# Predicting the impact and control of stoats: a review of modelling approaches

SCIENCE FOR CONSERVATION 191

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Published by
Department of Conservation
P.O. Box 10-420
Wellington, New Zealand

*Science for Conservation* is a scientific monograph series presenting research funded by New Zealand Department of Conservation (DOC). Manuscripts are internally and externally peer-reviewed; resulting publications are considered part of the formal international scientific literature.
Titles are listed in the DOC Science Publishing catalogue on the departmental website http://www.doc.govt.nz and printed copies can be purchased from science.publications@doc.govt.nz



ISSN 1173-2946
ISBN 0-478-22235-1

This report was prepared for publication by DOC Science Publishing, Science & Research Unit; editing by Michael Ashdown and layout by Ruth Munro. Publication was approved by the Manager, Science & Research Unit, Science Technology and Information Services, Department of Conservation, Wellington.

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# Predicting the impact and control of stoats: a review of modelling approaches

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## ABSTRACT

General approaches to modelling pest impacts and pest control are reviewed, to identify useful approaches to modelling stoats (*Mustela erminea*) and their effects on key prey species in New Zealand. The review is in two parts: models for stoat impacts; and models for stoat population dynamics and control. The first covers different kinds of predator–prey models and a preliminary model for effects of stoat control on mohua (*Mohoua ochrocephala*) viability. The second covers models for different control options, including culling, non-disseminating and disseminating fertility control, and pathogens. It includes a preliminary, single-species density-dependent model for stoat dynamics based on trapping data for beech (*Nothofagus*) and non-beech forest habitats. Recommendations are given for modelling and data needs, and it is envisaged that future work will involve the melding of the two components considered in the review into interactive models for both control and impacts.

Keywords: stoat, *Mustela erminea*, population modelling, biological control, pest management, predator–prey models


Barlow, N. and Choquenot, D. 2002: Predicting the impact and control of stoats: a review of modelling approaches. *Science for Conservation 191*. 46 p.

# 1. Introduction

This review of models useful for predicting stoat impacts and the effects of stoat control was carried out for the New Zealand Department of Conservation (DOC) by AgResearch and Landcare Research in April–May 2000. The aim of this project was to review models relevant to predicting (1) how integrated control based on existing methods and emerging biocontrol technologies will affect stoat (*Mustela erminea*) density, and (2) the influence stoat control will have on the long-term viability of prey species at risk. We also considered the availability of data required to estimate parameters for relevant models. Quantitative modelling is a powerful way of evaluating the likely effects of alternative stoat control strategies on stoat density and on the abundance of prey species affected by stoats. Models can be used to explore: (1) how the frequency and placement of current stoat control technology influences the viability of prey populations; (2) the likely effectiveness of novel technologies, and whether they could meet the requirements for sustained viability of threatened species; (3) how existing and emerging stoat-control techniques (including biocontrol) can be used and combined to maximise their cost-effectiveness; and (4) potential adverse effects stoat control may have on the abundance of other pest species.

If DOC staff use modelling to integrate the results of new stoat research in order to develop these predictive capabilities, they will be in a position to formulate highly prescriptive management recommendations and provide explicit policy advice. Implementation of these recommendations and policies will ensure that the results of new research are used to increase the efficiency of ongoing stoat management.

The review that follows is divided into two related sections that deal with: (1) models linking stoat management, stoat density, and the viability of prey populations; and (2) the effect of integrated conventional and biological control on stoat density. Each section provides a general review of relevant modelling approaches before focusing on the types of modelling that will best allow development of the predictive framework described above. While, for the purposes of this review, we have treated impact modelling and integrated control modelling as largely separate undertakings, in future they will need to be more formally linked to provide a comprehensive modelling framework that can be used formally to assess the influence that alternative strategies for integrating stoat control have on prey species at risk.

## 1.1 OVERVIEW OF POPULATION MODELS

If models are used to support estimates of control impacts, on both stoats and their prey, these models must incorporate the principal factors affecting population densities, particularly those giving a compensatory response. Such compensatory responses are broadly described as density-dependence. Population models that include density-dependence (as they all must) fall typically into two categories:

1. Single species—in which the density-dependence is intraspecific and *direct*, or can realistically be treated as such (e.g. logistic model, Ricker model)
2. Interactive—in which the density-dependence is indirect or *interactive*, resulting from a feedback via another trophic level (e.g. predator–prey, herbivore–plant models).

Both are density-dependent in the sense that an upward perturbation in density of the species of interest results in a compensatory increase in proportional mortality (or decrease in per capita recruitment), either directly or through the resulting change in another trophic level (e.g. a decline in resource or increase in natural enemy density). The reverse is also true, a downward perturbation giving an increase in survival.

Sometimes the interactive models can be realistically simplified to single-species ones with the interactive density-dependence treated implicitly as direct density-dependence. Alternatively, the fully interactive models may be simplified to partially interactive ones, in which the predator affects the prey's rate of increase but the predator density (rather than its rate of increase) is linked directly and empirically to prey density (but not its own density). We have called these models 'isocline' or 'prey-dependent' models. Stoat dynamics are known to depend on those of mice in beech forests and there is evidence that they may depend on rat populations in non-beech habitats (see below). There is also evidence for direct density-dependent survival within the stoat population. Consequently, we consider all these kinds of models, and it is likely that a final stoat model will need to combine features of both single-species and interactive models.

# 2. Thresholds for pest control in predator–prey systems

There are two broad approaches that can be used to conceptualise how reductions in predator abundance will affect the abundance of primary and secondary prey: (1) graphical analysis of isocline or prey-dependent predator–prey models, and (2) simulation of dynamic, fully interactive predator–prey models (Choquenot & Parkes 2000).

