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Modelling of poisoning for vertebrate pest control, with emphasis on poisoning feral pigs

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ABSTRACT

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Compartment models of poisoning vertebrate pests are described. The models estimate changes in the number of pests in each of three compartments; susceptible, eating poisoned bait and showing poison signs. The general models were applied to a field poisoning of feral pigs with warfarin. Parameters in the models were estimated from independent pen data. A variation on the basic model incorporating a maximum feeding rate is also described. Application of the models to planning and evaluation of poisoning is discussed.

INTRODUCTION

There have been few attempts to integrate the many patterns and processes which are involved when controlling vertebrate pests and to express the results in a mathematical form or model. Gentry (1971) developed a model of programs for eradication of rats. The model was based on a series of simultaneous integral equations which described changes in the number of rats of different ages. Natural changes in abundance were described and the effects of sterilisation and poisoning examined. Carpenter (1981) modelled the effects of chemical control on a population as a source of mortality additive to that of natural and disease-induced mortality. The basic model used differential equations to describe rates of change of disease status of a host population. Batcheler (1982)

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developed a simple probability model to estimate the number of random encounters with poisoned bait that were required to kill a pest. This was based on the poison content and piece-weight distribution of poisoned baits. Grant et al. (1984) described a Leslie matrix model to evaluate the effects of pesticides on non-target populations. The effects of four hypothetical control programs on coyote (*Canis latrans*) abundance were simulated.

Hone (1986) developed probabilistic models which estimated the probability of an animal dying in a poisoning program. The probabilistic approach reflected underlying uncertainty in describing the effects of all factors and interactions that may determine how many or what percentage of animals die. The models estimated the probability of an animal dying as the product of the probability of an animal eating the poisoned bait and the probability of dying given that the animal has eaten the poisoned bait. Those models prescribed relationships such as the dose–response curve, between factors. In this paper an alternative approach is used. Models are developed which describe temporal changes in a pest population during poisoning.

The models developed here were formulated by examining the similarities and differences between an infectious disease spreading through an initially susceptible population and the introduction and spread of poison through a pest population. The similarity between disease and poison spreading through a susceptible population was noted, independently, by Halpin (1975). Many mathematical models in ecology and epidemiology are based on dividing a population into segments or compartments. The rates of change of individuals between compartments are then described by differential or finite difference equations. Such an approach has been used widely in epidemiology (Bailey, 1975; Anderson and May, 1979; Anderson, 1981; Jones and Sleeman, 1983), though was not described by Halpin (1975).

A susceptible population can become infected with a disease, then infectious and possibly later an immune population. Each of these compartments is analogous to susceptible, poisoned and recovered individuals of a vertebrate pest population. The poisoning model is clearly different to disease models where disease can be transmitted from one host individual directly to another. Individual pests contract the poison by contact with poisoned bait, not by contact with infectious (poisoned) individuals. Hence a basic assumption of disease models needs to be changed. The poisoning situation is more analogous to infection of a host population from the environment, not from other hosts. Anderson (1981), Anderson and May (1980, 1981) and Hochberg (1989) described such models for infectious diseases.

Poisoning is a common method of control of feral pigs (*Sus scrofa*) in Australia (Tisdell, 1982). A widely used poison is sodium monofluoroacetate (1080) which has been evaluated in pen (McIlroy, 1983; Hone and Kleba, 1984; O'Brien et al., 1986, 1988; O'Brien, 1988) and field experiments (Hone and Pedersen, 1980; Hone, 1983; Bryant et al., 1984; O'Brien and Lukins, 1988). Problems with the acceptability and toxicity of 1080 have led to pen (Hone and Kleba, 1984; O'Brien and Lukins, 1990) and field (Hone, 1987; Saunders, 1988; McIlroy et al., 1989; Hone and Stone, 1989; Choquenot et al., 1990, Saunders et al., 1990) assessment of an alternative poison, warfarin.

The aims of this paper are to develop models of poisoning, examine the sensitivity of the predictions and compare predictions of the models with results from the field-use of warfarin for control of populations of feral pig. The variability of predictions is examined by sensitivity analysis.

