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ABSTRACT: Exposure, as well as toxicity, determines whether rodenticides present real environmental hazards to nontarget animals. In order to combine exposure and toxicity, a compartment model is proposed which distinguishes transfer processes from accumulation of residues. The published literature relevant to the model is analysed, and some important gaps in knowledge are highlighted. Simple sub-models of rat feeding behaviour and mortality are combined into a simulation model which generates data on both efficacy of control and build-up of residues in live rats and carcases. The roles of feeding parameters (e.g., palatability, availability of alternative food) as well as toxicity are emphasised by the simulation results.

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INTRODUCTION

In many countries, there is increasing concern about possible adverse environmental effects of pesticide use. Many registration authorities require some sort of evaluation of environmental risk before authorising or re-licensing use of a pesticide, and ideally this evaluation would involve objective assessment of the hazard that might be posed to a range of organisms exposed to a toxic compound, and whether the perceived risk of environmental damage outweighed the benefits of use of the toxin. In practice, toxicity data tend to outweigh other considerations, mainly because toxicity data are more easily and precisely estimated than exposure. In the United Kingdom, the brown rat Rattus norvegicus Berk, is the main rodent pest of agriculture, and control is based mainly on slow-acting, multiple-dose rodenticides. Warfarin is still widely used with surplus or sustained baiting, though the use of difenacoum, bromadiolone, brodifacoum and flocoumafen (called the second-generation anticoagulants) has increased since their introduction in the late 1970s and early 1980s.

Some authors have raised concerns about a higher acute toxicity of second-generation anticoagulant rodenticides to birds (Shawyer 1987). However, because the more toxic anticoagulants can be used to achieve effective rodent control with less frequent application over shorter time periods, it might be that the consequent reduction in exposure would outweigh their higher toxicity. Alternatively, the difference in exposure might be less important than the large difference in toxicity. Objective assessment of these ideas is not possible without examining the whole system and devising a means of quantifying hazards in an ecological context. Cox and Smith (1990) suggested that a compartment model of the ecotoxicology system might be of some use in evaluating both exposure and toxicity. We shall first describe a conceptual model of rodenticide ecotoxicology, then analyze the literature relevant to that model, and finally examine the properties of a simulation model based around control of the brown rat in the United Kingdom.

RODENTICIDE ECOTOXICOLOGY SYSTEM

A generalised compartment model (Fig. 1) describing the movement of a toxic compound through the environment was proposed by Cox and Smith (1990). There are two distinct components at the heart of the system: the levels of toxin in the species which make up the compartments, and the transfer processes which describe the movement of toxins between compartments. In general, it is easier to obtain data on laboratory toxicity and on the toxin levels in the compartments than on the dynamics of the transfer processes, and registration authorities generally place most emphasis on laboratory toxicity and residues found in the field when evaluating potential ecological hazards of pesticides.

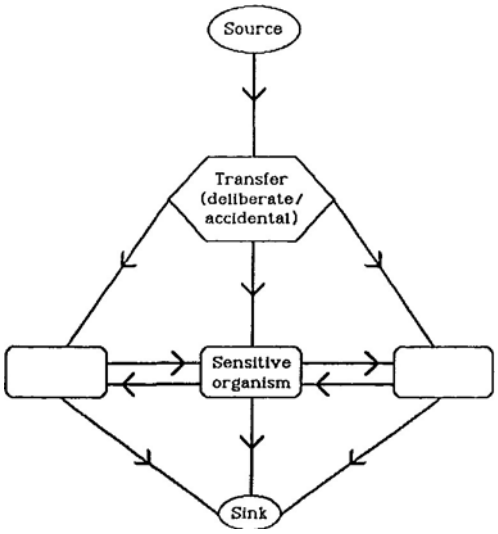

Figure 1. General ecotoxicology model.

The generalised model can be developed into a system more specific to rodent control (Fig. 2). Nontarget species which may be at risk are of three types:

1. nontarget bait feeders which risk primary poisoning by consuming bait (e.g., small mammals, granivorous birds).
2. predators which risk secondary poisoning by eating target rodents, nontarget small mammals or birds (e.g., foxes, owls).
3. scavengers which risk secondary (or tertiary) poisoning by eating dead bodies of intoxicated animals of any type.