## 2.1 ISOCLINE OR PREY-DEPENDENT PREDATOR–PREY MODELS

### 2.1.1 Predator–prey interactions

Considerable theoretical and empirical research has been conducted on dynamic interactions between predators and their prey in which the biological characteristics of both are modelled (Rosenzweig & MacArthur 1963; May 1973). These models use a functional response to describe variation in the

predators' per capita offtake of prey ($o$) as a function of prey density ($p$). A general form for the functional response is:

$$o = c(1 - e^{-d(p-b)}) \quad (1)$$

where $c$ is the maximum per capita rate of prey offtake, $b$ is the prey density below which prey offtake declines to 0, and $d$ is the relative foraging efficiency of the predator, describing how quickly the rate of offtake decreases with declining prey density. Two types of functional response are generally used in predator–prey models, a Type 2 response in which prey are consumed at a declining rate until their abundance falls to 0 ($b = 0$), and a Type 3 response, in which the rate at which prey are consumed falls to 0 while some prey remain ($b > 0$) (Fig. 1). This 'threshold' form of Type 3 response is different from the sigmoidal form used in many predator–prey models. Sigmoidal models imply that prey offtake declines toward 0 at low prey density, but does not actually reach 0 until all prey have been consumed (Fig. 2). Sigmoidal models apply to predator–prey systems in which the decline in prey offtake is due to a relative rather than absolute refuge from predation at low prey densities. Prey can find refuge from predators at low prey densities because either (1) there is some limited physical protection from predators (e.g. a limited number of cavities in a tree or some other predator-proof habitat that is in short supply), or (2) in switching to some other source of prey, predators lose their search image for the original prey. The functional difference between threshold and sigmoidal forms of the Type 3 response manifests itself only at very low prey densities.

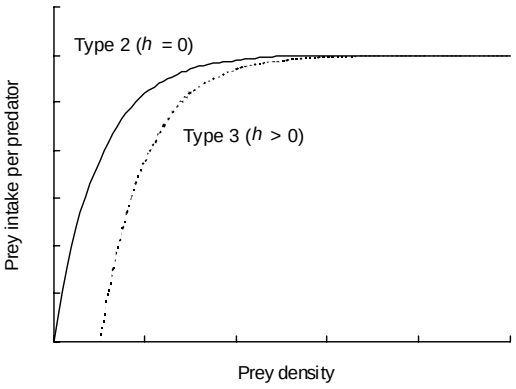


Figure 1. The form of Type 2 and Type 3 functional responses for predators consuming prey. The Type 3 response implies that offtake of prey by predators falls to 0 while some prey remain.

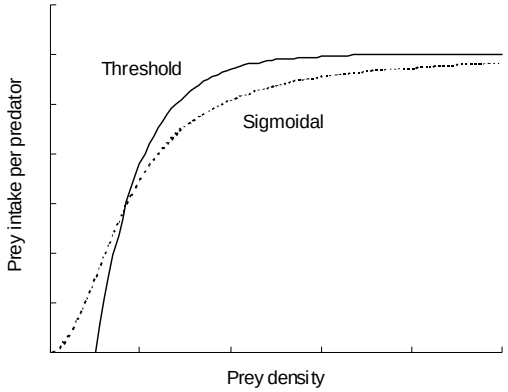


Figure 2. Threshold and sigmoidal forms of the Type 3 functional response. While both forms imply the existence of a refuge from predation at low prey density, the threshold form assumes an absolute refuge, whereas the sigmoidal form assumes a relative refuge.

In addition to its influence on the rate of prey offtake through the functional response, prey abundance potentially influences the abundance of a predator population through some form of numerical response. Two numerical responses have been used in predator–prey modelling: 'isocline' numerical responses, which relate the equilibrium or steady-state density of the predator population to the prevailing abundance of its prey; and 'dynamic' numerical responses, which relate the instantaneous rate of change in predator abundance to the prevailing abundance of both its prey and its own population. Isocline numerical responses represent the reciprocal interdependencies that link predator and prey abundance, and the social behaviours that ultimately limit predator density implicitly. In contrast, dynamic numerical responses represent

these interdependencies and social behaviours (Caughley & Sinclair 1994). In this section we focus on models that employ isocline numerical responses.

An isocline numerical response occurs where territorial behaviour or interference competition regulates the density of a predator population at high density, and the accessibility or availability of the affected prey population at lower densities (Caughley & Sinclair 1994; Sinclair & Pech 1996). The product of an isocline numerical response and a functional response is the 'total response' curve, which predicts prey offtake by predators as a function of prey availability. Prey offtake can be divided by prevailing prey density to estimate the proportional offtake of prey by the predator population. Proportional offtake can be contrasted with the proportional productivity of the prey population (i.e. proportional recruitment in absence of predators) to explore the dynamic behaviour of a predator–prey system (Fig. 3). Points where the recruitment curve intersects the offtake curve identify stable equilibria between the prey and predator populations (points A and C), and unstable boundaries (point B). Stable equilibria are points where a decline in prey abundance leads to higher proportional recruitment than proportional offtake, while an increase in prey abundance leads to higher proportional offtake than recruitment. Unstable boundaries are points where a decline in prey abundance leads to lower proportional recruitment than offtake and further declines in abundance, while an increase in prey abundance leads to higher proportional recruitment than offtake and further increases in abundance. The predator–prey system shown in Fig. 3 tends toward the stable equilibrium at C, unless prey abundance declines below levels corresponding to the unstable boundary at B, below which the prey population will continue to decline to the stable equilibrium at A. Because the differential between recruitment and offtake effectively traps the prey population at this lower equilibrium point, it is commonly known as a 'predator-pit'.

