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Estimating the Exposure of Birds and Mammals to Pesticides in Long-term Risk Assessments

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Abstract. This paper reviews current EU pesticide risk assessment guidance [European Commission (2002) Guidance document on risk assessment for birds and mammals under council directive 91/414/EEC, SANCO/4145/2000EC 2002], and examines some of its assumptions and problems arising from them. Issues associated with obtaining data that adequately describes exposure over the appropriate time-scale are common to both acute and long-term risk assessments but are probably less problematic for long-term exposure. Improvements in problem formulation and ways in which temporal and spatial factors might be incorporated into long-term risk assessments are suggested. The most important temporal issue for long-term risk is how best to model the degree to which wildlife habits are predictable from day to day. In relation to spatial factors, it is suggested that long-term risk assessments could make better use of pesticide usage data that sample usage patterns throughout the UK. The usefulness of detailed simulated farming landscapes populated by wildlife represented as agent-based models, should be explored.

Keywords: exposure; long-term; birds; mammals

Introduction

Current assessments (EU 91/414) of the acute risks of pesticides compare acute toxicity (as measured by mortality in the form of an LD50) with acute exposure to the pesticide. The long-term risk to birds and mammals is assessed when there is 'continuous or repeated exposure of adults' or when there is exposure of nest sites during the breeding season. Some pesticides, particularly (but not exclusively) if they accumulate, may have toxic effects that may only reach their peak, or only appear at all, in the longer term. Until recently, exposure in the long-term was assumed for simplicity to be the same as acute exposure. In other words pesticide residues on insects and vegetation were assumed to remain at a constant level (equivalent to the residues immediately after spraying) for the whole period making up the longer term. Animal behaviour, habitat use, diet composition, and food intake rate were also assumed to be unchanged. This exposure was compared to long-term toxicity tests expressed as the concentration of pesticide that produced No Observable Effect. Where the toxicity-exposure ratio (TER) <5, manufacturers need to provide further evidence that in practice the pesticide will not cause unacceptable effects.

The purpose of this paper is to examine the problems inherent in estimating wildlife exposure to pesticides and to consider how spatial and

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temporal factors might impinge on long-term exposure and how they might best be incorporated into long-term risk assessments.

How long is long-term?

Recent European guidance (EC 2002) recognises that residues on vegetation will not remain constant and suggests that, employing a suitable decay constant, they should be averaged out over a 3-week time period. But it admits it has no very good grounds for settling on this rather than another time period. Another option might be to set the exposure time scale to match that of the toxicity test it is being compared with. For example the current avian reproductive toxicity test (OECD Guideline 206) exposes subjects to contaminated food for 18–20 weeks. However, the length of the study was probably determined by logistical needs arising from the choice of quail as the test species and the time required for parents to raise young. (Furthermore, the subjects are exposed to fixed concentration levels throughout the test period. See Fischer (2005) on allowing for different exposure patterns in laboratory and field.) For mammals, there is a wider range of 'long-term' toxicity tests including a 90-day dietary test and multi-generation reproductive studies. These tests have been developed to protect the health of human beings and therefore their durations can have only an accidental ecological relevance to wild mammals exposed to pesticides on farmland.

In reality, we want to know what effects will occur as a result of the patterns of exposure that actually occur in the field. In principle therefore, the effects assessment should be driven by exposure assessment not the other way round. Bennett et al. (2005) show how existing laboratory long-term toxicity test endpoints may be linked to the time course and natural history of particular wildlife species at risk. Therefore, this paper does not settle on a particular duration for "long-term" but rather outlines a general model that will allow exposure to be estimated over the course of any term. Case studies (Shore et al., 2005; Roelofs et al., 2005) then attempt to link exposure to toxic effects presumed to occur in the field and ultimately to consequences for wildlife populations.

Factors determining exposure

If we assume that the only way an animal comes into contact with pesticides is through its food, then its simplest form the estimated theoretical exposure (ETE) of an individual to pesticides can be written as

$$\mathrm{ETE\ (mg/kg\ bw/day)} = \frac{\mathrm{FIR}}{\mathrm{bw}} \times C \qquad (1)$$

where FIR, food intake rate (g fresh weight diet/day); bw, body weight of indicator species (g); C, concentration of pesticide (mg/kg fresh weight diet).

If we know how much an animal eats and what residues are present on its food, we can calculate its exposure. (But see Mineau 2002, for a model that incorporates dermal and inhalation exposure). The current EU guidance takes its lead from the US EPA Ecological Committee on FIFRA Risk Assessment Methods (ECOFRAM 1999) and includes additional factors to account for the likelihood that animals may find pesticide residues distasteful or otherwise aversive, that different diets will have different residues and that not all of a given food type may be contaminated. Thus:

$$\mathrm{ETE}_k = \frac{\mathrm{FIR}_{\mathrm{tot}}}{\mathrm{bw}} \times C_k \times \mathrm{AV}_{k_c} \times \mathrm{PD}_k \times \mathrm{PT}_k \qquad (2)$$

where ETE_k, estimated theoretical exposure from consuming food k (mg/kg bw/day); $\mathrm{FIR}_{\mathrm{tot}}$, total food intake (g fresh weight/day); C_k, concentration of pesticide residues on food k; AV_{k_c}, avoidance of food k at residue concentration C (1 = no avoidance, 0 = complete avoidance); PT_k, proportion of food k obtained in treated area; and PD_k, proportion of food k in diet.

Equation 2 specifies the estimated theoretical exposure of an animal eating a particular food k on a particular day. The total exposure on any given day will be the sum of the ETEs calculated for all food types (k = 1 to n) in the diet. Equation 2 represents the current EU guidance but there are some dangers in this formulation, particularly when AV, PT and PD are not independent of each other. (We discuss later in the paper how the exposure equation might reformulated to make it less likely that interdependencies will be overlooked).

In case of multiple applications and/or long-term considerations current guidance (EC 2002) suggests that the concentration C may be expressed as

$$C = C_0 \times \mathrm{MAF} \times f_{\mathrm{twa}} \tag{3}$$

where C_0 = initial concentration after a single application; MAF, multiple application factor (concentration immediately after the last application compared to a single application (Gonzales-Valero et al., 2000); f_{twa}, time-weighted-average factor (average concentration during a certain time interval compared to the initial concentration.).

In first tier risk assessments a default value for f_{twa} of 0.53 is suggested, based on a half-life (DT50) of 10 days, an averaging time of 21 days and assuming first order kinetics as in Equation 4.

$$f_{\mathrm{twa}} = \frac{1 - e^{-kt}}{kt} \tag{4}$$

where k = ln 2/DT50 (velocity constant); t, averaging time.

Incorporating long-term temporal and spatial factors into exposure estimates

An obvious development from EC (2002) tier I risk assessment, in which long-term risk is assumed to last for 3 weeks, is to index Equation 1 so that we can explicitly take into account the time factor. In this way we are not confined to use a generic multiple application factor (MAF) but can make use of actual spray timings for a particular pesticide. We do not need to rely on default values for a pesticide's decay rate (DT50) and we can model residues on vegetation using empirical data or kinetic models as in Equation 4. In this way we can model likely exposure day by day and consider later the most appropriate interval over which to integrate the data.