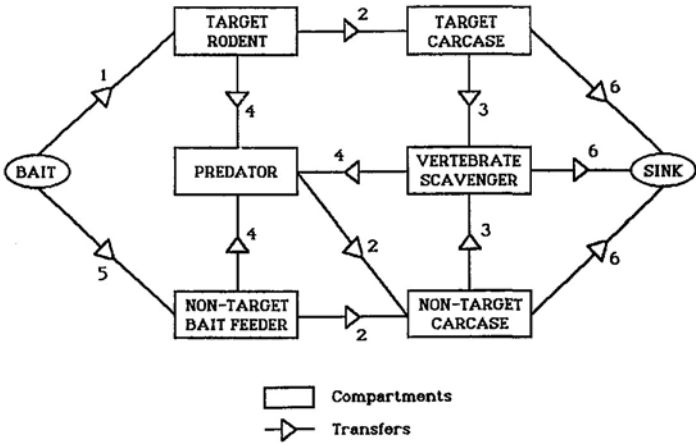

Figure 2. Conceptual model of rodenticide ecotoxicology.

Invertebrate scavengers (e.g., blowflies) and excretion in faeces and urine contribute to the "Sink" and will not be considered in the model.

Although oversimplified, the compartment model in Figure 2 is already complex. Each transfer function will be different according to the species and toxins involved, and Hone (1986) in modeling only two of the transfers (feeding and death of target rodents) invoked 15 to 17 control parameters. Clearly, in considering the ecotoxicology system, we have to reduce the system to its qualitative elements in order to show where data are lacking before we can hope to simulate toxin movement with any degree of accuracy. Our approach here is to reduce the transfer processes to a small number which we can represent with a manageable set of parameters. First, we will summarise the relevance of the published literature on transfer and accumulation of anticoagulant rodenticides in relation to Figure 2. The six transfer processes that we will consider are as follows:

1. primary feeding on poison bait by the target rodents,
2. mortality due to primary and secondary poisoning of targets and nontargets,
3. feeding on carcasses by vertebrate scavengers,
4. predation of target and nontarget vertebrates,
5. primary feeding on poison bait by nontarget vertebrates,
6. transfer of poison to soil from carcasses.

Table 1 shows that some processes (e.g., mortality, predation) are covered quite well, whereas others (e.g., transfer to the soil) are hardly covered at all.

A MODEL OF TRANSFER AND ACCUMULATION

There are several approaches that could be adopted to modelling Figure 2. We will first consider a simple probabilistic model of feeding and then incorporate mortality to produce a simple model which we believe is at least conceptually useful and may shed some light on, for example, the relative hazards of different baiting systems, bait concentration, or levels of toxicity.

Although the frequency distribution of rodents carrying different levels of rodenticide is of primary interest in assessing hazard to predators and scavengers, there are few examples of published data on residue analysis in live-trapped rats and carcasses. Dubock (1984) contrasts the distribution of brodifacoum residues in carcasses of dead R. norvegicus Berk, collected following pulsed baiting and saturation baiting with 0.005% brodifacoum bait on UK farms. The mean levels of rodenticide residue were lower with pulsed baiting (1.4 mg/kg) than saturation baiting (3.2 mg/kg); the distributions were different in that saturation baiting produced a higher frequency of carcasses with higher residues, including 8% (2/26) with residues above 10 mg/kg body weight. These and similar data reported by Hoque et al. (1987) and Johnson and Scott (1986), however, are the outcome of two transfer processes (feeding and mortality), and may be affected by a third transfer process (predation) if predation is not random with respect to rodenticide level. We will now attempt to model the two transfer processes producing target residues.

Simple Probabilistic Model of Feeding

The primary determinant of rodenticide level in an individual animal (target or nontarget) is feeding. Of particular interest are a neophobic response to bait, the probability of feeding on bait on successive occasions, and the effect of intoxication on feeding. There are few data against which to compare predictions. Buckle et al. (1986) used chemical bait markers to study bait uptake in R. norvegicus on two UK farms. In a 4-day baiting period, it was possible to determine how many rats in a trapped sample had fed 0, 1, 2, 3 or 4 times (Table 2a), and we will use their data to test the predictions of two models.