The tendency that the predator–prey system shown in Fig. 3 has toward equilibria greater than 0 reflects the stabilising properties a Type 3 functional response and isocline numerical response impart to the predator–prey systems. Systems that lack these features will generally display an inversely density-dependent or entirely density-independent total response at low prey densities. In the absence of any decline in proportional offtake at low prey density, such systems are prone to prey extinction (May 1973; Sinclair & Pech 1996). For example, if the availability of a threatened prey species does not limit predator density (e.g. the prey species is a secondary prey item), the predator population will generally lack a numerical response to the density of that species. In the absence of a numerical response, predator density will not decline as the abundance of the secondary prey species is progressively depleted, and unless

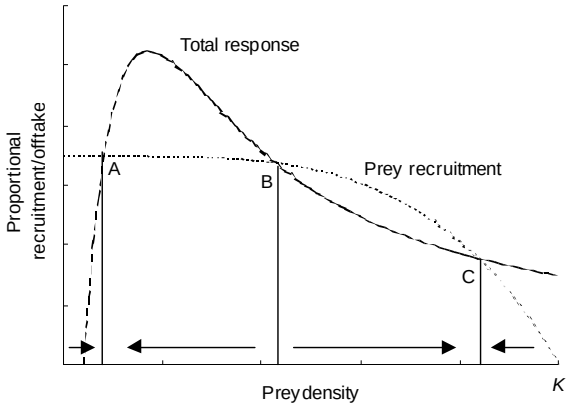


Figure 3. A density-dependent predator–prey system, consisting of a proportional total-response curve (describing proportional offtake of prey by predators assuming a Type 3 functional response and an isocline numerical response to prey density), and a proportional prey-recruitment curve. Arrows show the direction of change in prey density displaced from stable equilibria at A and C, either side of an unstable boundary at B. Prey carrying capacity in the absence of predators is shown as *K*.

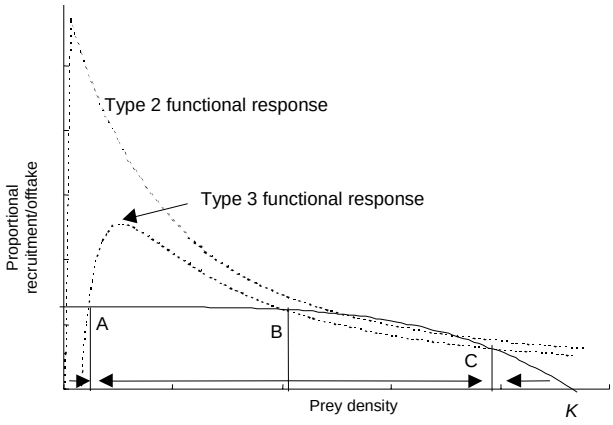


Figure 4. Proportional prey recruitment and offtake curves, corresponding to Type 2 and Type 3 functional responses, where predators have no numerical response to prey density (i.e. prey are a secondary food item that does not regulate predator density). For the Type 3 response, points A and C are stable equilibria, and B is an unstable boundary. The carrying capacity of the prey population in the absence of predators is shown as *K*.

some other mechanism reduces offtake of the species, it will undergo extinction. Figure 4 shows two predator–prey systems in which predators have Type 2 or 3 functional responses to prey density, but lack a numerical response. Proportional prey offtake by the predator population with a Type 3 functional response declines in a density-dependent fashion, leading to a stable lower equilibrium in prey abundance. In contrast, for the predator with a Type 2 functional response, proportional prey offtake is inversely density-dependent below the unstable boundary at B, and prey abundance has no stable lower equilibrium. Under these circumstances, a reduction in prey density below levels corresponding to the unstable boundary B would cause the prey population to collapse to extinction.

### 2.1.2 Implications of predator–prey models for setting threshold predator densities

In the context of isocline or prey-dependent predator–prey models, predator control reduces prevailing predator density, and either continuously or periodically disrupts any numerical response predators have to prey abundance. This can artificially elevate prey density above the equilibria where they would normally be regulated by predation, or can lead to dynamic state changes in prey abundance where prey density moves from lower to higher stable equilibria. For example, if a prey population to which a predator has a Type 3 functional response but no numerical response (e.g. the prey species is a secondary dietary item that does not regulate predator abundance) is occupying its higher equilibrium at C, predator control will reduce offtake relative to recruitment and the prey population will increase toward *K* (Fig. 4). If predator control ceases, predator density and prey offtake will increase, and prey density will decline back to the equilibrium at C. If the prey population occupies the lower equilibrium at A, while predator control again reduces offtake leading to increased prey abundance, the effect of stopping predator control is more complex. If control has allowed prey density to increase beyond levels corresponding to the unstable boundary B, the prey population will continue to increase until it reaches stable equilibrium C, regardless of whether or not predator control ceases (i.e. predator control has allowed the prey population to escape the 'predator pit'). In contrast, if predator control

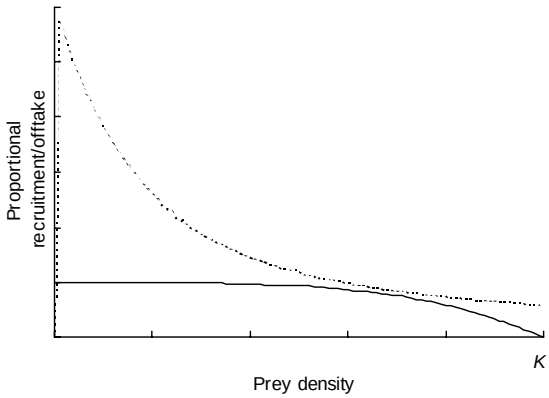


Figure 5. Proportional prey recruitment and offtake curves corresponding to a Type 2 functional response for the predator and no numerical response to prey density (i.e. prey are a secondary food item that does not regulate predator density). There are no stable equilibrium points, implying that the prey population will be driven to extinction (zero carrying capacity, *K* ) through predation.

stops before the prey population recovers to densities exceeding the unstable boundary B, offtake will once again outstrip recruitment and prey density will fall back to equilibrium A (i.e. predator control has not been sustained long enough for the prey population to escape the predator-pit). Hence, predator control can potentially move prey density between equilibria at A and C, or allow density to increase toward *K*. While sustained predator control is necessary to increase prey density beyond levels corresponding to C, ongoing control is unnecessary to increase prey density from A to C, as long as control is sustained long enough to allow prey density to increase beyond B. Hence, in addition to the effect of control on predator density, the response of prey populations will depend on the duration of a control programme and the abundance of prey when control is undertaken.