A key factor determining the degree of exposure experienced by wildlife is the proportion of their diet that is contaminated with pesticide. And this is likely to vary according to location. If a large proportion of the diet is taken from untreated fields then exposure will be reduced accordingly. As a proxy for estimating the proportion of diet treated with pesticide we can incorporate the spatial dimension by indexing Equation 1 according to different habitats or fields visited by the organism in question. The US EPA Ecological Committee on FIFRA Risk Assessment Methods (ECOFRAM 1999) similarly indexes their exposure equation according to the organism's field use.

Finally, it is clear that the FIR, the daily food eaten by an organism is likely to comprise of several different food types, each with different energy values and containing different pesticide residues. If diet composition changes, so will the amounts of food consumed and so will the pesticides residues ingested. Therefore, we need also to index the equation according to different food types.

Combining these indices in Equation 1 gives us

$$\begin{aligned}&\text{ETE on day } i(\text{mg/kg bw/day})\\ &\quad = \sum_{j=1}^{nj}\sum_{k=1}^{nk}\frac{\mathrm{FIR}_{ijk}}{\mathrm{bw}} \times C_{ijk}\end{aligned} \tag{5}$$

where i, index for different foraging days; j, index for foraging in different fields (e.g. treated, untreated); k, index for different food types (e.g. insects, seeds, etc); n_j, total number of different fields visited; n_k, total number of different food types eaten; FIR_{ijk}, food eaten (g fresh weight) on day i, in field j of type k; bw, body weight of animal (g); and C_{ijk}, concentration of pesticide on day i, in food type k in field j (mg pesticide / kg fresh wt food). If field j has not been sprayed or subjected to spray drift by day i then $C_{\mathrm{ijk}} = 0$.

This indexing removes the need for introducing separate factors (as in Equation 2) for dietary composition (PD) and proportion of diet obtained from treated area (PT). The equation can also be used to estimate cumulative exposure (ignoring depuration) by summing over the appropriate number of days (i to n_i)

$$\begin{aligned}&\mathrm{ETE(mg/kg\ bw)}\\ &\quad = \sum_{i=1}^{ni}\sum_{j=1}^{nj}\sum_{k=1}^{nk}\frac{\mathrm{FIR}_{ijk}}{\mathrm{bw}} \times C_{ijk}\end{aligned} \tag{6}$$

Equation 6 implies that if we know the concentrations of pesticide present on different days, in different places on different foods, and we know an animal's intake of these foods, then we can

calculate its exposure. Given that we know the application rate of a pesticide, where and when it was sprayed and we have empirical or theoretical models of residues and their decay, then it is possible to estimate the concentration of pesticide residues remaining on any given food item. Estimating wildlife intake of different foods can be more difficult.

For wild animals living in natural habitat good quantitative data on the amount of food eaten daily or in the longer term (FIR in Equation 1) are rare. But there is increasingly good information on their daily energy expenditure (DEE). Using the doubly labelled water (DLW) technique (see Nagy, 1987) it has been possible to estimate DEE for a wide range of wild birds and mammals living in their natural surroundings and to derive allometric equations relating DEE to body weight (See Crocker et al., 2002). Knowing an animal's energy needs, it should be possible to estimate its fresh food intake rate by also exploiting knowledge about food energy content, moisture values and digestive efficiency. For an animal feeding solely on food type k:

$$\mathrm{FIR}_k = \frac{\mathrm{DEE}_k}{\mathrm{GE}_k \times (1 - M_k) \times \mathrm{AE}_k} \tag{7}$$

where FIR_k, food k intake rate (g fresh wt/day); DEE_k, daily energy expenditure of individual feeding on food k (kJ/day); GE_k, gross energy content of dry food k (kJ/g dry wt); M_k, moisture content of fresh food k (proportion between 0 and 1); AE_k, assimilation efficiency of food k (proportion between 0 and 1).

An animal's daily diet will typically consist of several food types with varying energy and moisture content and assimilation efficiency. Because some foods are easier to find and exploit than others, DEE may also vary according to the composition of the diet. Therefore, the total amount of food consumed in a day will vary according to the proportions of the diet made up by different foods (PD_k).

$$\mathrm{FIR}_{\mathrm{tot}} = \frac{\sum_{k=1}^{nk} \mathrm{PD}_k \times \mathrm{DEE}_k}{\sum_{k=1}^{nk} \mathrm{PD}_k \times \mathrm{GE}_k \times (1 - M_k) \times \mathrm{AE}_k} \tag{8}$$

Because different food types found in different habitats at different times are also likely to contain different pesticide residues, we will also need to know how dietary composition varies with time and place ($\mathrm{PD_i}$ and PD_j). Combining these factors and Equation 8 into Equation 5 gives where ETE_i,

$$\mathrm{ETE}_i = \sum_{j=1}^{nj} \sum_{k=1}^{nk} \frac{1}{\mathrm{bw}_{ijk}} \times \frac{\left(\sum_{j=1}^{nj} \sum_{k=1}^{nk} \mathrm{PD}_{ijk} \times \mathrm{DEE}_{ijk}\right)}{\left(\sum_{j=1}^{nj} \sum_{k=1}^{nk} \mathrm{PD}_{ijk} \times \mathrm{GE}_{ijk} \times (1 - M_{ijk}) \times \mathrm{AE}_{ijk}\right)} \times \mathrm{PD}_{ijk} \times C_{ijk} \tag{9}$$

estimated theoretical exposure on day i (mg/kg bw/day); DEE_{ijk}, daily expenditure of animal on day i, in field j eating food k (kJ/day); bw_{ijk}, body weight of animal on day i, in field j eating food k (g); PD_{ijk}, proportion of diet by fresh weight on day i, in field j, comprising food k (proportion between 0 and 1); GE_{ijk}, gross energy content on day i, in field j of food k (kJ/g dry weight); M_{ijk}, moisture content of fresh food on day i, in field j of food k (proportion between 0 and 1); AE_{ijk}, assimilation efficiency on day i, in field j of food k (proportion between 0 and 1); and C_{ijk}, concentration of pesticide on day i, in food type k in field j (mg/kg fresh weight food).