MODELLING

In a classic model of contagious disease the rate of change of the susceptible population is assumed to be proportional to the product of the number of susceptible individuals and the number of infected individuals (Bailey, 1975; Anderson, 1981). The latter occurs because infected individuals can spread the pathogen from one infected individual to another without going back to the source of infection. An analogous situation occurs in poisoning vertebrate pests when individuals are poisoned by ingesting part or all of another individual that has been poisoned and died. This is called secondary poisoning.

In conventional poisoning, assuming secondary poisoning does not occur, the change in the number of susceptibles (X) is proportional to the product of the number of susceptibles (X) and the amount of poison in the environment (W). The rate of change of susceptibles is independent of how many individuals have been poisoned or are dead. Anderson (1981), Anderson and May (1980, 1981) and Hochberg (1989) described a similar relationship for infection with a pathogen which is in the environment of the host. This is the central assumption in the first model (Model A) described here. The assumption is varied in Model B.

Model A: the basic model

The process of poisoning feral pigs usually occurs in two steps. Initially feral pigs are offered non-poisoned bait. This step is also called free-feeding or pre-baiting. When removal of bait by feral pigs has reached a high and stable level, then the second step is instigated which involves switching poisoned for the non-poisoned bait.

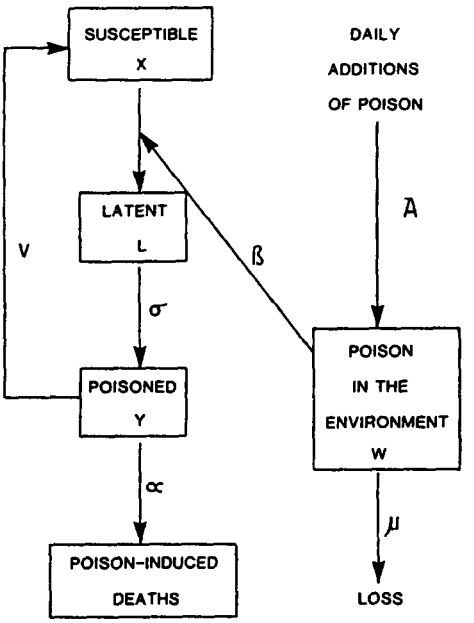

Fig. 1. Compartment model of poisoning a vertebrate pest population. Arrows show direction of transfer and associated Greek symbols indicate per capita rates of transfer per unit time. Symbols in boxes show actual numbers of animals, and are those susceptible animals (X), eating poisoned bait (L) and showing signs of poisoning (Y). The number of lethal doses of poison in the environment is W.

When poisoned bait is offered, susceptible animals can become poisoned (L) at a per capita rate β, and after a latent period ($1/\sigma$), during which poison signs are not apparent, they show signs (Y) (Fig. 1). Animals showing signs can die at a per capita rate α, or recover at a per capita rate v, and again become susceptible. There is no group of "immune" animals. During poisoning, animals in each compartment can die naturally at a per capita rate b, or births can occur at a per capita rate a. The rates of change are assumed to be constant. The amount of poison in the environment (W) is increased by additions (A) and decreases because of intake by pests (N) and by loss from weathering at a per capita rate μ.

The change in the number of individuals in compartments in a feral pig population can be described by a series of differential equations. The unit of time is one day.

$$dX/dt = aN - bX - \beta XW + vY \quad (1)$$

$$dL/dt = \beta XW - (b + \sigma)L \quad (2)$$

$$dY/dt = \sigma L - (b + \alpha + v)Y \quad (3)$$

$$dW/dt = A - (\beta N + \mu)W \quad (4)$$

Classic models of infectious diseases predict a threshold abundance of susceptible hosts below which the pathogen becomes extinct (Bailey, 1975; Anderson, 1981). The model derived here has no such threshold pest abundance. However, during protracted poisoning campaigns, there is an equilibrium ratio of the number of susceptible animals to that of the number of poisoned animals. The ratio occurs when $dX/dt = dL/dt = dY/dt = dW/dt = 0$. The ratio is:

$$\frac{X}{Y} = \frac{(b+\sigma)(b+\alpha+\upsilon)(\beta N+\mu)}{\sigma\beta A} \tag{5}$$