If prey offtake by predators becomes inversely density-dependent at low prey densities (e.g. predators have no numerical response to prey density and a Type 2 functional response), offtake will be higher than recruitment at any level of prey density below that corresponding to the unstable boundary B (Fig. 4). Under these conditions, if prey density falls below levels corresponding to B, predator control will be necessary to avert prey extinction. However, if predator control elevates recruitment above offtake for a period sufficient to increase prey density above B, ongoing control will not be necessary for prey persistence. Of course, many other combinations of prey recruitment and total predator response to variation in prey density are conceivable. However, most of these cases will be variations on the predator–prey systems summarised here, and their implications for predator control will generally be intuitive. For example, where the total response always exceeds the prey population's capacity for recruitment, continuous predator control is necessary to avoid prey extinction (Fig. 5).

These examples demonstrate that by taking into account the reciprocal influence predators and prey exert over each other's abundance, strategic components of predator control, beyond the implications of simple measures of prey offtake, can be considered. These strategic components focus on three aspects of predator control in relation to consequent changes in prey abundance:

1. The reduction in predator density necessary to halt or reverse declines in prey abundance, or to initiate recovery in a prey population that has undergone a decline in abundance.
2. The timing of predator control to avoid unnecessary expense (i.e. avoiding ongoing control following attainment of prey densities where recruitment is higher than offtake).
3. The timing of predator control to avoid wasted expense (i.e. cessation of control before prey densities rise to where recruitment is higher than offtake).

Taken together, these components suggest that not all predator control is equal in terms of its contribution to changes in prey abundance. Depending on the configuration of the predator–prey system and the current level of prey abundance, small changes in predator density maintained over long periods can have a more marked effect on prey abundance and viability than periodic large-scale reductions. Moreover, the notion that predator eradication is always necessary for recovery of prey populations is not always true. Indeed, even where

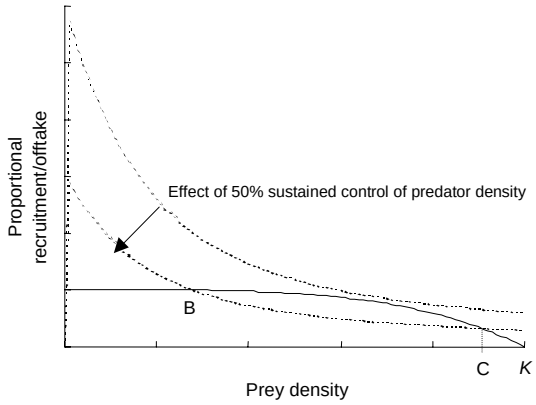


Figure 6. The effect of imposing a 50% reduction in predator density on proportional prey recruitment and offtake curves corresponding to a Type 2 functional response for the predator and no numerical response to prey density (i.e. prey are a secondary food item that does not regulate predator density). The reduction in predator density establishes a potentially stable equilibrium between predators and prey at about 90% of carrying capacity (*K*) for prey.

prey offtake by uncontrolled predators exceeds recruitment at all levels of prey abundance, constraining predators at moderate densities may allow recruitment sufficient to avert prey extinction and maintain relatively high levels of prey abundance. For example, imposing a 50% sustained reduction in predator density on the predator–prey system shown in Figure 5 produces a stable equilibrium with prey abundance at around 90% of *K* (equilibrium point C in Fig. 6). This outcome assumes that predator control elevates prey abundance above levels corresponding to the unstable boundary B. While eradication of predators from this system would allow recovery of prey density to *K*, the value of the additional 10% increase in prey density would have to be weighed against the opportunity costs associated with the permanent removal of the residual 50% of predators.

### 2.1.3 Limitations of isocline predator–prey models

While isocline predator–prey models provide useful insights into the implications predator control has for predator–prey interaction, they do not generally address the causes of initial prey decline. For example, the two predator–prey systems shown in Fig. 4 have very different dynamics below prey densities corresponding to the unstable boundary B, with different implications for levels of predator control necessary to increase prey abundance permanently. But what would cause prey density to decline below levels corresponding to B in the first place? Any fluctuation that elevates predator offtake or inhibits prey recruitment will reduce the abundance of a prey population, independently of the trophic processes summarised in predator–prey models. For example, assuming a predator has an isocline numerical response to the abundance of its primary prey species, a transient increase in the abundance of some secondary food source may temporarily elevate predator density above that associated with the abundance of its primary prey (Fig. 7). The resultant increase in offtake of the primary prey species may be high enough to counteract recruitment, leading to a decline in subsequent prey density. If the temporary increase in predator density persists long enough, the density of the primary prey species may decline below the unstable boundary B, at which point their abundance will collapse to the lower equilibrium at A. The primary prey population will thence

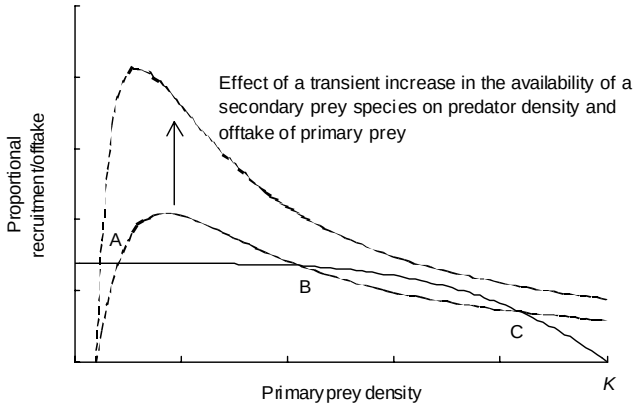


Figure 7. The effect of a transient increase in secondary prey availability on a predator population and its implications for the density of a primary prey species. The temporary release of predator from the numerical constraint of primary prey density temporarily elevates proportional offtake of the primary prey such that the higher equilibrium prey density disappears. If the transient increase persists long enough that primary prey density falls below the unstable boundary B, the primary prey population will collapse to the lower equilibrium at A.