For any indexing, the individual dietary proportions (PD_{ijk}) must sum to 1. Thus on any given day (i),

$$\mathrm{PD}_i = \sum_{j=1}^{n_j} \sum_{k=1}^{n_k} \mathrm{PD}_{ijk} = 1 \tag{10}$$

For the sake of completeness, in Equation 9 all the parameters have been indexed for time, place and food type. In reality, it may be possible and often necessary to make some simplifying assumptions (see Table 1). For example, although it is likely that energy expenditure will vary with the food availability (e.g. animals might choose to feed on low energy foods only if the cost of finding and digesting them is correspondingly low) we rarely have information on

Table 1. Alternative formulae for estimating exposure, making increasing assumptions of about diet, habitat, residues, etc

Formulae for *Daily* estimated theoretical exposure	Assumptions (cumulative)	Remarks
a $\sum_{j=1}^{nj}\sum_{k=1}^{nk}\frac{\mathrm{FIR}_{ijk}}{\mathrm{bw}_{ijk}}\times C_{ijk}$	Exposure is wholly through residues on food	Evidence exists that exposure through skin contact and/or inhalation may be important (Mineau, 2002; Driver et al., 1991)
b $\frac{1}{\mathrm{bw}}\times\left(\sum_{j=1}^{nj}\sum_{k=1}^{nk}\mathrm{FIR}_{ijk}\times C_{ijk}\right)$	Individual body weight (bw) is constant across time, place and diet	Depending on species, body weight may be expected to be relatively stable over the course of a few days, but for longer-term exposure this may be invalid. It is likely that there are correlations between body weight and habitat or diet e.g. animals in cold climates or eating fatty foods may be fatter.
c $\frac{1}{\mathrm{bw}}\times\frac{\left(\sum_{j=1}^{n_j}\sum_{k=1}^{n_k}\mathrm{PD}_{ijk}\times\mathrm{DEE}_{ijk}\right)}{\left(\sum_{j=1}^{n_j}\sum_{k=1}^{n_k}\mathrm{PD}_{ijk}\times\mathrm{GE}_{ijk}\times(1-M_{ijk})\times\mathrm{AE}_{ijk}\right)}\times\sum_{j=1}^{n_j}\sum_{k=1}^{n_k}\mathrm{PD}_{ijk}\times C_{ijk}$	Food intake rate is determined by daily energy expenditure	It may be invalid to assume that DEE is given and that individuals must find sufficient food to satisfy it. E.g. animals may eat in excess of energy requirements to lay down fat., which they may later draw on when running negative energy budgets.
d $\frac{\mathrm{DEE}}{\mathrm{bw}}\times\frac{\sum_{j=1}^{n_j}\sum_{k=1}^{n_k}\mathrm{PD}_{jk}\times C_{ijk}}{\sum_{k=1}^{n_k}\mathrm{PD}_k\times\mathrm{GE}_k\times(1-M_k)\times\mathrm{AE}_k}$	Daily energy expenditure is constant over time, place and diet.	DEE values for birds are mostly taken from the breeding season when they may be expected to be working harder than average (see text). The energetic costs of foraging are also likely to vary according to habitat and food type.
	Food quality (GE, M, AE) is constant over time and place	Food quality may change with time and habitat.
	Diet composition (PD_{jk}) is constant over time	Diet is likely to vary from day to day
e $\frac{\mathrm{DEE}}{\mathrm{bw}}\times\frac{\sum_{j=1}^{n_j}\sum_{k=1}^{n_k}\mathrm{PD}_j\times\mathrm{PD}_k\times C_{ijk}\times\mathrm{AV}_{ijk_c}}{\sum_{k=1}^{n_k}\mathrm{PD}_k\times\mathrm{GE}_k\times(1-M_k)\times\mathrm{AE}_k}$	Diet composition is not related to choice of feeding place.	Unlikely to be valid where available feeding habitats are dissimilar containing different food types. (e.g. fish is unlikely to figure in seagull diet on arable land)
This formula is equivalent to that used in current EU guidance (Equation 2) where PD_j & PD_k above are replaced by PT and PD, and Daily Energy Expenditure/Food Energy are replaced by Food intake rate	Avoidance occurs additionally to choice of feeding place and choice of food type	Avoidance of pesticide-treated food is most likely to show itself in the choice of feeding habitat (i.e. untreated rather than treated fields, perhaps already accounted for in PD_j) and in choice of food type (PD_k). Including an avoidance factor together with a spatial factor (PD_j) and a diet composition factor (PD_k) is only justified if either (a) values for PD_j and PD_k have been obtained from untreated environments or (b) if it can be shown that, within a given field or food type wildlife will forage selectively on less contaminated food items.
	Avoidance is proportional to residue concentration	Avoidance is most often investigated in laboratory tests which may not be good predictors of behaviour in the field. Avoidance may occur after a particular dose has been ingested rather than in proportion to concentration

the energetic costs of feeding on particular foods. In practice, data from DLW studies give estimates of DEE for wild animals eating varied diets across varied habitats.

For any given day, therefore, we now need to know how diet composition varies from field to field. We can then use our knowledge of energetics to calculate how much of any food type will be eaten and our knowledge of pesticide residues to calculate exposure. Often however our knowledge of diet composition is limited to proportions obtained from gut or faecal samples which are necessarily averaged over different foraging habitats, giving us an estimate of PD_k. Similarly, close observational data such as that provided by radio-tracking can give us information on how much one field is used compared to another giving us an estimate of PD_j (or PT in Equation 2) but the observations will not often distinguish between food types obtained there. Therefore while we may have good information on PD_j and PD_k, we cannot infer PD_{jk} unless we additionally assume that an animal's diet remains the same regardless of where it is feeding (or more strictly, unless we assume there is no association between food type and foraging habitat). For animals that are highly dependent on a given crop type (e.g. skylarks in cereal fields) for their normal foraging habitat this may not be an unreasonable assumption, but even here it would seem likely that skylarks foraging in a field treated with insecticide are less likely to feeding on insects there than in the untreated set-aside alongside. For a species with a varied foraging routine and which, in the course of the day finds food from very different habitats, it may be especially misleading to assume that diet and place are unrelated. Brent geese for example are known to feed principally on salt-marsh vegetation but at high tide will come inland to feed on cereal shoots. If we assume that a Brent goose divides its time evenly between salt-marsh and a pesticide-treated cereal field, and further assume that it feeds at a similar rate in both, then about 50% of its food will be obtained from the treated area (PD_j) and about 50% of its diet may be cereal shoots (PD_k). If the goose is on cereal land it cannot be eating salt-marsh vegetation and if it is eating salt-marsh vegetation it cannot be on pesticide treated habitat. PD_j and PD_k are not independent. Clearly it would be mistaken to combine these factors as a simple product and assume that $PD_{jk} = PD_j \times PD_k$ and thus infer for example that because a goose spends only half of its time on pesticide-treated habitat and because on average its diet consists only half of cereal leaves that therefore the proportion of a goose's diet made up of pesticide-treated cereals will be $0.5 \times 0.5 = 0.25$.

In the example of the Brent goose, it is not difficult to see that exposure is wholly determined by diet composition and to take account of this in risk assessment calculations. However, experience suggests that the exposure formulation in current EU guidance (Equation 2) and in the US EPA Ecological Committee on FIFRA Risk Assessment Methods (ECOFRAM 1999) which present dietary composition (PD) and proportion of diet obtained from the treated area (PT) as separate entities, arguably, increase the likelihood that correlations between them will be ignored.

PT in Equation 2 represents the proportion of diet obtained from treated areas. As such it rather simply divides an animal's environment into treated and untreated habitats. Replacing PT with PD_j, a spatially explicit factor, enables one to model more subtle variations in residues. Each location represented by PD_j will have associated with it a value C_j, the concentration of pesticide residues at that place. C_j could for example, incorporate different application rates at different locations, the effects of spray drift from crop into field margin or different decay rates on different crop structures.

In Equation 2, it may also be mistaken to treat the avoidance factor (AV) as independent of PD or PT. Rather avoidance is likely to manifest itself as a change in diet or foraging habitat to one that it is less contaminated with pesticide. If there are no data available on the relative use made of treated and untreated fields, then data on avoidance of treated food in the laboratory might perhaps be used as a substitute (though see the section below on avoidance).