(a) Simple binomial-In a simple binomial model, the probability p that an animal feeds from bait on a given night is independent of whether that animal feeds on any other night and of the behaviour of other animals. The frequency distribution of the number of times an animal feeds from bait in the four nights is given by the terms of the binomial expansion $(p + q)^4$ where $q = 1-p$ is the probability of not feeding on any given night. Out of 600 rat-nights in the data of Buckle et al. (1986), there were 416 rat-nights when animals fed, giving an estimate of $p = 0.693$. The expected frequencies generated by the simple binomial are given in Table 2b and the model clearly does not fit (combining groups 0 and 1, $X^2_{(4)} = 152$; $P<0.001$). The model fails because there were too many rats which fed not at all or on all four nights.

(b) Binomial neophobia-In what we call "binomial neophobia" the probability that an animal feeds on bait for the first time on any given night is p, and the probability that it feeds on bait on each successive night is r>>p. For simplicity, we first assume that r = 1, that is, neophobia is overcome completely to the extent that an animal becomes "bait-happy" once it has eaten bait. A crude estimate of p can be obtained by noting that the proportion of animals which feed on all four nights is p while the proportion of those which do not feed at all is $(1-p)^4$. Setting $p = 0.5$ gives expected proportions of 0.5 and 0.0625 for animals which feed on bait four or zero times, respectively, corresponding well with the data (Table 2a). The proportion of animals feeding on n out of 4 days is $p(1-p)^{(4-n)}$ for $n>0$ and $(1-p)^4$ for $n = 0$. The expected numbers in each of the five feeding categories are shown in Table 2b. Although this model clearly underestimates the number of animals which feed on bait once only and in consequence does not fit in detail ($X^2_{(4)} = 63.7$; $P<0.001$),

on bait (a) on no nights, (b) on some but not all nights, or (c) on every night, the predictions are remarkable good ($X^2_{(2)}$ = 0.05; P>0.95). We shall therefore use the binomial neophobia model of feeding as a first approximation because it has a minimum number of parameters and accurately predicts the main features of the only relevant published data.

Table 1. Analysis of literature relevant to the rodenticide ecotoxicology compartment model. See Table for key to transfer processes (1-6) and compartments (A-F).

	Transfers						Compartments[a]					
Publication	1	2	3	4	5	6	A	B	C	D	E	F
Askham (1986)						X						
Balcomb (1986)			X									
Brown et al. (1988)							X	X		X	X	
Buckle et al. (1986)	X											
Bunn et al. (1982)				X								
Butcher (1965)			X									
Colvin (1984)				X								
Cox and Smith (1990)	X	X			X							
Dubock (1984)		X						X	X			
Dubock (1986)		X						X				
Duckett (1984)				X								
Edwards et al. (1988)			X	X	X					X		
Erlinge (1975)				X								
Fink and Jaber (1981)												X
Glue (1974)				X								
Godfrey (1985)		X			X	X						
Grand (1976)		X										
Grolleau (1983)		X										
Hegdal et al. (1981)		X									X	
Hegdal et al. (1984)				X								
Hegdal and Blaskiewicz (1984)				X							X	
Hegdal and Colvin (1988)				X							X	
Hoque et al. (1987)					X		X	X				
Huckle et al. (1988)							X					
Huckle et al. (1989)						X	X					
Johnson and Scott (1986)					X		X	X				
Kaukeinen (1982)							X					
Kotler et al. (1988)				X								
Lorgue (1989)		X			X							
Lorgue et al. (1986)		X			X							
Lund (1981)		X										
Mendenhall and Pank (1980)		X										
Metzgar (1967)				X								
Myllymaki (1984)		X										
North (1985)				X								
Papworth (1958)		X										
Parmar et al. (1987)							X					
Rudebeck (1951)				X								
Saxena and Sharma (1984)		X										
Southern (1954)				X								
Southern and Lowe (1968)				X								
Taylor et al. (1968)		X			X						X	
Townsend et al. (1981)		X		X								X
Townsend et al. (1984)		X									X	
Totals	2	17	3	16	8	3	7	5	1	3	6	1

[a]A = target rodent, B = target carcass, C = nontarget bait feeder, D = nontarget carcass, E = predator, F = vertebrate scavenger.