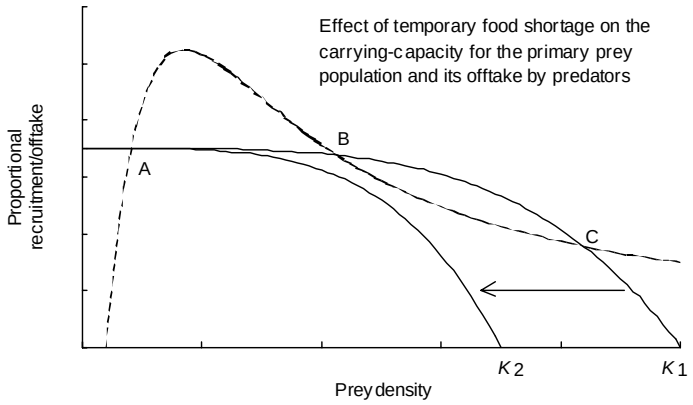


Figure 8. The effect of a temporary food shortage for a prey population and its implications for prey offtake. The upper equilibrium C disappears as the carrying capacity (*K*) for the prey population declines. This elevates offtake above recruitment and the prey population collapses to the lower equilibrium at A.

be trapped in a predator pit, even though the transient increase in the abundance of the secondary prey passes and predator density declines.

Alternatively, if the availability of food to a prey population undergoes a temporary decline because of unseasonable conditions, its density at carrying capacity (*K*) may shift downwards, modifying the form of the proportional recruitment curve (Fig. 8). If prey density at *K* declines far enough, the upper equilibrium at C will disappear and the prey population will collapse to the lower equilibrium at A, where it will be trapped in a predator pit regardless of whether or not food resources return to their former levels.

It is important to appreciate that because low-density populations are far more likely than higher-density populations to be driven to extinction by chance events (Caughley 1994), the consequences of transitory effects will usually be more critical for prey populations trapped in predator pits than those at the higher equilibrium C. Hence, while analysis of the stability points for predator–prey systems may imply that a prey population can remain indefinitely at densities corresponding to equilibrium A, the probability of such a population being driven extinct through stochastic (chance) effects are substantially greater than they are for a prey population at the higher equilibrium C. The inability of isocline predator–prey models adequately to represent the effects of environmental stochasticity on the dynamics of predator and prey populations may be particularly telling where predation holds the prey population at a low densities.

While isocline predator–prey models can be used to consider the influence of density-independent perturbations in predator and prey populations on the subsequent dynamics of the system, they are of limited use in understanding predator–prey systems in which such perturbations occur regularly. This limitation stems from the implicit nature of the isocline numerical response used to predict changes in predator abundance from prey density. In many biological systems prey abundance does not remain at any given density long enough for a predator population to adjust its demographic rates such that it settles at a new equilibrium density. In these systems, rates of change in predator abundance will shift more or less constantly as prey abundance varies with the vagaries of weather, the effects of offtake by other predators, and a range of other environmental factors (Caughley 1987; Caughley & Gunn 1993). Because continual perturbation in predator–prey systems has consequences for how predator control influences prey abundance, models that allow environmental variation to be considered directly may be more useful for understanding predator control strategies than isocline predator–prey models, in cases where such variation is significant.

## 2.2 INTERACTIVE PREDATOR–PREY MODELS

Caughley (1976, 1977) formulated a class of models describing interactions between resources and consumers in which the isocline numerical response was replaced with a more explicit feedback loop between resource and consumer abundance. This feedback loop comprised the same functional response used in the isocline or prey-dependent predator–prey models described in the previous section, but replaced the isocline numerical response with a dynamic numerical one that relates rates of change in consumer abundance to resource availability. The resultant feedback loop operates by adjusting the abundance of consumers and the amount of the resource each consumer eats in response to changes in the resource. Hence, a decline in resources leads to a decrease in consumer density and the per capita rate at which consumers are eating the resource, which allows resource abundance to increase leading to an increase in the density of consumers and their rate of resource consumption, and so forth. In contrast to isocline predator–prey models, this feedback loop allows the effect of density independent perturbations in resource or consumer abundance on the consequent changes in the dynamics of the consumer–resource system to be considered explicitly.

While most attention on this class of models for vertebrates has focused on their application for understanding vegetation–herbivore interaction (Caughley 1976, 1987; Caughley & Lawton 1981; Caughley & Gunn 1993, 1996; Choquenot 1992, 1998), the potential application of interactive modelling for understanding predator–prey dynamics in systems subject to regular density-independent perturbation has been recognised (Caughley 1977; Choquenot & Parkes 2000). Caughley (1977) formulated an interactive vegetation–herbivore–predator model, in which functional and dynamic numerical responses were used to link vegetation and herbivores, and herbivores and predators (Fig. 9, Appendix 1). The use of a dynamic numerical response to drive changes in predator density as a function of prey availability ignores the potential limit placed on predator density through territorial spacing behaviour or other forms of social interaction. An alternative numerical response that allowed these effects to be considered was described by Tanner (1975). While the parameter set given in Caughley (1977) did not produce the stable outcomes he reported, a modified parameter set does (W. Ruscoe & D. Choquenot, unpublished).

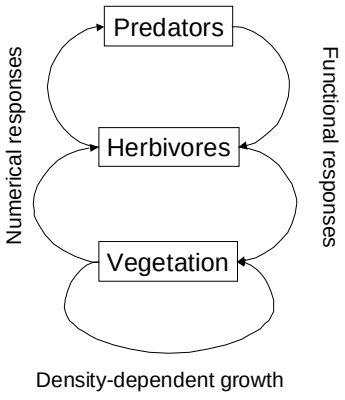


Figure 9. The structure of a three-level vegetation–herbivore–predator model formulated by Caughley (1977). Variation in vegetation biomass is dictated by density-dependent growth and offtake by herbivores via their functional response. The density of herbivores is dictated by vegetation availability via a numerical response, and offtake by predators via their functional response. The density of predators is dictated by the availability of herbivores via a numerical response. Caughley used dynamic numerical responses in his original formulation of the model.