To summarise: wildlife exposure to pesticide is a function of when, where and what is eaten. Given that we can estimate pesticide residues on a food type found in a particular place and time (C_{ijk}), and how much of that food is eaten by a wildlife individual (FIR_{ijk}), then we can estimate its exposure. If we do not have good empirical data

on the *absolute* amount of different foods eaten (FIR_k), we can estimate it using information on the *proportion* of food types normally taken by an animal in a typical day (PD_k), and using energetics equations, estimate how much of the different food types must be eaten in order to satisfy DEE. Estimates of PD_k for many species can be found in the ecological literature as analyses of stomach or faecal contents. If we know the proportion of diet that comes from the treated area (PD_j) and we are prepared to assume that diet does not change with habitat then we can calculate the proportion of a given food type that is likely to be contaminated with pesticide as $PD_{jk} = PD_j \times PD_k$. Estimates of PD_j can be obtained from radio-tracking data (PT in current EU guidance) or perhaps from laboratory avoidance studies (AV in current EU guidance). If we are aware that diet does change with habitat then we need to incorporate this knowledge in estimating PD_{jk}.

A progressive list of different exposure formulations and the assumptions that go with them is shown in Tables 1 and 2. As we move from formula ***a*** which is fully indexed for time, place and food type, to formula ***f*** these constraints are gradually relaxed at the expense of increasingly simplified assumptions about reality. Paradoxically, as we make more and more simplifying assumptions the equations appear more complex. But this is largely because we are not omniscient: we do not always know how much of a given food type was eaten in a given field on a given day, and therefore we must attempt to parameterise these variables using other variables about which we do have information (such as energy expenditure and food quality).

The initial formulation (***a***) is perhaps pedantic: it is not very likely that risk assessors will normally need to take account of an individual's day to day fluctuations in body weight and therefore the assumption that body weight remains constant (formula ***b***) is a realistic simplification. Similarly, we may acknowledge that the moisture content of earthworms changes from field to field but we might prefer to accept the simplifying assumption that they do not. It might seem reasonable to treat food energy, moisture content and assimilation efficiency as varying only with food type and not with time and space. In other words, a weed seed eaten on May 1 from a barley field is assumed to be nutritionally identical to a weed seed found on May 2 from a wheat field. However, if there is evidence that these attributes vary in time or space then they should also be indexed as in formula ***c***.

Other simplifications may be more incautious. We might, for example assume that DEE is constant from day to day and place to place and does not depend on food types available. But we know that for some species in some circumstances the reaction to a poor food supply is not to seek out better ones but rather to reduce unnecessary energy expenditure and to hibernate until the food supply improves. If there is evidence that DEE does vary consistently then it should be indexed appropriately (see section on "Differences in food intake rate (FIR)" below).

Some simplifications are perhaps inevitable because the necessary data are not available. The object of Table 1 is not to undermine current exposure formulations but rather to make clear what assumptions they entail, to highlight the data gaps and to encourage risk assessors to carry out risk assessments at the level most appropriate to the data they have available to them. The case studies (Shore et al., 2005; Roelofs et al., 2005) give concrete examples of how to implement exposure formulae.

From the point of view of routine regulatory risk assessment, the indexing in Equation 6 and formula ***a*** in Table 1 may appear to be overcomplicated. The point of including this level of detail is not to suggest that it should inevitably be included in risk assessment schemes. Table 1 shows how exposure might be calculated using less detailed information and it makes clear what assumptions necessarily accompany these simplifications. It may be useful to explore how faithfully these simpler schemes reflect a more complex reality. With more detailed models we may be able to make recommendations for changes to EU guidance on Tier I long-term risk assessments. For example, current guidance imagines an animal visiting a single field, treated with a single pesticide, with allowance made for repeated applications. For pesticides where treatment is restricted to a single application, assessors will presumably ignore the Multiple Application Factor. Perhaps this is incautious. A higher tier assessment taking spatial and temporal factors into account may indicate that the focal species visits a variety of

Table 2. Key to parameters used in Table 1 and comments on quality of data available

Parameter		Data availability
FIR_{ijk}	Amount eaten on day *i*, from field *j* of food *k*	Few data available on absolute quantities wildlife food intake in the field. Fewer available on how quantities eaten of different foods change with time and with feeding habitat
C_{ijk}	Concentration of pesticide residues on day *i*, from field *j* on food *k*	Empirical data from pesticide manufacturer. Good generic data on foliage residues. Little currently available on insect residues
bw_{ijk}	Body weight on day *i*, from field *j* of food *k*	Little data on how body weight changes from day to day, place to place or according to diet. Adult body weights typically categorised according to sex and season. Fewer data on juveniles or chicks.
DEE_{ijk}	Daily energy expenditure on day *i*, in field *j*, feeding on food *k*	Available from energetics equations, if body weight is known. Little data to show how it varies with time, place or food type.
GE_k	Gross energy in food *k*	Data on nutritional quality of different foods is available from literature but fewer data on specific wildlife foods e.g. moisture content of rape-seed at the time when linnets are feeding on it.
M_k	Proportion of moisture in food *k*	
AE_k	Assimilation efficiency of food *k*	
PD_{ijk}	Proportion of diet on day *i*, from field *j* of food *k*	Little data on how time, place and food type all interact in diet composition
PD_{ik}	Proportion of diet on day *i* of food *k*	Seasonal data often available but little data on day to day changes in diet composition
PD_{jk}	Proportion of diet from field *j* of food *k*	Some ecological data linking diet composition to foraging habitat (e.g. skylarks on cereal versus pasture fields)
PD_k	Proportion of diet comprising food *k*	Ecological data often available from gut or faecal samples on diet composition.
PD_j	Proportion of diet from field *j*	Some radio-telemetry data for some species on relative use of farming habitats.
PT	Proportion of diet from treated area	Term used in EC and US EPA guidance. Equivalent to PD_j above and assumed to be reflected in relative proportion of feeding time spent in different locations
AV_{ijk_c}	Proportional avoidance of food treated with pesticide at concentration *c*.	Data typically from laboratory studies with uncertain relationship to behaviour in the field. Some studies have shown evidence of field avoidance (e.g. McKay et al., 1999). Avoidance may not be proportional to residue concentration (see text)

fields over several days and could experience high levels of exposure on repeated days. On the other hand, the number of visits to fields where no pesticide has recently been applied may offset this. By making full use of detailed data it may be possible to recommend a MAF for lower tier assessments that takes account typical wildlife field use and spray regimes.

Serial autocorrelation

For an acute risk assessment we might simulate exposure on any particular day by repeated random sampling from the distributions representing the key exposure parameters (field type, food type, pesticide concentration and so on). However, it is clear that the exposure experienced on one day will not be independent of the exposure experienced on preceding days. A particular individual is not free to travel randomly over the full range of habitats available to the species as a whole. Thus a bird found foraging in a given field on one day is likely to be feeding somewhere near it the next day. The foods it selects are also unlikely to change randomly from day to day but will depend on predictable changes in abundance. We should try to take account of this sameness (serial autocorrelation) in our modelling.