Table 2. Data on the number of nights out of four nights of successive baiting that rats fed from bait-points in two U.K. farm trials (Buckle et al. 1986). The data from the two farms are homogeneous (test of heterogeneity: $X^2 = 3.23$, 4 d.f.; $P > 0.10$).

2a. Number of nights rats were feeding.

Number	Farm 1	Farm 2	Total	%
0	6	4	10	6.7
1	11	21	32	21.3
2	4	10	14	9.3
3	7	13	20	13.3
4	24	50	74	49.3
Total	52	98	150	100%

2b. Values predicted by models compared with data.

Number	Observed	Expected	
		Binomial	Neophobia
0	10	1.3	9.4
1	32	12.0	9.4
2	14	40.7	18.7
3	20	61.3	37.5
4	74	34.7	75.0

Mortality Model

There is a much larger set of literature on mortality than on feeding. All rodenticides are extensively tested against a range of target and nontarget species in laboratory toxicity tests, the conceptual basis of which is that the dose of toxin received by an animal affects the probability that it will die. Unfortunately, it is not straightforward to incorporate such data into a mathematical model representing Figure 2. Acute toxicity differs to a greater or lesser extent from chronic toxicity (Ashton et al. 1986) and for anticoagulants there is a variable time-delay between ingestion of a lethal dose and onset of lethal symptoms. Here we will concentrate on developing a simple sub-model of mortality which is specific to anticoagulant poisons and again uses a minimal number of parameters.

Once an individual rodent has consumed a lethal dose of any anticoagulant, it progresses through a series of physiological states before it dies. We will represent the progression by a series of compartments, for each of which a probability is specified that on the next day an animal will progress to the next (as opposed to stay in the same) compartment (Table 3a). Since it is believed that all anticoagulants act in the same way, our model is independent of the particular compound once an animal has been lethally dosed. The transition probabilities listed in Table 3a were not estimated from a particular set of data, but are simply numbers chosen because they are consistent with experience and generate a realistic distribution of time to death (Table 3b). According to the model, no animals die until four days after consuming a lethal dose (though in reality, a few might die sooner), the median time to death is 4 to 5 days, and less than 5% of animals survive beyond 8 days. The device that we use of setting up several compartments with equal rates of transfer has been used to model distributed developmental time-delays in population dynamics (Smith and Mead 1974) and generates a unimodal gamma distribution (the special Erlangian) for the total time between lethal dose and haemorrhage (Cox 1962).

Table 3. Mortality model

3a. Daily transition probabilities.

State	Transition probabilities	
	Same state	Next state
Lethally dosed	0.1	0.9
Clotting factors down	0.1	0.9
Increased clotting time	0.1	0.9
Haemorrhage	0.5	0.5
Death	1	0

3b. State changes in time following lethal dose.

Day	% haemorrhage	% dead
0-2	0	0
3	73	0
4	58	36
5	34	66
6	18	82
7	9	91
8	4	96

Having specified a structural model of mortality, the only parameters which are compound-specific and determine entry to the mortality sub-model are the median toxicity (LD_{50}) and the variability of response (the slope of the dose-response curve); these parameters determine whether an individual is lethally dosed or not, having consumed a given quantity of poison bait.

COMPUTER SIMULATION OF RESIDUE ACCUMULATION

In order to predict the likely exposure of predators and scavengers to rats carrying rodenticide, we simulated the build-up of anticoagulant rodenticide in both live rats and carcasses. As noted in the Introduction, it could be that a more toxic compound might present less hazard than a compound with lower toxicity if rapid and effective rodent control resulted in much reduced exposure. In our simulations, we therefore compared two hypothetical compounds A and B with toxicities defined as follows: A) a first-generation anticoagulant with a multiple-dose oral LD_{50} of 25 mg/kg, and B) a more toxic, second-generation anticoagulant with an oral LD_{50} of 0.25 mg/kg. In both cases, the concentration of active ingredient in bait was set at 50 mg/kg. This defines A as a multiple-feed poison (an animal must consume bait amounting to half its body weight to take in one LD_{50}), while B is potentially a single-feed poison (bait amounting to less than 1% of its body weight contains one LD_{50}).