The lack of studies based on the Caughley (1977) interactive modelling approach probably reflects (1) the daunting data requirements necessary to obtain parameter estimates to produce a valid model, and (2) the focus of most vertebrate studies on isocline or prey-dependent predator–prey models such as those described earlier in this section. However, for many predator–prey systems, some appreciation is desirable of how density-independent perturbation influences the interaction of predators and their primary and secondary prey beyond that achievable in isocline models. This will be particularly so where predator–prey models are used to assess the implications

of predator control for the abundance of prey populations (Choquenot & Parkes 2000). For example, Choquenot (unpubl. data) used the conceptual framework developed by Caughley (1977) to assess the implications of density-independent fluctuations in the food resources of wild house mice for the effectiveness of stoat control to enhance the viability of mohua (*Mohoua ochrocephala*), in southern beech (*Nothofagus*) forest. Stoats are limited by the availability of mice (*Mus musculus*) in these forests, but cause significant predation impacts on mohua nests and nesting females when high mouse availability elevates their density (see Section 3). While Choquenot (unpubl. data) did not elaborate the full interactive model laid out by Caughley (1977) (i.e. reciprocal effects of increasing mouse density on the availability of their food resources, and of stoat density on mouse density were not considered), there were compelling biological reasons to suspect that the links that were left out are of little significance to the primary dynamics of the system. More importantly, the use of an interactive predator–prey framework provided insights into the relative efficiency of stoat control for mohua conservation that static isocline models could not (Choquenot & Parkes 2000; Choquenot unpubl. data).

## 2.3 MODELLING PREY RESPONSE TO STOAT IMPACT

Field observations and some limited data suggest that while stoat density is more stable in some forest types than others (e.g. podocarp–broadleaf forest compared with southern beech forest), stoats undergo semi-periodic fluctuations in all forest types. The likely cause of these fluctuations is periodic increases in the density of their primary prey, driven by density-independent semi-periodic fruiting or seeding by common forest plants (O'Donnell 1996; Wilson et al. 1998). For example, Fig. 10 shows relationships between indices of density for rodents (rats (*Rattus rattus*) in podocarp–broadleaf forest, mice in beech forest) and stoats in two forest types, which imply strong numerical relationships between rodents and stoats. While some dietary studies support the implication that stoat density is primarily limited by rodent density (e.g. Murphy & Bradfield 1992; King 1983; Murphy et al. 1998), other studies have suggested that the general increase in bird and invertebrate abundance, which parallels that of rodents, is also important (Murphy & Dowding 1995).

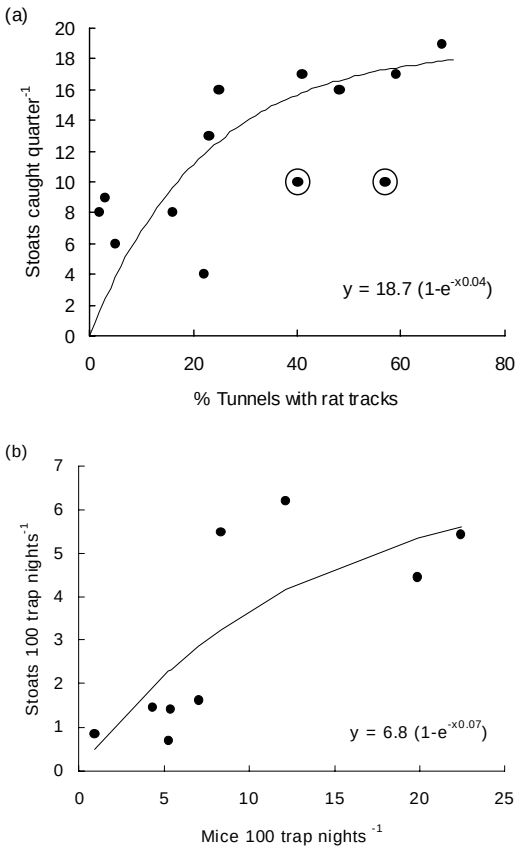


Figure 10. Variation in indices of stoat density with indices of (a) ship rat (*Rattus rattus*) density in podocarp–broadleaf forest at Mapara (Murphy et al. 1998), and (b) mouse (*Mus musculus*) density in beech forest habitat in Fiordland (King 1983), and fitted exponential models. The circled data points in (a) represent densities after aerial poisoning with 1080, and have been excluded when fitting the model.

Regardless of the actual mechanism driving fluctuations in stoat density, the strong apparent influence of density-independent perturbations in stoat density suggests that interactive approaches to modelling the effect of stoat management on the abundance of their prey will be of more use than approaches based on static, prey-dependent models.

The major limitation of interactive predator–prey models is the lack of data from which to estimate them. Collecting sufficient data to estimate the functional links between the food of primary prey and primary prey density, the density of primary prey and that of predators, and the density of predators and offtake of secondary prey, is a daunting task. Choquenot (unpubl. data) was able to construct a dynamic model linking seedfall–rodents–stoats–mohua in beech forest only because (1) work on the relevant components of the system had been undertaken (often fortuitously) over the preceding two decades, and (2) the system responded rapidly and systematically to the regular density-independent perturbation of the system caused by semi-periodic beech masting. This suggests that relationships such as that shown in Fig. 10a may provide a useful starting point for developing dynamic models of interactions between stoats and their prey in other habitats, particularly in podocarp–broadleaf habitats. However, while the apparent numerical response of stoats to rats shown in Fig. 10a is encouraging, the data upon which it is based need to be treated cautiously. A significant component of variation in rat density captured in these data represents the effects of poisoning with brodifacoum, which is known to cause secondary poisoning in stoats (Murphy et al. 1999). Hence, it is possible that the association between stoat and rat density suggested by these data represents coincident effects of poisoning on both species, rather than any functional relationship between stoat and rat density. Nevertheless, if some form of numerical relationship between rat and stoat density exists, it would hold the key to developing models that could be used to predict the costs and effectiveness of stoat control in podocarp–broadleaf forest. We return to this point when discussing research priorities for development of better predictive stoat impact models in Section 3.4.