Other non-random behaviour of wildlife may also need to be considered. For example it may not be justified to assume that wildlife encounter treated food randomly in the course of their foraging activities. Birds which follow the plough in search of invertebrates or newly exposed seed banks may be particularly vulnerable to seed treatments or granular treatments. Similarly if animals are attracted to a field because it is rich in insect pests then they may also be particularly vulnerable to sprays applied by the farmer in response to the pest problem. On the other hand farmers that spray prophylactically may create environments with so few wildlife food sources that they are avoided as foraging habitats. The direct effects of pesticide use on the pests may interact with indirect effects such that intensive pesticide use may paradoxically lead to lower pesticide exposure. Non-random attraction or avoidance of treated food might be reflected in adjustments to PD_{jk} (PT, PD or AV in current EU terminology).

Multiple pesticides

A regulatory assessment of pesticide risk will normally consider only a single product at a time. In reality animals may be exposed to several products and formulants in short succession or even simultaneously. The longer the time course we are considering the more likely that more than one product may be encountered. Different pesticides may be mixed in the same tank but even when they are applied separately, in the long-term animals subsisting on farmland will be exposed to several different chemicals. This is also likely to be true in the short term where animals may visit several different fields containing different crops receiving different pesticide treatments. There are likely to be synergistic effects between some pesticides (Thompson, 1996), though for human beings at least their importance is thought to be small (Committee on Toxicity, 2002) Ideally, Equation 6 would also be indexed by pesticide product. The issue is an important one but probably intractable within current regulatory framework. A risk assessment that considered multiple products would consider the safety of a product not only on its own merits but would also need to consider the potential damage it might do in possible combination with a competitor's product.

How exposure factors might differ in the long-term

With the appropriate information Equation 5 and Equation 6 enable us to predict pesticide risks to wildlife in both the short- and long-term and across different patterns of habitat use. Unfortunately appropriate information, particularly about wildlife foraging preferences across time and space is very limited. The following sections examine what information is available, how it may best be used for long-term risk assessment, and how it might differ from acute exposure.

Differences in food intake rate (FIR)

Crocker et al. (2002) estimated food intake rate by using information on DEE in the field and com-

bining it with information on the energy provided by wildlife foods. A review of the literature found examples of field DEE, estimated using DLW isotopes, for 96 bird species and 73 mammal species. For both birds and mammals, there was a strong relationship between body weight and DEE. There were significant differences between taxonomic groups and species occupying different habitats. Therefore separate equations were calculated for the mutually exclusive categories, sea-birds, desert birds, hummingbirds, passerines (terrestrial, non-desert species) and others (Table 3). Mammals were similarly divided into eutherians, non-eutherians, and into desert mammals, sea mammals and terrestrial mammals (Table 4).

There is a range of issues that needs careful thought concerning the methodology of predicting food intake from wildlife daily energy requirements. In brief

- The above allometric equations link mean bodyweight of a species with DEE. The same equations might be used to predict the DEE of individual animals. For example, the equation may be used to predict not only the DEE of an average great tit but also of heavier and lighter great tits. However, further analysis suggests that the relationship between body weight and DEE scales differently within a species than between species. For example Tinbergen and Dietz (1994) have shown that the intra-species slope linking bodyweight to DEE within great tits is much steeper the inter-species slope in Table 3. Therefore if it is important to model *individual* variation in energy expenditure then the allometric equations above may not be adequate. It may be necessary derive an allometric relationship based on individual data points rather than species' averages.
- The premise of this method of estimating food intake is that DEE is met by daily food intake. In the longer term the energy budget must be balanced or the animal will die. But over shorter time scales animals may build up energy surpluses and store them to draw upon in leaner times.
- There is an assumption in this method that the DEE is given and the animal is attempting to find sufficient food to satisfy it. However, an alternative to solution to balancing an energy budget is to reduce expenditure to match availability. Thus animals may prefer to feed on a nutritionally poor food source if it is easily accessible and demands little energetic effort to exploit.
- Although Equation 9 takes into account the assimilation efficiency with which an animal converts the energy in food into energy available for its own purposes, it does not explicitly take into account the energy spent finding, pursuing, subduing, dismembering its prey. Optimal foraging theory has demonstrated that that it should pay animals to select the most *profitable* food items rather than those that are simply more energy rich (e.g. Pyke et al., 1977).
- The unthinking equation of DEE with food intake can result in absurd results. For example if one assumes that an animal has only a single very poor food source available to it then the energy equation may suggest that it would need to consume several times it own body weight each day to satisfy its DEE. Extreme results like this do not often occur when one is considering mean DEE and mean

Table 3. Relationship between body weight and field DEE in birds for five avian habitat groups

Group	log *a*	SE Log *a*	*b*	SE *b*	*N*	R^2
Desert	0.6107	0.1727	0.7299	0.0663	7	0.95
Sea-bird	1.1482	0.1022	0.6521	0.0356	35	0.91
Hummingbird	0.7495	0.0822	1.2064	0.1090	5	0.97
Passerine	1.0017	0.0647	0.7034	0.0503	38	0.84
Other	0.6768	0.1896	0.7723	0.0861	11	0.89
All birds	1.0220	0.0392	0.6745	0.0180	96	0.94

(Crocker et al., 2002) Log(DEE) = log *a* + b. (log Body weight g).

Table 4. Relationship between body weight and DEE in mammals for five mammal habitat groups

Group	log a	SE Log *a*	*B*	SE *b*	*N*	r^2
Desert	0.5120	0.0625	0.7843	0.0290	18.00	0.98
Marine	2.4203	0.7592	0.4266	0.1567	6.00	0.56
Terrestrial	0.8459	0.0526	0.7050	0.0250	30.00	0.96
All non-eutherian	1.0232	0.0749	0.5814	0.0251	19.00	0.97
All eutherian	0.6794	0.0445	0.7646	0.0173	54.00	0.97
All mammals	0.7401	0.0467	0.7204	0.0174	73.00	0.96

Log(DEE) = log *a* + *b*. (log Body weight g).

food energy content but they are not unusual in probabilistic risk assessments where one is simulating individual food consumption and drawing random values from DEE and food energy distributions. One solution to the problem is to build in a positive correlation between the distributions so that a poor food source is rarely paired with a high DEE. Or we might truncate the output distribution of predicted food intake to give plausible range. However, both these stratagems prompt the question: do we have objective criteria for choosing correlation coefficients or truncation values which engineer a result that we find subjectively plausible?

- Food consists of more than energy. Animals may be eating to satisfy protein, mineral or vitamin requirements rather than simply balancing an energy budget.

There are issues arising from this discussion that are particularly relevant to long-term estimates of FIR.