Feeding

We assumed provision of excess bait (surplus or saturation baiting) throughout the simulated control operation (up to 25 days). The feeding sub-model was "binomial neophobia" with p = 0.5 and r = 1 as defined previously. For each neophobic individual in the population on each day, a random number x was drawn from a uniform distribution between 0 and 1, and the rat moved from the "neophobic" state to the "feeding" state if x > 0.5.

Daily food intake was fixed as a proportion of individual body weight. Once in the feeding state, on each day an animal took a proportion of its daily food intake from poisoned bait (given average values of 10% or 30% in our simulations). We also introduced some daily variability in individual bait-take. An individual took the average value with probability 0.8, half the average with probability 0.1, or twice the average with probability 0.1; thus the average values of 10 or 30% represent geometric rather than arithmetic means.

Lethal Dosing and Elimination

The dose that would be just sufficient to kill an individual was assumed to be approximately normally distributed with a mean equal to the LD_{50} specified and a standard deviation set at 10% of the mean; this generates a much steeper dose-response curve for the more toxic compound B than for A, which is in accord with general experience. For a given residue level, the dose-response curve specifies the probability s that an individual animal will move from the "feeding" to the "lethally dosed" state (Table 3a). A random number x uniformly distributed between 0 and 1 was generated for each "feeding" individual each day to determine whether transition to "lethally dosed" would take place (if s > x). Transitions between the different states in Table 3a were similarly determined by whether a random number x was less than a transition probability.

Not all rodenticide consumed is absorbed and retained. First-generation anticoagulants such as warfarin are in part excreted unchanged and also metabolised by various species (e.g., Townsend et al. 1981). Second-generation anti-coagulants such as flocoumafen are characterised by a biphasic elimination in rats, with substantial excretion of unmetabolised compound in the first few days after ingestion followed by much slower elimination of a very small quantity of compound from a specific binding site in the liver (Parmar et al. 1987, Huckle et al. 1988, Huckle et al. 1989). In our simulations, we approximated these processes by assuming 30% elimination of residue each day, which corresponds to a realistic two-day half-life of residue in the rat.

Elimination was assumed to carry on unchanged when an animal was progressing through the different stages of anticoagulant poisoning prior to death. Feeding was reduced to zero in about 4 days from the entry into the "lethally dosed."

Table 4. Computer simulation results

Control of rat populations (100 individuals) was simulated using the feeding and mortality sub-models specified in the text for two compounds with contrasting LD_{50} values: A. 25 mg/kg, B. 0.25 mg/kg. Animals do not start feeding on bait until they overcome their initial neophobia (the second day, on average). The "mean lag, feeding-death" is the mean number of days between when an animal starts to feed on bait and when it dies. The rodenticide residue levels are determined by the amount of bait consumed and the elimination of compound (30% per day).

LD_{50} (mg/kg)	25		0.25	
Bait as % daily diet	30%	10%	30%	10%
% dead after 25 days	100%	89%	100%	100%
Mean lag feeding-death (days)	10.69	17.91	5.05	5.50
(standard deviation)	(1.99)	(2.39)	(1.25)	(1.54)
Mean carcass residue (mg/kg)	15.10	10.21	5.11	1.47
(standard deviation)	(7.29)	(5.20)	(2.37)	(0.66)
Maximum carcass residue (mg/kg)	35.36	31.49	12.16	3.11
Mean residue, living (mg/kg)	-	8.29	-	-
(standard deviation)	-	(0.80)	-	-

Results

Table 4 summarises the main results of the computer simulations. Each simulation dealt with a population of 100 rats. Since the simulations were in part stochastic (with degrees of randomness in feeding and mortality sub-models), five replicate simulations were run. However, the results of replicates were so similar that only the first replicate of each of the four parameter combinations is presented here. The main features of Table 4 are discussed below.

DISCUSSION

In this paper, we have attempted to look at rodenticide ecotoxicology as a system, and in our analysis we found it useful to distinguish between the parts of the system where rodenticide might accumulate and the transfer processes that lead to accumulation (Figs. 1, 2). In doing this, we hoped to emphasise the ecological context of toxicity data on which registration authorities understandably place so much emphasis.