# 3. Existing models of prey responses to stoat control

## 3.1 KIWI

A preliminary model of stoat predation on kiwi (*Apteryx* spp.) is briefly detailed in McLennan et al. (1996), and described more fully in Basse et al. (1999). While the model described by Basse et al. draws predominantly on demographic data for northern brown kiwi (*A. mantelli*), McLennan et al. contrast much of these data with those available for other species. The model uses estimates of fecundity and non-predation mortality to predict the dynamics of kiwi populations in the absence of stoats (the principle predator of kiwi), and estimates of chick (< 1 month old) and juvenile (1 to 18 months) predation to

explore the effect of stoat density on trajectories of kiwi density. Although stoats occasionally kill adult kiwi, these studies considered this source of mortality to be relatively unimportant, and it was not considered in the models. Similarly, all predation was assumed to be due to stoats, although in practice 10 out of the 13 known predation deaths of immature kiwi in McLennan et al. were from mustelids. We have revisited these data and estimated stoat impacts using a slightly different approach to that of Basse et al., namely age-specific life-tables. For an exponentially growing population in the absence of density-dependence (as assumed in Basse et al.), this gives an accurate assessment of rates of increase in the face of various age-specific fecundities and survival rates. We used this analysis to revisit the estimates of stoat impacts, and the reductions required in stoat densities to allow kiwi to survive.

For the most part the figures in Table 1 are based on those in Table 4 of McLennan et al. (1996), but the following two points of clarification are necessary in interpreting those figures. Firstly, the 7 'unknown' juveniles did not have transmitters attached so are ignored in our analysis; Table 1 therefore begins with 20 juveniles monitored rather than the 27 in McLennnan et al.'s Table 4. Secondly, as mentioned in these authors' caption to Table 4, the 'alive' category of juveniles had actually shed their transmitters and, as stated in the text, it is thought that 2 of these birds survived while 3 disappeared (J. McLennan pers. comm.). Thus, only 2 out of 20 juveniles survived, not 5, and the remaining 3 of the 'alive' group in Table 4 we added to the 'disappeared' category in our Table 1.

Table 1 suggests that 8.2–26.5% of chicks and 45–85% of juveniles die from predation, with predation comprising 18–59% of total chick mortality and 50–94% of total juvenile mortality. Combining chicks and juveniles and considering overall fates of young kiwi, total mortality is 94.5% (based on a survival of 2/20 for juveniles and 27/49 for chicks). If all 5 juveniles in the 'alive' category in McLennan et al.'s Table 4 which shed their transmitters actually survived, the total mortality becomes 86%. Assuming 94.5% total mortality based on the most likely fate of these 5 birds (McLennan et al. 1996), that attributable to predation

TABLE 1. SURVIVAL DATA FOR KIWI CHICKS AND JUVENILES SUMMARISED FROM MCLENNAN ET AL. (1996), WITH LOSSES DUE TO PREDATION ESTIMATED IN 3 WAYS, ASSUMING: 1) NO 'DISAPPEARED' LOSSES WERE DUE TO PREDATION; 2) 'DISAPPEARED' ATTRIBUTED TO PREDATION IN PROPORTION TO KNOWN DEATHS; 3) ALL 'DISAPPEARED' ATTRIBUTABLE TO PREDATION.

| | INDIVIDUAL FATE (% OF SAMPLE) | | PREDATION AS % OF TOTAL MORTALITY | |
|---|---|---|---|---|
| | CHICKS | JUVENILES | CHICKS | JUVENILES |
| Monitored | 49 (100) | 20 (100) | | |
| Lived | 27 (55) | 2 (10) | | |
| Died (non-predation) | 9 (18.4) | 1 (5) | | |
| Died (predation) | 4 (8.2) | 9 (45) | | |
| Died (disappeared) | 9 (18.4) | 8 (40) | | |
| Total died | 22 (45) | 18 (90) | | |
| Total killed by predators (1) | 4 (8.2) | 9 (45) | 18 | 50 |
| Total killed by predators (2) | 6.8 (13.8) | 16.2 (81) | 31 | 90 |
| Total killed by predators (3) | 13 (26.5) | 17 (85) | 59 | 94 |

ranges from 50–89% of young birds, given the different predation assumptions in Table 1. As a proportion of the 94.5% total mortality, predation ranges from 53% to 94%.

To translate these figures into kiwi rates of increase, the life-table in Table 2 assumes that (a) birds first breed 2 years after hatching (McLennan et al. 1996), (b) fecundity is 0.425 young/bird/year (McLennan et al. 1996, Table 3; Basse et al. 1999), (c) survival to breeding in Year 2 is the overall survival of young (currently 0.055 as above) multiplied by one year's 'adult' survival of 0.918, and (d) birds live for a potential maximum of 40 years, as recorded in captivity (J. McLennan, pers. comm.). Given these data, we solve the Euler equation to find the exponential rate of increase, r:

$$\sum_{x=0}^{x=40} l_x m_x e^{-rx} = 1$$

where $x$ = age (years), $l_x$ = survival to age $x$ and $m_x$ = productivity at age $x$. Table 2 shows only part of the life-table, which was run in Excel using trial values of r until the equation balanced (i.e. the sum of all the $l_x m_x e^{-rx}$ values equalled 1).

The result was an exponential growth rate of $r$ = –0.106, given the current parameters, which translates to a yearly finite rate of increase of exp(–0.106) = 0.90, or a 10% decline per year. This is slightly higher than the 5.8% decline obtained in the model of McLennan et al. (1996) and Basse et al. (1999).

To find out what change in predation allows the population to stabilise, means that we need to change the overall survival rate of young birds from 0.055 in Table 2 to a higher value that makes $r$ = 0. This higher rate is 0.23. The original overall survival rate of 0.055 is partitioned between predation and other mortality depending on the assumptions in Table 1, and for each of these assumptions there will be a different adjustment to survival from predators which will raise overall survival to 0.23 and make $r$ = 0. These assumptions are reiterated in Table 3, which also presents the calculated adjustments to the predation part which are needed for $r$ to increase from –0.106 to zero.