- As the name suggests, DEE concerns days rather weeks. The usual way of obtaining information on DEE is to catch a wild animal inject it with DLW and catch it again a day or two later. Energy expenditure is estimated by measuring the relative turnover of heavy isotopes of hydrogen and oxygen. Currently there are few data on longer-term energy expenditure. In the absence of other evidence, it may be simplest to assume that energy expenditure over the longer term is approximated by summing daily expenditure with successive days linked with an appropriate serial auto-correlation factor.
- For birds, the DLW data are heavily focused on the breeding season. (When a bird is feeding chicks at the nest, it is much easier to catch it on successive days than it is to recapture a widely ranging bird in winter.) In the breeding season birds are more likely to be working harder for longer periods than in winter. DLW evidence supports this (Crocker et al., 2002). Where a pesticide is used largely outside the avian breeding season, it may be worth calculating DEE using allometric equations that take season into account. For small mammals, Speakman (1999) has shown that latitude and ambient temperature of the capture site are also significant factors determining DEE.
- Data on calorific and moisture content of foods are very variable in quality and appropriateness for the job of calculating wildlife food intakes. Ideally we want to know the calorific value of a particular food eaten by a particular wildlife species, at a particular time and place. (Animals usually choose the most profitable exemplars of the foods available to them so we may even want to know which individual seeds a birds picks, or what part of the plant a vole grazes.) Unfortunately the calorific values available in the literature have not been measured with these needs in mind. For example, the energy value of rape seed in the literature will be averaged over many seeds, collected at harvest for the benefit of agronomists. Whereas we may prefer to know the value of unripe seeds selected by linnets in spring. It may be possible to select examples from the literature that have more or less relevance to long-term or short-term risk assessments.

These shortcomings in data quality probably pose more of a problem for acute rather than longer-term risk assessments. Given that values for food energy and moisture contents are usually mean values of many food items, perhaps of different provenance then they will be less appropriate for estimating food consumption of a particular individual feeding on a particular food type at a particular place. Our data may not be at the fine scale needed to capture transient fluctuations in day to day food energy content but given that long-term exposure will average across these fluctuations it may be less important to capture them in our models. If this argument is correct however, we need to consider more precisely how the averaging process in long-term exposure might resemble the averaging in process in food sample data.

Changes in body weight (bw)

Depending on the time scale over which we are considering the "longer term" it is unlikely that changing body weight of adult birds or mammals

will have a significant impact on exposure. Exceptions might include periods where birds are gaining weight in preparation for migration or mammals laying down winter fat. However, for young animals still growing then changing body weight will need to be factored in to exposure estimates. Depending on the geographical scale, the risk assessment may need to take account of body weight variations with locality. In the UK, for example, the water vole (*Arvicola terrestris*) lives near water and weighs about 300 g, (Corbet and Harris, 1991), whereas on the European continent it ranges much more widely and is less than a third as heavy (e.g. Grenot et al., 1984).

Pesticide concentration on wildlife foods (C)

Interception factors

Before a pesticide spray reaches its target or it non-target, the droplet may evaporate, drift out of target zone or be intercepted by something else. Droplet size, wind strength density crop canopy may each affect the likelihood that the pesticide will be precisely delivered to the target surface. The EC guidance uses interception factors recommended by FOCUS (2000) working group. It seems unlikely that these will differ between long-term and acute exposure.

Residues on insects

The EC guidance reviews recent work on estimating residues on vegetation and insects (Luttik, 1992; Fletcher et al., 1994; Pfleeger et al., 1996; Brewer et al., 1997; Fischer and Bowers, 1997; Edwards, 1998; Joerman, 1998). In the past the majority of data concern pesticide residues on plants and residues on insects have been derived from these. Thus it was proposed by Kenaga (1973) to treat large insects as if they were grains of cereal and small insects as if they were seeds and forage crops. And this has been common practice in risk assessment in the USA and UK.

The EC guidance reviews the latest data and proposes new RUDs (residues per unit dose) for different vegetation types and insect categories. Multiplying RUDs by application rate gives an estimate of the initial residues on insects. Recognising that little is known about the time course of insect contamination, the EC guidance advises that it is inappropriate to speculate about the residue decay on insects and suggests that risk is calculated using the initial concentration received. This would seem to be a conservative approach but it implies that maximum residues occur immediately after application. However Scientific Committee on Plants (SCP, 2002) in its comments on an earlier draft of the EC guidance notes that there are clear differences between plants (which stay put) and arthropods (which move around). Residues on vegetation may be expected to decay (Willis and McDowell, 1987) but residues on insects can increase with time. Hart and Thompson (2001) found that in some existing datasets the first sampling period did not give the maximum residues. The biggest increases after the first sampling period were seen in studies with organochlorine pesticides (e.g. Zaranyika and Mugari 1996), but substantial increases were also seen for other types of chemical, including organophosphorus and carbamate pesticides and a herbicide.

The analysis by Hart and Thompson (2001) showed that, for studies where pesticides were applied at less than 1 kg a.s./ha, measured residues on insects were about an order of magnitude higher than would be expected based on the assumption of a simple linear relationship between dose rate and initial residues. Therefore, using the RUDs may substantially under-estimate actual residues at low application rates. This may be caused by factors such as differences between insect types or pesticide types.

It is unclear how common such cases are. Research is underway to carry out empirical measurements of residues on insects (Glass, 2003). In the meantime, some degree of conservatism is advisable when using initial residues to allow for the possibility that higher residues occur later. If there is reason to expect an increase in residues over time, based on pesticide persistence, the mechanism of uptake, or the type and life-stage of insect concerned, then consideration should be given to requiring appropriate field measurements.

Pesticide residues on insects are likely to vary with the type of pesticide used. Insects exposed to insecticides are likely to disappear quickly from the food chain, whereas insects exposed to herbicide may survive longer. In the short term, the appearance of large numbers of freshly killed insects may prove an attractive food source, while

in the longer-term desiccated insect remains may be avoided (Stafford et al., 2003). In the longer-term also, the amounts of pesticide residues will be diluted by insect immigration and emigration.

For some pesticides, the breakdown of the parent compound may give rise to more toxic metabolites (e.g. dimethoate breaking down to omethoate, or fipronil to its desulfinyl) in which case separate risk assessment should be carried out in relation to pesticide breakdown products.

Residues on plants

Once deposited, residues on plants may be expected to disappear over time through volatilisation, or they may be washed off by rain or dew, they may break down naturally or be metabolised by the plant. Residues may also be diluted in the longer term by plant growth. Although many researchers assume that this disappearance is exponential and well-modelled by first order kinetics, Willis and McDowell point out that residues often decline very rapidly to begin with but that the rate of loss slows down "so that many residues ultimately persist for longer than predicted by first order kinetics". We should consider what effect might follow from other models of pesticide residue decline (Hamaker, 1972; Willis et al., 1985, Stamper et al., 1979).

The best source of information on longer-term loss of pesticide residues is likely to come from empirical data provided by the manufacurers of particular pesticides. The best available generic data on half lives calculated assuming first order kinetics is given in a review of 81 pesticides by Willis and McDowell (1985). They group pesticides into four categories with the following DT50 values (in days) and standard deviations

Organochlorines	5.8 ± 6.0 days
Organophosphates	3.3 ± 2.6 days
Carbamates	2.7 ± 1.2 days
Pyrethroids	5.9 ± 5.0 days

Data on typical spray regimes and field pattern in UK

CSL's Pesticide Usage survey routine collects information on spray regimes used by UK growers (e.g. Garthwaite et al., 2003). For any given crop we know which products were used on which dates and how long elapsed between sprays. These data should allow us to construct realistic models of longer-term exposure in any given crop and season and with realistic assumptions about repeat applications. These data are also spatially referenced at the level of farm. With information on cropping pattern it maybe possible to infer likelihood that particular fields within an animal's home range may or may not be sprayed with a particular pesticide.