A strategic aim of poisoning is to lower the ratio to a minimum by reducing X. That can be achieved by, for example, increasing the rate of addition of poison (A). If an aim is to have fewer susceptibles (X) than infectives (Y), then $X/Y < 1$, therefore by rearranging, the amount of poison added daily must satisfy:

$$A_c > \frac{(b+\sigma)(b+\alpha+\upsilon)(\beta N+\mu)}{\sigma\beta} \tag{6}$$

Hence there is a daily threshold rate of pesticide addition (A_c) that must be used to control a pest population.

An alternative aim is to maximise the number of pests that die from poisoning. Rearranging equation (5) for the estimate of the number of dead pests (αY) gives:

$$\alpha Y = \frac{\sigma\beta XA}{(b+\sigma)(\beta N+\mu)} - (b+\upsilon)Y \tag{7}$$

Equation (7) simply says that to maximise the number of poison-induced deaths, then the number of pests that pass from susceptible to poisoned with signs $[\sigma\beta XA/(b+\sigma)(\beta N+\mu)]$ must be a maximum relative to the number of pests poisoned with signs that die naturally or recover $[(b+\upsilon)Y]$.

Model B: saturation of transmission

Pest animals, such as mammals and birds, have a maximum rate at which they can eat food, such as poisoned bait. This is described in the functional response relationship of predator–prey theory (Holling, 1959; Begon et al., 1986). Historically a linear functional response was assumed wherein food intake-per predator increased as food availability increased.

Modifications of this recognise a maximum feeding capacity, or the effects of alternative prey, to generate curvilinear functional responses (Holling, 1959; May, 1981; Begon et al., 1986).

The linear functional response assumes that the rate of change, per predator, in prey intake, is directly proportional to prey availability. This is analogous to the assumption in Model A that the rate of change in susceptibles, per susceptible, is directly proportional to the abundance of infectives in the environment. The proportionality constant is the transmission coefficient. As animals have maximum rates of food intake they must have maximum rates of poisoned bait intake, so the assumption in Model A needs to include such a maximum. May and Anderson (1979) described a similar phenomenon in disease models as transmission saturation occurs. In the poison model here (Model B), the rate of change of susceptibles, per susceptible, is proportional to the number of available infectives in the environment, and not the absolute number of infectives in the environment (W). One possible mathematical formulation of that is that the rate of change of susceptibles is $BXW/(1+cW)$. When $c=0$, then the transmission term reduces to BXW, in which case Model B is the same as Model A as $B=\beta$.

The equations for Model B are:

$$dX/dt = aN - bX - BXW/(1+cW) + \upsilon Y \tag{8}$$

$$dL/dt = BXW/(1+cW) - (b+\sigma)L \tag{9}$$

$$dY/dt = \sigma L - (b+\alpha+\upsilon)Y \tag{10}$$

$$dW/dt = A - \{[BN/(1+cW)] + \mu\}W \tag{11}$$

At equilibrium:

$$\frac{X}{Y} = \frac{(b+\sigma)(b+\alpha+\upsilon)[BN+\mu(1+cW)]}{\sigma BA} \tag{12}$$

When $c=0$, the equilibrium ratio of Model B reduces to that of Model A (equation 5). The effect of the transmission saturation coefficient (c) is to increase the threshold level of poison to be added daily.

APPLICATION

Field situations and modifications to the basic model

The models can be applied in different field situations. If the free-feed period is not used then obviously only the second part of the model is appropriate. If a latent period is absent, such as with cyanide, then $1/\sigma = 0$ and $L=0$ so the equation for the rate of change of poisoned animals (dY/dt) changes to include the transmission term. If poison bait is left available to pest animals then the existing models can be used. If however the poisoned bait is removed after t days then for time periods after that,

the transmission rate (β) equals 0. If bait shyness (that is an aversion to eat the poisoned bait) occurs then the rate of recovery (υ) approaches or equals zero. If births and natural deaths are absent, as in a non-breeding season, or the poisoning campaign is short, then $a = b = 0$. Inhibition of reproduction by a poison results in no births for that segment (Y) of the population and so $a = 0$ for that segment. If pesticide resistance occurs then the mortality rate (α) is a negative function of time.