Our literature survey (Table 1) revealed a paucity of published information in some areas of our compartment model (Fig. 2), and we therefore decided to concentrate on establishing realistic sub-models which we could put together in order to predict rodenticide residue levels in live rodents and carcasses. We chose to concentrate on brown rats in the United Kingdom because most of our work is in this area, and there is a reasonable body of information about this system. In our simulations, we compared two hypothetical anticoagulant rodenticides with differing toxicities (A. LD_{50} = 25 mg/kg; B. LD_{50} = 0.25 mg/kg in brown rats). The remainder of this Discussion is about the simulation results summarised in Table 4, and how we intend to continue with this approach.

In our simulations, considering efficacy first it is clear (and not surprising) that the more toxic compound B achieved surer and faster control than compound A. Indeed, when bait amounted to 10% of total daily food intake, compound A with LD_{50} = 25 mg/kg achieved only 89% control in 25 days of surplus baiting. Compound B with LD_{50} = 0.25 mg/kg killed most rats within one week of starting baiting whether bait was 30% or only 10% of total daily food intake, and within the range of parameter values used here was truly a "single-feed" poison. The lower toxicity of compound A led to most animals dying between 2 and 3 weeks after starting baiting.

Turning to the carcass residue levels, for the more toxic compound B, most dead rats (99%) had residues higher than the LD_{50} as an inevitable consequence of the delayed action of anticoagulants. In our simulations, animals carried on feeding after consuming a lethal dose and indeed up until death, and we will have exaggerated the carcass residue levels if (as we have observed in the laboratory) animals go off eating as symptoms of haemorrhage appear. Nevertheless, the results of the simulations for compound B are remarkably close to the mean carcass residue level of 3.2 mg/kg reported by Dubock (1984) for a saturation baiting treatment with brodifacoum, generally reckoned to have an LD_{50} of 0.26 mg/kg. This gives us some confidence in the ability of our model to predict realistically the transfer and accumulation of anticoagulant rodenticides.

We had to assume what seemed to us to be reasonable values of bait intake (10% or 30%). Reliable field data on bait consumption by individuals as a proportion of their total daily food intake are not available, but clearly the proportion is related to bait palatability as well as to the availability and palatability of alternative food. Under the assumptions of our model, it seems that the concentration of the active ingredient of compound B in bait could be reduced well below 50 mg/kg in order to lower mean carcass residue levels without adversely affecting efficacy, though efficacy might then be reduced if average bait consumption was substantially less than 10% of daily food intake or there was more variability than assumed in the dose-response curve.

Although the residue levels were substantially higher for compound A, they were not increased in proportion to the l00x higher LD_{50} value. Most carcass residues were less than the LD_{50} because, in contrast with the single-feed poison B, 30% elimination per day was not outweighed by accumulation through continued feeding (in our model, during the 4 or more days between consumption of a lethal dose and death, more than 75% of that lethal dose would be eliminated). Thus, if residues were measured as rat LD_{50} equivalents, the carcass residues for compound B could represent a greater potential hazard to scavengers, depending on exposure (if most rats die underground, there will be very little exposure for either compound).

However, the story might be different for predators. Predator exposure to hazard depends very much on the time lag between when a rat starts to feed on bait and when it dies; for compound A, this lag was doubled (10.7 days) or more than trebled (17.9 days) compared with compound B (5.0 or 5.5 days) and, if bait consumption was low (10%), more than a tenth of the rats in our simulation would be wandering around alive with substantial levels of compound A more than 25 days after the start of control.

Whether there is a real risk of secondary poisoning to either predators or scavengers depends on their feeding behaviour, and how they may respond to the presence of rats at different stages of poisoning. We are currently investigating experimentally changes in various aspects of rat behaviour after anticoagulant poisoning, and we hope soon to be able to incorporate realistic representations of the transfer functions 3 and 4 in Figure 2 in order to be able to predict residue levels accumulating in predators and scavengers. Using our existing model, we will first examine the effects of different baiting strategies (pulsed baiting and permanent bait-points, compared with fixed period saturation baiting), and also of behavioural exclusion of subdominant rats from bait-points (Dubock 1982, 1984) for which we have recently found support in a field experiment (Cox and Smith 1990, Fig. 4). Also, we must carry out a fuller sensitivity analysis to discover which parameter estimates are the most critical.

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