Detailed information on cropping patterns is not readily available. The UK government agricultural census gives information about crop hectarages according to parishes but not by fields. Field patterns are available from the Ordnance Survey and land coverage based on satellite imaging is available from the UK Centre for Ecology and Hydrology (Fuller et al., 2002). Unfortunately the land cover mapping does not easily distinguish between crops, although it may be possible to identify winter cereals from spring planted crops. We know that farmers prefer as far as possible to plant fields in blocks of the same crop. Between these different data sources it may be possible to simulate a range of cropping patters from clumped to over-dispersed. If we know what a typical cropping pattern looks like we might be able to estimate what crops might be simultaneously available to a bird nesting in the crop centre or in the margins between crops. (CSL has data on short term habitat use of several arable birds and the woodmouse.) Even where the crop on either side of a hedge is the same and both are treated with the same product, they may not necessarily be treated at the same time. When the spatial scale is taken into account, wildlife may be vulnerable to multiple exposures not evident when considering only acute events.

Even if data on spatial spraying patterns can be inferred from knowledge of cropping pattern and pesticide usage, the best way to model their effects on wildlife exposure is not entirely clear. One solution to this problem (e.g. Topping and Odderskaer, 2004) makes use of improved computing power to build spatially and temporally explicit models that attempt to simulate real landscapes of fields and hedges and real farming practices. Such model environments may then be populated by virtual individuals (or agents) of wildlife species which follow a set of rules distilled

from their natural history. Exposure then becomes an epiphenomenon of the biological agents (e.g. skylarks, wood mice) going about their normal business of feeding and breeding.

An advantage of this approach is that it makes use of fine-grained information to capture responses of individual animals to particular contingencies of landscape, farming practices and climate. The simulation thus attempts to fully encompass the stochasticity of real lives in away that is very difficult to build into generic models. The effect on a wildlife population is then the sum of the particular individual fates comprising that population. The fate of the population itself may be simulated by running the model over many years. The success of the enterprise of course depends on the adequacy of the rules that are built into the biological agents. A disadvantage of this approach may be that the fine detail that is necessary to run these simulations may not be available for all scenarios of interest and risk assessors running such models would have to confine themselves to a few standard scenarios.

Avoidance of pesticides (AV)

Many pesticides taste unpleasant or cause animals to feel unwell later and it may be demonstrated in short-term laboratory tests that, given a choice, animals by instinct or by experience, avoid them. Depending on the mechanism by which a pesticide causes aversion, the effects on food consumption can be rather different.

- Equation 2 (the format preferred in current EU and US guidance) implies that avoidance of treated food is a constant proportion of the amount consumed. Thus if the avoidance factor is 0.1 and daily food consumption of food after taking into account PD and PT is expected to be 10 g then the animal will eat 1 g, whereas if expected consumption were 20 g the animal might be expected to eat 2 g. However, for products that are not immediately disgusting then avoidance may occur as a result of sickness experienced after a given dose. In this case avoidance is related to the absolute amount of product consumed and may not be proportional to the amount of treated food available (Fryday, personal communication). If avoidance occurs as a result of learned association between food and illness then we may need to take account of the learning process in the risk model and, in particular, estimate for a given concentration of pesticide and a given foraging pattern whether an effective dose is likely to be consumed and whether it is likely to be associated with a particular food or location. For example, a very hungry animal may feed so quickly that it takes in a lethal dose of pesticide, long before it starts to feel ill and learns to avoid the crop in future.
- Even where the pesticide is immediately detectable and disliked by the animal it may be mistaken to include AV in the same equation with PT and PD. If data on PT and PD have been collected in field studies, where crops are treated with the pesticide under consideration (or another pesticide with aversive properties) then they may already include the avoidance factor. If a field is sprayed with a pesticide and the animal finds it aversive then the most obvious demonstration of this aversion is to forage elsewhere. Similarly, if a particular food type is likely to be contaminated with high residues then the animal may eat less of that food type. Any modification to dietary composition is likely to result in changes to the gross amount eaten. If for example, treated seeds are avoided in preference for leaves then it will take a large weight of leaves to make good the energy loss. If animals avoid a food type because it is tainted then this will be reflected in the proportion of the diet the particular food occupies and by the proportion of time the animal spends in areas treated with pesticide. Therefore if avoidance is to be included in risk assessments then it may be better to model its effects on PT and PD rather than to treat it as an additional, independent factor.
- Including an avoidance factor as well as PT and PD will lead to double counting unless it is clear that the values used for field use and dietary composition are obtained from untreated habitats or it can be shown that the avoidance factor describes only the additional ability of animals to select between contaminated and uncontaminated food items within a treated field or food type. If avoidance refers only to selection between individual food items

containing different pesticide residues then we additionally need to know how residues differ between individual food items.
- Avoidance is assumed to be proportional to pesticide concentration. As residues decay over time, so will avoidance and so will toxicity. If avoidance decreases at a rate greater than toxicity then risk may be greater in the longer term.
- How representative are short-term lab studies of longer-term behaviour in the field? In the laboratory, neither a simple 2-choice test between contaminated and uncontaminated food, nor a short-term no-choice test properly capture conditions as they occur in the field. For wild animals there is usually a choice but there is usually a cost (travel, search time) in making that choice, between remaining in one food patch and moving to another. In the short-term it may be relatively easy to avoid unpleasant-tasting foods, but if those foods remain unpleasant tasting for days or weeks and few alternatives are available then avoidance may be overcome. For example, in pen trials diazinon was strongly avoided by geese, yet many geese were poisoned in the wild (Mineau, personal communication). On the other hand, if a pesticide causes illness but is otherwise hard to detect then it may take the animal several days to learn the association and avoidance may increase with time.

Diet composition (PD_k)

Depending on time period, diet is likely to change because of

- season (soil gets dry, earthworms retreat lower, plants grow, fruit, set seed, senesce, insects lay eggs, hatch larvae, pupate)
- physiological development (fledglings may be feeding for themselves on seeds rather than being brought insects by parents). (And normally granivorous parents may be feeding on insects that they catch for chicks). Females laying eggs may attempt to find calcium-rich food sources at this time e.g. increasing grit consumption making birds more vulnerable to granular pesticides.
- Short versus long-term averaging. It is not hard to think of circumstances when diet composition in the short term will be more variable than in the longer term. For example an individual insectivore is likely to exploit highly abundant food sources in its local environment as and when they become available. An eruption of insects that occurs on one day may disappear the next, so that the presence of that food source in the diet may be very variable within and between individuals. In the longer term however one would expect that birds occupying similar environments to converge on similar dietary compositions. It is also clear that as time passes and seasons change, the number of different foods present in individual's diet will increase, as say starlings change from a largely invertebrate diet in summer to one including grain in the winter.
- If we are to generate data, particularly for probabilistic risk assessments, then it is important to try to obtain information on individual diets over the appropriate time- and spatial scales. In the literature however, information on dietary composition is typically reported as means across individuals and may or may not include values for variance. Although dietary composition tends to be estimated from crop, stomach or faecal contents and therefore represents food intake over a matter of hours, it is not often reported at time periods shorter than a month. As with data on food energy and moisture contents, this suggests that data from the literature on dietary composition is more suited to long-term risk assessments than short-term assessment. Detailed information on how diet varies between different habitats, especially between treated and untreated habitats is often missing.

