g. Burt *et al.*, 1966), but the discrepancies appear to have been resolved now that it has been discovered that there are several isozymes of AChE (Tripathi and O'Brien, 1973). The housefly contains four isozymes of AChE in the head, and another three different isozymes in the thorax. Groups of houseflies were given the $LD_{50}$ dose of four insecticides, and the minimal activity estimated for each of the seven isozymes (Table 8.2). It can be seen that isozyme 7 always loses virtually all of its activity, even in the survivors, so it cannot be the relevant site of action. Most of the other isozymes are inhibited to different degrees by the different insecticides, but isozyme 5, in the thorax, has a constant degree of inhibition, of just over 80%. These data suggest that this isozyme is the critical site of action.

Care is needed when applying the compartmental model to analytical results from an animal that has received enough pollutant to seriously impair its normal functions. At the present stage of the model's development it is usually assumed that the animal is in a state of equilibrium. However, an animal near to a lethal exposure will often depart from such a state. Thus it has been found many times that animals killed by insecticides have reduced fat reserves. The highest concentrations of organochlorine insecticides occur in fatty tissues, so that if these fat reserves start to be mobilized, the concentrations of insecticide in all tissues are likely to increase suddenly. This will include the critical organ, the brain. It follows that if we know that 10 ppm of an insecticide in the brain indicates that the animal is likely to, or has, died from that insecticide, and a wild population has say, a steady-state concentration of 2 ppm of insecticide in the brain, we cannot deduce

Table 8.2   The Minimal Percentage Activity of Seven Acetylcholinesterase Isozymes in the Housefly after Exposure to the $LD_{50}$ Dose of Four Insecticides. (Reproduced by permission of Academic Press, New York from Tripathi and O'Brien, 1973)

| Body region | Isozyme | Activities following treatment with | | | | Range of minimal activities |
|---|---|---|---|---|---|---|
| | | malaoxon | paraoxon | diazinon | dichlorvos | |
| Head | 1 | 18 | 45 | 54 | 39 | 36 |
| Head | 2 | 42 | 53 | 28 | 72 | 44 |
| Head | 3 | 51 | 78 | 32 | 57 | 46 |
| Head | 4 | 28 | 67 | 67 | 70 | 42 |
| Thorax | 5 | 15 | 18 | 22 | 21 | 7 |
| Thorax | 6 | 5 | 38 | 1 | 16 | 37 |
| Thorax | 7 | 1 | 1 | 1 | 1 | 0.0 |

that the population's exposure is one-fifth of that needed to kill it. It may in fact be very near the critical level at which fat will be mobilized, brain levels rise, and the animals die (Jefferies and Davis, 1968).

It is important to distinguish between pollutants whose toxicity is intrinsic to one of the elements contained within the molecule, and those whose toxicity resides in the precise molecular configuration. Mercury, for example, has many inorganic and organic chemical 'species', some of which are much more toxic than others, but the toxicity depends in all instances on the presence of the mercury atom, which is, for practical purposes, indestructible. $p, p'$-DDT is also regarded as a persistent pollutant, but its toxicity resides in the whole molecule, which can in time break down into non-toxic components. The critical organ can change according to chemical 'species' for the elemental pollutants. With mercury, methylmercury affects the central nervous system, several other forms of mercury affect the kidney, while the vapour of elemental mercury affects both these and the peripheral nervous system.

The classical measure of a pollutant's toxicity is the $LD_{50}$, but this is of limited value. Strictly, this is the result of a bioassay, and although the results are reproducible, they have limited relevance to the problems of pollution. Other criteria, such as changes of behaviour or enzyme activity can be, and often are, used but these may more conveniently be considered as sublethal effects.

## 8.4. SUBLETHAL EFFECTS

Numerous types of sublethal effects have been described, and those for pesticides have been reviewed several times. Moriarty (1969) has reviewed them for insects, while Jefferies (1975) has made the most recent review for vertebrates. A few general points merit attention here.

Death is obvious. Sublethal effects may be very subtle and not at all obvious. So we are often faced with the question: have we missed observing a subtle sublethal effect? There is probably no complete answer to this question, because it is usually difficult to prove a negative. The usual, empirical, approach is to look for effects on functions, such as growth and reproduction. The alternative is to look for effects on specific systems, such as enzyme activity. This is most useful when some clues exist about the pollutant's mode of action. Then tests are sometimes done *in vitro*, rather than *in vivo*, but extrapolation of results to the whole organism may be difficult. For example, inhibition of carbonic anhydrase has been suggested as the cause of thin eggshells in birds exposed to DDT. *In vitro* tests have demonstrated inhibition of carbonic anhydrase activity by DDT, but this appears to be an artefact, caused by precipitation of DDT occluding the enzyme from solution (Pocker *et al.*, 1971).