Application

Predictions of the models were compared with results of a field poisoning of feral pigs using bait poisoned with warfarin. The poisoning occurred in Namadgi National Park (35°30'S, 149°E), Australia, in May 1986, as part of control of feral pigs and their sign (Hone and Stone, 1989) and research into feral pig control during simulated outbreaks of exotic livestock diseases (McIlroy et al., 1989). Details of the site were given by Hone (1988), Hone and Stone (1989) and McIlroy et al. (1989), and details of the poisoning described by McIlroy et al. (1989). Predicted results were compared with observed results using the known deaths among 32 feral pigs that had been fitted with radio-transmitters prior to the poisoning, as described by McIlroy et al. (1989).

The models assumed no secondary poisoning of feral pigs would occur with warfarin, and that ingestion of poison did not cause rapid behavioural changes to scare off other feral pigs. The concentration of warfarin in the wheat bait was 0.2%. Free-feeding with bread and wheat occurred for several days and poisoned wheat was left available for the pigs. Additional poisoned bait was distributed when bait previously distributed was eaten by the pigs. Bait shyness was assumed to be absent based on results of Hone and Kleba (1984). For the short duration (3 weeks) of the poisoning, births and natural deaths were assumed to be zero ($a = b = 0$). It was also assumed that resistance to warfarin did not develop during the poisoning and that no portion of the population was isolated. The latter was based on the very wide distribution of bait and no prior use of warfarin in the area.

Parameter estimation

Population density of feral pigs was estimated to be 1.8 pigs/km^2 (McIlroy et al., 1989). The average time to development of signs of poisoning was assumed to be 3 days based on pen experiments (Hone and Kleba, 1984). Hence, $\sigma = 1/3 = 0.333$ day^{-1}. The rate at which feral pigs died from warfarin (α) was estimated from the inverse of the average time from appearance of signs till death and the proportion of feral pigs killed

by the poison at the concentration to be used. The average time till death was assumed to be 4 days and the proportion dying 0.92 (Hone and Kleba, 1984). The proportional kill is converted to an instantaneous rate then divided by the time. Hence the mortality rate (α) is $[-\log(1-0.92)]/4 = 0.274\ \text{day}^{-1}$. The mortality for both sexes is combined, though Hone and Kleba (1984) reported males were significantly more tolerant than females. The rate at which pigs recovered and became susceptible (υ) was assumed to be zero in the field study.

The rate of change (β) from susceptible to eating the poisoned bait was assumed initially to be 0.01 $\text{km}^2\ \text{day}^{-1}$. There was no empirical a priori estimate of the transmission coefficient so a range of values was investigated by sensitivity analysis. The amount of poison in the environment (W) on day 1 of poisoning was assumed to be 100 lethal doses km^{-2} as McIlroy et al. (1989) did not report the actual amount. Poison bait was always provided in ad libitum amounts. During the field study poison bait was added each day, though McIlroy et al. (1989) did not report the amount. For the simulations the daily weight of poison bait added (A) was assumed initially to be 0 lethal doses km^{-2}. The rate of loss of poison in the environment as a result of weathering (μ) was assumed to be zero.