Proportion of diet obtained from treated area (PD_j or PT)

For several small UK passerines CSL have data on likely short-term exposure to pesticides on food items collected in agricultural habitats. We have used radio-telemetry to measure active time spent in orchards by blackbirds, robins, chaffinches and blue tits; in the spring and summertime. In the arable landscape, we have similar data for skylarks, yellowhammers, blackbirds, linnets and

wood mice in winter and summer. We have assumed that active time spent in a given habitat will reflect the exposure to pesticides used there. For the most part these data have been collected over brief time periods in order to capture "a day in the life" of their foraging habits. Preliminary data are shown in Figure 1.

It is conceivable that each bird for which we collected "a day in the life" will continue to spend successive days locked into its particular routine (groundhog day scenario (Ferson, 2001)); a bird that spent all its time in barley on day 0 will continue to forage in only barley thereafter. At the other extreme (fresh start scenario), within the limits of experience shared by its species, an animal might venture out into a world made new each day. In reality, habitat use of individual animals will become more varied as observations accumulate and the particular choices of an individual will eventually converge toward the commonality of choices. Assuming similar habitats were available to all birds of a species, then in the long-term they may be expected to show similar habitat preferences. Average habitat utilization may be the same in both these scenarios but the variance between individuals will be much greater in groundhog scenario than in the fresh start scenario. Without knowing precisely how much "one day is much like another" we may need to conduct assessments using both extremes.

Conclusions and recommendations

The most obvious way in which long-term might differ from acute exposure assessments is in the greater need to take account of changing circumstances. Standard physico-chemical models may be able to describe changes such as the decay and dissipation of pesticides in the environment, but on their own such descriptions are likely to be increasingly inaccurate when biological agents enter the picture. Individual animals will not do exactly the same things from day to day, nor do they behave completely unpredictably. We know that the foods found by a skylark in June will not be the same in July and we may take account of such dietary shifts in our models. But we are still very ignorant of the day to day variations in individual diet and habitat choice. If our models are to capture the true variation in long-term risk then we need more information about the degree to which animals are creatures of habit. Failing that, we should try to encompass the bounds of the range exemplified by fixed or fickle behaviour. Failing that, if we prefer our risk assessments to be

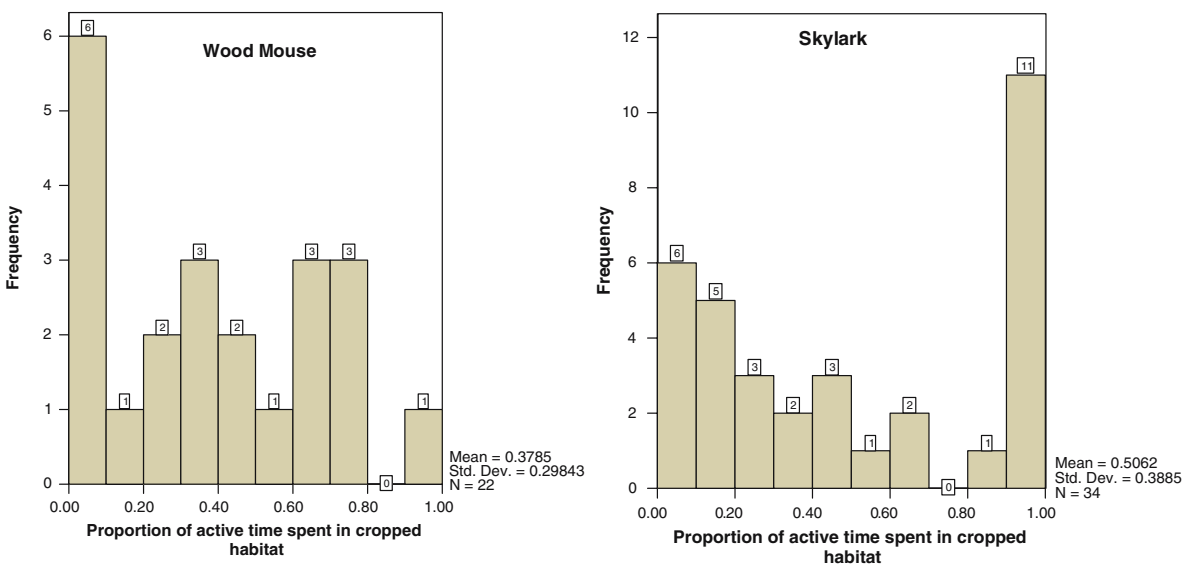

Figure 1. Distribution of active time, as indicated by radio-telemetry, spent by individual skylarks and wood mice in cropped habitats during their breeding seasons.

conservative, then we might assume that population of creatures at risk will consist of individuals which not only suffer extremes of exposure on one day but which continue to suffer the same fate every day (a fixed, serial autocorrelation of 1).

Circumstances vary through space as well as time. Not all fields are sprayed simultaneously and within an animal's normal foraging range both treated and untreated habitat may be available. We can try to take account of spatial variation by using actual pesticide usage data and cropping patterns to derive a distribution of pesticide in space and time that represents the full range (both typical and extreme) of usage in general UK agriculture. Or we can model a very specific typical arable landscape and simulate the outcome when we populate it with virtual skylarks or other species. Knowing what the outcome would be in less typical landscapes depends on having similarly detailed spatial and temporal information to adequately describe them.

Unresolved questions and some possible courses of action (*italics*):

- How to model non-independence of successive days exposure

  1. *Collect empirical data for scenarios of concern*
  2. *Compare extremes of dependence (serial autocorrelation of 0 versus 1)*
  3. *Choose most conservative extreme (serial autocorrelation of 1)*

- How to improve estimates of wildlife food intake based on DEE

  1. *Collect empirical data on wildlife food intake*
  2. *Collect empirical data on correlation between diet quality and energy expenditure*
  3. *Collect empirical data to estimate an upper limit for daily food consumption*
  4. *Collect empirical data on food quality (energy, moisture content) selected by species of concern in the field*

- How to model pesticide residue decline

  1. *Plants. Consider other models apart from first order kinetics*
  2. *Insects. Collect empirical data. Possible increases in residues with time*

- How to model spatial effects arising from wildlife habitat use, cropping and pesticide usage patterns

  1. *Collect data on wildlife use of arable habitat in long-term time scales*
  2. *Use spatially and temporally referenced pesticide usage data to provide scenario that is properly representative of the whole of the UK farming practice.*
  3. *Develop detailed spatially and temporally explicit standard scenarios to represent typical farming landscapes within of UK agriculture (agent-based modeling)*

- How to use information on pesticide avoidance in laboratory compared with field data.

  1. *Conduct research to elucidate the relationship between laboratory avoidance tests as currently constituted or proposed (CSL 1999) and likely effects in the field*
  2. *Conduct research on long-term avoidance (e.g. effects of learning)*
  3. *Consider alternative models to incorporate avoidance in dietary exposure assessments*

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