With both approaches there is a need to distinguish between a deleterious effect and an adaptive response. Animals do respond to their environment — they would not survive if they didn't, and it can be difficult to decide when an effect has become deleterious. Enzyme induction for instance is an adaptive response, and

occurs in response to a wide range of liposoluble foreign compounds, including organochlorine insecticides. It can, however, have unfortunate consequences. Wergedal and Harper (1964) fed one group of rats on a high protein diet, and another group on a low protein diet. These diets were given for from five to twenty days, after which the rats were starved for twelve hours. Each rat was then injected with glycine. All the rats on the high protein diet died from ammonia poisoning. Their diet had induced the formation of enzymes that enabled them to metabolize amino acids more rapidly. Ammonia is one of these metabolites, which is normally metabolized to urea before excretion. The high protein diet meant that the injected glycine was metabolized more rapidly, and the twelve hours starvation meant that there was a shortage of the intermediate compounds needed to convert ammonia to the less toxic urea. By contrast, half the rats fed a low protein diet survived the injection of glycine.

This was a rather unnatural situation, but does illustrate an important point. An adaptive response does not necessarily increase the individual's chances of survival, it just increases the probability of survival in normal environmental conditions. That is an ecological question that can be difficult to answer.

One of the greatest deficiencies in current toxicity testing is the paucity of information on the effects of very long low-level exposures. Long-term experiments are of course routine in testing for carcinogenic and mutagenic action on mammals, but not for other sublethal effects. To put it in its simplest form, very rarely do we try to determine how far below a lethal exposure will still produce deleterious effects.

## 8.5. EFFECTS ON POPULATIONS

Apart from our own species and some domesticated species, we are not usually concerned with the effects of pollutants on individual organisms. Rather we are concerned with the effects on populations of individuals. This introduces an extra order of complexity: we cannot automatically assume that because, in the field, a pollutant kills some individuals from a population, therefore the population will become smaller. Most of our knowledge on this topic relates to the effects of insecticides on insects, where resurgence is well known (Dempster, 1975). Other possible results include increases in the populations of other species, replacement of the affected species by another, and resistance.

Sublethal effects also can affect population size, by their effects on either survival ability or reproductive ability. No field studies have been made so far in which the separate quantitative consequences of the individual lethal and sublethal effects of a pollutant have been estimated. It is therefore difficult to estimate the significance of sublethal effects alone, distinct from lethal effects, for population dynamics.

There have been many studies of the ecological effects of pollutants, especially the unexpected consequences of pesticide usage, on wildlife (three examples taken

more or less at random: Grolleau and Giban, 1966; Borg *et al.*, 1970; Craig and Rudd, 1974). However, study of effects on populations is difficult. The communities within which populations exist are usually too complex to permit sufficient resources for a strictly experimental approach, with control and experimental areas and adequate replication – the work on *Tribolium* species is a notable, but special, exception (Park, 1962). Because of the difficulties in understanding these complex situations, conclusions have usually to rely heavily on experience and judgment, and the most effective way to illustrate the difficulties of interpretation is to consider a specific example in some detail.

### (i) Pesticides and the Peregrine Falcon (*Falco peregrinus*)

The peregrine falcon, widespread throughout Eurasia and North America, feeds almost entirely on live birds, caught in flight. It is the largest native falcon in Great Britain, where it is one of the few wildlife species for which extensive and acceptable data on past population size exist. The total number of breeding pairs in Great Britain appears to have been remarkably stable over the last four centuries. This species is at the end of a food chain, and the number of breeding pairs appears to be limited in part at least by the occurrence of suitable breeding sites – the nest or eyrie is usually on a steep rock face, often in the same spot year after year. Ratcliffe (1970) discovered that during the years 1947 and 1948 an index of eggshell thickness (eggshell weight/egg length x egg breadth) decreased significantly by, on average, 19.1% (Figure 8.6). Before 1947 there were no geographical variations in this index, but after that date eggshells from the eastern and central Scottish Highlands decreased in thickness by only 4.4%. There has been much controversy about the causes and significance of this phenomenon (Moriarty, 1975b). The fundamental problem is that the field data are essentially of correlations between different measurements, and to deduce cause and effect from correlations is always difficult. However, it does seem highly probable that DDT, or its metabolite DDE, or both, initiated the thinning of eggshells. More recently, other organochlorine insecticides, and other pollutants too, may have been partly responsible. Whether that diagnosis be right or wrong, a marked and widespread change occurred among the individuals in a field population, for which a reasoned argument can be advanced to suggest pollution as the cause, but it has been impossible to prove beyond all doubt that insecticides were the cause.

One important piece of evidence would be an experiment in which, when a pollutant is administered:

(1) amounts in the eggs are comparable to those found in field specimens;

(2) eggshells are comparably thinner.

The results of many experiments have been published, for a range of pollutants and species, but Lincer (1975) has been the first to satisfy these two criteria

Figure 8.6   Changes   from   1901–1969   of   the   eggshell   index   for   the peregrine   falcon   in   Great   Britain.   ○,   eggshells   from   the   central   and   east Scottish   Highlands;   ●,   eggshells   from   other   districts.   (Reproduced   by permission of Blackwell Scientific Publications Ltd., from Ratcliffe, 1970)

(Figure 8.7). He worked on a related species, *Falco sparverius*, for which, in New York state, DDE was the most abundant insecticide found in eggs, although it must be   noted   that   the   available   data   suggest   that   PCBs   were   present   in   similar concentrations.