The equations for Model A were:

$$\mathrm{d}X/\mathrm{d}t = -0.01XW \tag{13}$$

$$\mathrm{d}L/\mathrm{d}t = 0.01XW - 0.333L \tag{14}$$

$$\mathrm{d}Y/\mathrm{d}t = 0.333L - 0.274Y \tag{15}$$

$$\mathrm{d}W/\mathrm{d}t = -0.01NW \tag{16}$$

The equations for Model B with the same parameter estimates as in Model A, and the transmission saturation coefficient (c) of 0.01, were:

$$\mathrm{d}X/\mathrm{d}t = -0.01XW/(1+0.01W) \tag{17}$$

$$\mathrm{d}L/\mathrm{d}t = 0.01XW/(1+0.01W) - 0.333L \tag{18}$$

$$\mathrm{d}Y/\mathrm{d}t = 0.333L - 0.274Y \tag{19}$$

$$\mathrm{d}W/\mathrm{d}t = -[0.01N/(1+0.01W)]W \tag{20}$$

Sensitivity analysis

The sensitivity of the predicted results to changes in model parameters was examined by successively varying the value of each parameter and rerunning the model. The transmission coefficient was successively halved to simulate slower removal of poisoned bait to examine the effects. The mortality rate was reduced to correspond to deaths occurring over a longer

time period, and the saturation coefficient was increased from zero, to correspond to a lowering in intake of poisoned bait. The daily rate of addition of poisoned bait was varied from zero to 50 lethal doses km^{-2}.

RESULTS

Predictions

Model A

After poisoned bait was offered, pigs acquired the poison and the percentage of pigs poisoned increased and the percentage of pigs still alive decreased after 2 days (Fig. 2). The predicted trends in pigs still alive varied with changes in the transmission coefficient (Fig. 3); an increase in the transmission coefficient decreased the percentage still alive, for any given time.

The sensitivity of the predictions was examined for varying levels of the mortality rate (Fig. 4). An increase in the rate resulted in a decrease in the estimated percentage of pigs alive.

There was virtually no effect on the percentage of pigs alive of varying the daily rate of addition of poisoned bait. The estimated percentage of pigs alive differed by less than 1% over the range examined. The threshold

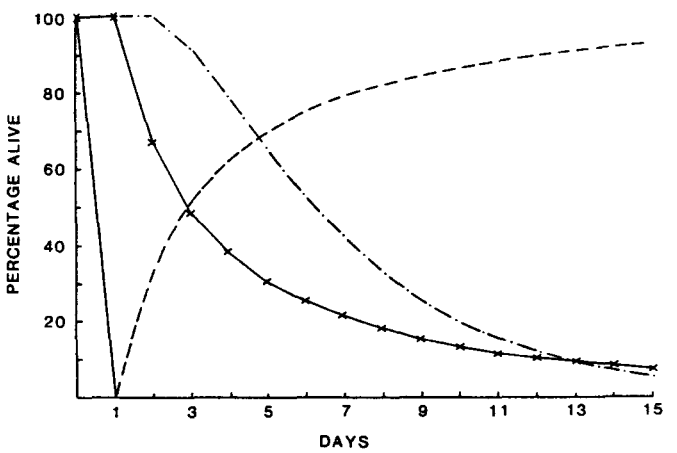

Fig. 2. Temporal trends in the predicted percentage of a population of feral pigs that is still alive (—·—·—) and in each class of susceptible (———), latent (×———×), and poisoned (- - - - -) pigs. Predictions are for Model A with the transmission coefficient of 0.01, inverse of the latent period of 0.333, poison-induced mortality of 0.274, the initial number of lethal doses of poison of 100 km^{-2}, with no daily additions of poison or recovery of pigs after poisoning.

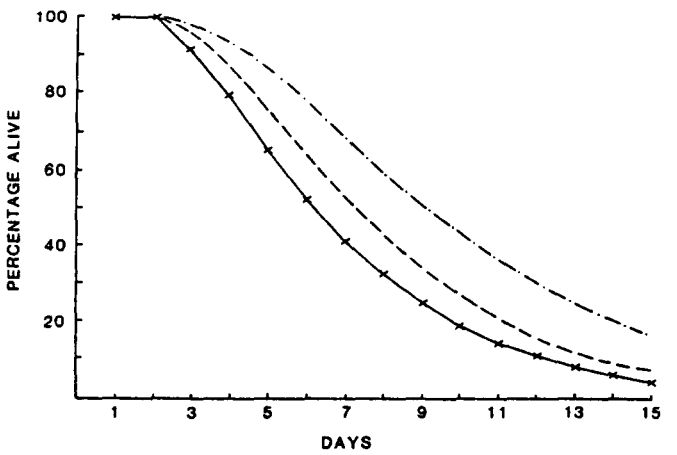

Fig. 3. The predicted percentage of feral pigs still alive after the start of warfarin poisoning, for differing values (0.01, × ——— ×; 0.005, — — —; 0.0025, —·—·—) of the transmission coefficient (β) in Model A.

daily level of poison addition (A_c), estimated by equation (6), was 0.49 lethal doses km^2 day^{-1}.