Ratcliffe discovered this phenomenon of thin eggshells as an indirect result of complaints by owners of racing pigeons that the peregrine falcon was exacting a heavy toll of their birds. In the event Ratcliffe found the complaint was ill founded; in fact the peregrine falcon had all but disappeared as a breeding species from southern England. Nobody had been aware of this.

Several theories have been advanced to explain how eggshells are thinned (Cooke, 1973). The most favoured theory is perhaps that an enzyme in the shell gland is inhibited: either carbonic anhydrase (Bitman *et al.*, 1970; Peakall, 1970), or Ca-ATPase (Miller *et al.*, 1976).

The effect of thin eggshells on the population size is again not simple to understand. Eggshells of the peregrine falcon in Great Britain have been thinner from 1947/8 onwards. Correlated with this, and presumably as a result, the proportion of eggs broken in the eyrie rose from 4 to 39%. It was another 8–10
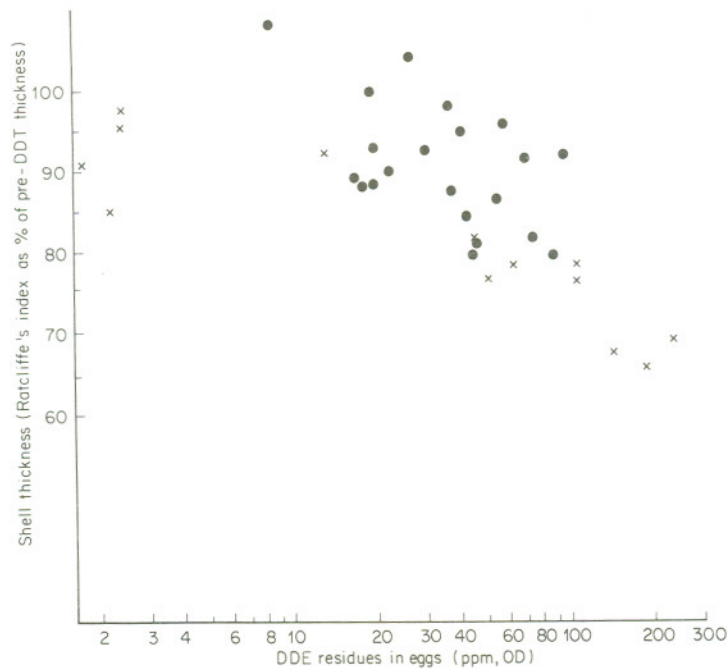
Figure 8.7   Relationship between mean clutch shell thickness and DDE residue of kestrel eggs collected in Ithaca, New York, during 1970 (●) and same relationship experimentally induced with dietary DDE (X). (Reproduced by permission of Blackwell Scientific Publications Ltd., from Lincer, 1975)

years however, before the number of peregrine falcons declined. This drop in the size of the breeding population was associated with the use of dieldrin as a seed dressing. Dieldrin too may cause thin eggshells, but it also killed many pigeons in the spring, when they ate the newly sown dressed cereal seed. It is estimated that a peregrine falcon could acquire a lethal dose from eating two or three heavily contaminated pigeons.

To summarize, this incident illustrates:

(1) Even an obvious complex of effects, such as thin eggshells, reduced breeding success and reduced population, in a very 'popular' species, can escape notice.

(2) It is difficult, particularly after the event, to decide on causes and effects.

(3) The detailed mechanism of the effect on the individual is almost impossible to predict, and difficult to discover, when the site of action is unknown;

organochlorine insecticides are usually considered to interfere with the transmission of nerve impulses (Narahashi, 1971), whereas this presumably is not the primary lesion that causes thin shells.

(4) The ultimate effects on the population, and even more on the community, are again not simple. The reduced breeding success of the peregrine falcon had no apparent effect on population size, although it has been suggested for the golden eagle (*Aquila chrysaëtos*) that a similar reduction in breeding success would result initially in an ageing breeding population. As this is a long-lived species the impact of reduced breeding success would be slow to appear in the population (Dempster, 1975).

## 8.6. CONCLUSIONS

We have become aware only during the last two decades of the need to consider the possible ecological effects of pollutants. It should be clear that we lack both toxicological and ecological understanding. Current practice, exemplified in particular by regulatory procedures for pesticides, consists of a modicum of toxicology, field trials and safety factors (Moriarty, 1977). Present testing methods are relevant and important, but if we are to increase our predictive abilities we must also, as has been emphasized by many advisory committees, undertake some long-term research projects. Important areas for research include:

(1) Development of the compartmental model as a means of understanding the factors that determine the amount and distribution of pollutants within animals.

(2) Understanding of pollutant modes of action, and the consequent sublethal effects on the whole animal's functions.

(3) Studies of relatively simple ecosystems, with simulations of significant aspects in laboratory experiments, in an attempt to improve our ability to understand and predict ecological effects.

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