Model B

The predictions of Model B were generally similar to those of Model A. However, an increase in the value of the transmission saturation coefficient

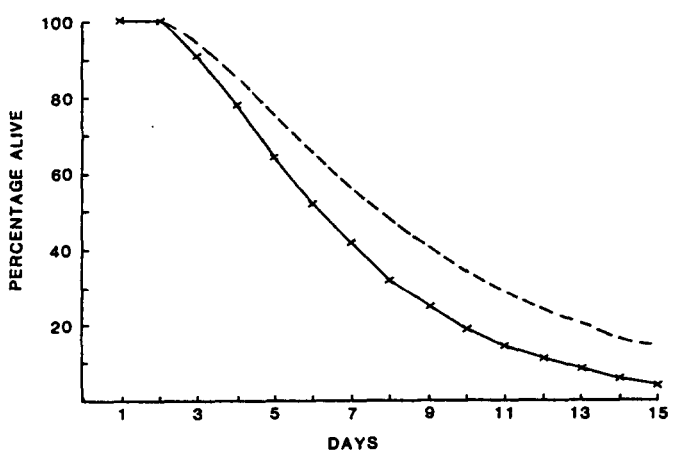

Fig. 4. The predicted percentage of feral pigs still alive after the start of warfarin poisoning, for differing values (0.274, × ——— ×; 0.175, — — —) of the poison-induced mortality rate (α) in Model A.

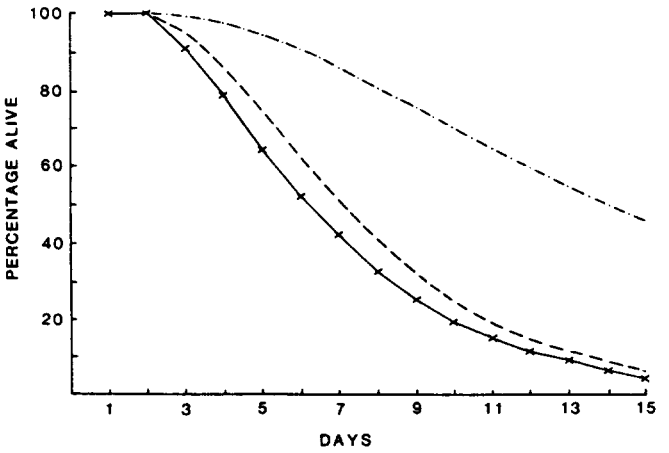

Fig. 5. The predicted percentage of feral pigs still alive after the start of warfarin poisoning, for differing values (0, ×——×; 0.01, — — —; 0.10, —·—·—) of the transmission saturation coefficient (c) in Model B. Parameter estimates were the same as in Model A.

(c) resulted in an increase in the percentage of the pests alive (Fig. 5). The increase in the saturation coefficient corresponds to the occurrence of a maximum rate of intake of poisoned bait by feral pigs.

Field results

The percentage kill of feral pigs was 94% (30 of 32) after 15 days. The trend in mortality is shown in Fig. 6. The decline in the percentage of pigs alive occurred later and was then steeper than most predictions.

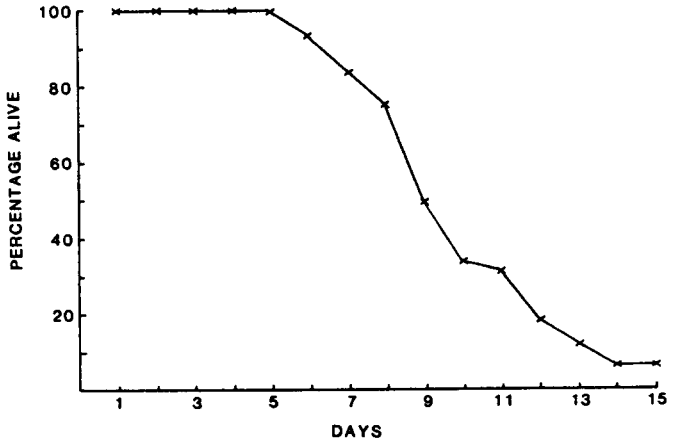

Fig. 6. Observed trends in the percentage of feral pigs still alive after the start of warfarin poisoning.

DISCUSSION

The models outlined describe the essential features of poisoning of feral pigs and probably many other vertebrate pests. The models have strategic predictive use to evaluate the effect of pesticides on vertebrate pests and complement the model of Hone (1986). The simulation model also indicates which data would be useful to collect in preliminary pen and field experiments in the process of pesticide evaluation. The analogy between the spread of infection through a susceptible animal population and the spread of poison through a vertebrate pest population appears to be useful.

The predictions of Model B are likely to be more accurate than those of Model A as they are based on the more realistic assumption of a limit to the intake of food by individual pests. The analysis here indicates that predictions are quite sensitive to changes in the value of the transmission saturation coefficient. There is a need for a method of estimating the value of the coefficient for pest animals in the field, so that realistic values can be incorporated in future modelling.

The accuracy of the models' predictions depends on the robustness of the models, their assumptions and the accuracy of the data used. The field results suggest that the marked pigs may have been slower to find and eat the poisoned bait than predicted. McIlroy et al. (1989) reported that bait consumption increased to day 5 then declined, which supports the suggestion of slow bait-take. Ideally the models described here should be used on a pest population that has reached peak intake of bait when poison is first introduced.

Use of the model to predict mortality or other changes in field populations of vertebrate pests will require either some knowledge of likely or actual bait-take such as either estimating the rate of bait-take (β) or running the model for different bait-take scenarios, such as low, medium and high, and obtaining estimates of mortality. Deaths may occur over a longer time in pen experiments than they do in the field. McIlroy et al. (1989) reported that the average time till death of the marked pigs was 9.7 days. The estimate assumes that each pig started eating poisoned bait on day 1 of poisoning.

The models complement the results of research on poisoning feral pigs by Saunders (1988) and Saunders et al. (1990) who reported a significant positive relationship between bait-take (y) and days (x), and Choquenot et al. (1990) who reported a significant relationship between days of poisoning and the reduction in bait-take by feral pigs. In both studies the trends in bait-take were not however compared with the trends in deaths of pigs in the area which were radio-collared.

The predicted trend over time in the percentage of animals still alive was

reversed sigmoidal (Figs. 2, 3 and 4). The predicted trend in deaths is then sigmoidal, which is similar to the expected trend in the number of animals infected with disease during the early stages of infection of a host population (Anderson, 1981). The models derived here describe the process behind the patterns described by Saunders (1988), Saunders et al. (1990) and Choquenot et al. (1990) and indicate the need to report trends in deaths after poisoning. The sigmoidal trends predicted by the models suggest that the saturation relationship with no point of inflection used by Saunders (1988) and the analysis used by Choquenot et al. (1990) may be approximations, especially for a chronic poison like warfarin. Saunders et al. (1990) reported a sigmoidal relationship between bait-take and time and assumed that the proportion of bait eaten was equivalent to the proportion of pigs killed.

The apparently reversed sigmoidal trend between time and the percentage of pests still alive may be more general than just for poisoning feral pigs. Results reported by Robinson and Wheeler (1983) for rabbit (*Oryctolagus cuniculus*) abundance after poisoning clearly show a reversed sigmoidal trend when either pindone (a chronic anticoagulant poison) or sodium monofluoroacetate (1080) were used. Data reported by Thomson (1986) were, however, not so clear. Trends in the percentage of dingoes (*Canis familiaris*) alive after poisoning with sodium monofluoroacetate showed variable patterns, sometimes a rapid drop then a flattening in the relationship, and at other times no clear trends. That variability may be associated with variable times of dingoes finding the poisoned bait, which was distributed from the air. In the other studies, poisoned bait was spread from the ground.

The analogy between poison spreading through a pest population and the spread of disease through a host population can be examined more thoroughly. The "pathogen" of poison results in a short, sharp peak in mortality compared with a generally longer pattern of mortality caused by conventional pathogens such as viruses (Halpin, 1975). Different poisons may have different pathways of action. For example, if a poison caused secondary poisoning of pests, then the model of pest dynamics would need to incorporate changes in the number of susceptibles because of contacts with poison in the environment (βXW) and poison in the infected hosts (βXY). Such a model would be very similar to that of the disease tuberculosis in badgers (Anderson and Trewella, 1985), wherein the badgers get infected from infected hosts or the environment. Acute poisons like sodium monofluoroacetate (1080), with shorter times to death than warfarin, would require use of shorter units of time than one day in a model. With such quick actions the latent period may be deleted from the model.

If the poison in the environment was only partly accessible to the pest population, then a model of the process would resemble the model of Hochberg (1989). That model predicted substantial changes in host dynamics if the environmental source of infection had variable accessibility. Such variable accessibility corresponds here to variable availability of poison during a prolonged poisoning program.

The basic model and the saturation model predict a threshold rate of daily pesticide addition to control the pest. This is analogous to the threshold rate of pathogen addition, described by Anderson (1982), needed for biological control of a pest. The results suggest that the modelling of pathogen dynamics (Anderson, 1982) and pesticide effects, described here, could be combined in a different way to that described by Carpenter (1981). In a combined model there would be separate transmission coefficients for each source of mortality – pathogen and pesticide. The pattern of bait distribution and pest behaviour is an area for future field and modelling research. Different patterns, such as random, clumped or regular, could generate different rates of pesticide uptake and hence overall effectiveness.

The disease model of Anderson (1981) and Anderson and May (1981) assumed that pathogens in the environment increased at a per capita rate λ, as a result of pathogen production by infective hosts. An alternative assumption for modelling poisoning for future evaluation is that poison bait is added at a rate proportional to the abundance of pests (N), so that as pests die and N decreases then the rate of addition decreases. The models predict that if poison is left available within the environment of the pest then eventually all the pests will die, unless some become isolated and such pests do not become susceptible. The models could be extended to include an isolated segment of any population that by definition cannot be poisoned. Hone (1983) developed a model to estimate the proportion of a field population that was isolated.

Pesticide resistance could produce similar negative results to isolation on the effectiveness of poisoning. Resistance will change the mortality rate in the models. Isolation, in contrast, changes the transmission coefficient. The mechanisms of pesticide resistance (Greaves, 1985) could be modelled using a similar approach to the study of genetics and epidemiology described by May and Anderson (1983), that is to link the dynamics of pesticide effects to the frequency of resistance genotypes in the population.

The models reported here could also be used to describe and study the spread through an animal population of bait-derived disease immunity. Foxes (*Vulpes vulpes*) in parts of Europe are vaccinated against rabies by distribution of baits containing an oral vaccine (Pastoret et al., 1988; Brochier et al., 1988). The model would require inclusion of an immune

segment of the population and would then predict temporal changes in the immune status of foxes.

Two general cautions need to be applied to the results of the modelling. Agreement between predictions and results does not prove the model is correct, as the results may be explained by an alternative model some time in the future. Secondly, the predictions describe average results and the actual data describe one unreplicated result. Hence some difference between the predictions and the results is to be expected.

The models could be developed for training of field and advisory staff, by application on a micro-computer. Staff would then learn the decisions involved in poisoning vertebrate pests and the consequences of making different decisions. The various field situations outlined earlier could be included as an introductory menu from which an operator chooses the relevant field situation for the particular vertebrate pest-poison situation being evaluated and appropriate parameter estimates.

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