The 'maximum' predation scenario assumes that all disappeared birds were predated, the 'medium' scenario assumes that the disappeared birds suffered predation and 'other' mortality in proportion to these mortalities in birds whose fates were known, while the 'minimum' scenario assumes that no disappeared birds were predated. The last scenario results in a situation where even with

TABLE 2. KIWI LIFE-TABLE, BASED ON PARAMETERS IN MCLENNAN ET AL. (1996) AND BASSE ET AL. (1999); SEE TEXT. $l_x$ = SURVIVAL TO AGE $x$, $m_x$ = PRODUCTIVITY AT AGE x, AND r = EXPONENTIAL GROWTH RATE.

| $x$ (= AGE IN YEARS) | $l_x$ | $m_x$ | $e^{-rx}$ | $l_x m_x e^{-rx}$ |
|---|---|---|---|---|
| 0 | 1 | 0 | 1 | 0 |
| 1 | 0.055 | 0 | $e^{-r}$ | 0 |
| 2 | $0.055 \times 0.918$ | 0.425 | $e^{-2r}$ | $0.055 \times 0.918 \times 0.425 \times e^{-2r}$ |
| 3 | $0.055 \times 0.918^2$ | 0.425 | $e^{-3r}$ | $0.055 \times 0.918^2 \times 0.425 \times e^{-3r}$ |
| 4 | $0.055 \times 0.918^3$ | 0.425 | $e^{-4r}$ | $0.055 \times 0.918^3 \times 0.425 \times e^{-4r}$ |

100% removal of predators the population still declines (at a rate of 6.2% per year). This is because, if most of the observed substantial mortality of young birds (94.5%) is not due to predators, taking predators away will have little effect. Conversely, and apparently paradoxically, if predators account for most of this mortality, then it takes the smallest percentage reduction in predation to offset the 10% decline in the population—a 39% reduction in predation, hence a 39% reduction in stoat density. This 39% is calculated as follows. Firstly, given survival from non-predation sources of 0.5, survival from predation must be increased from 0.11 to 0.39 to give the overall increased survival rate of 0.23 (= 0.5 × 0.39, Table 3, columns 3–6). Secondly, this means that mortality from predation must be reduced from 0.89 to 0.54, a 39% reduction. If the most likely scenario is the 'medium' one, then by similar logic with a different non-predation survival rate of 0.335 (Table 3, column 3), a 63% reduction in predation, hence stoat density, eliminates the observed 10% decline in kiwi. All the estimated changes in predation levels translate into the same change in total survival of young from 5.5% to 23%. (column 5 in Table 3). This 23% compares closely with McLennan et al.'s and Basse et al.'s estimate of a 19% 'recruitment rate' (i.e. survival of young from hatching to adulthood—J. McLennan pers. comm.) necessary for population stability.

Our figure of a 39% minimum reduction in stoat density, given any assumed partitioning of mortality, is slightly higher than the 34% quoted by McLennan et al. (1996) but substantially lower than the 80% estimated by Basse et al. (1999) in years of high stoat density. Moreover, there seems little justification for the 'threshold' stoat density of 2 $km^{-2}$ suggested by the latter authors, given that the stoat densities in their study were completely unknown. Only an average proportional reduction in stoats required for kiwi persistence can be estimated, as above. Better estimates of how mortality from stoats varies from year to year, or of indices of stoat densities themselves that can be associated with known kiwi mortalities, would help overcome this problem but are obviously difficult to acquire. Even the estimated proportional reduction in predation is subject to considerable uncertainty and very sensitive to the assumptions about the fate of birds that disappear—if fewer of these fall victim to predators than is the case for birds of known fate, then the reduction in predation needed could be anywhere between 39% and 100% (Table 3).

Field observations suggest that while many kiwi populations (at least those inhabiting mixed podocarp–broadleaf forests in the North Island) are indeed

TABLE 3. ESTIMATED RATES OF KIWI PREDATION BY STOATS ASSUMING THREE DIFFERENT STOAT DENSITIES AT THE TIME THE MORTALITY ESTIMATES SUMMARISED IN TABLE 1 WERE OBTAINED. [1] NOTE: FOR THE 'MINIMUM' ASSUMPTION, THE POPULATION STILL DECLINES IN THE ABSENCE OF ANY PREDATION.

| PREDATION ASSUMPTION | OVERALL SURVIVAL | SURVIVAL (NON-PREDATION) | SURVIVAL (PREDATION) | OVERALL SURVIVAL NEEDED FOR r = 0 | SURVIVAL (PREDATION) NEEDED FOR r = 0 | % REDUCTION IN PREDATION NEEDED FOR r = 0 |
|---|---|---|---|---|---|---|
| Maximum | 0.055 | 0.500 | 0.110 | 0.230 | 0.390 | 39% |
| Medium | 0.055 | 0.335 | 0.164 | 0.230 | 0.630 | 63% |
| Minimum | 0.055 | 0.109 | 0.505 | 0.230 | 1.000 | 100%[1] |

undergoing a chronic decline, average rates of decline are generally perceived to be substantially less than –10% (Rod Hay pers. comm.). However, considerably more data have become available since the analyses of McLennan et al. (1996) and Basse et al. (1999) and these suggest that the above overall mortalities of young birds are, if anything, conservative (J. McLennan, pers. comm.). Given these uncertainties, current data appear inadequate for making sensible decisions about stoat management. There is certainly no reliable basis for any stoat density threshold for maintaining kiwi. The lack of any mechanistic understanding about key factors driving stoat densities in podocarp–broadleaf forest (see Section 2.3), compounds the problem and adds to the uncertainty through unpredictable yearly variations in mortality. Furthermore, immigration of stoats following control is a major problem, to the extent that even the apparently modest reductions in predation pressure suggested by the models may in practice be extremely difficult to achieve through trapping in years of high stoat abundance (J. McLennan, pers. comm.). Thus, to optimise management of stoats in order to protect kiwi, three things are needed. First and foremost is more data, particularly relating stoat density indices to kiwi mortality and on stoat density changes from year to year, including immigration and density-dependence. Second is analysis of kiwi data collected post-1996, using the above life-table approach and with reduced uncertainty about the contribution of predation to unknown mortality. Third is a more comprehensive modelling approach, based on simulation models of stoat density and the processes driving stoat density (including control), and linking changes in stoat density to chick and juvenile survival for kiwi. This would allow realistic levels of uncertainty arising from demographic variation, variation in the efficacy of control, and measurement error for model parameters to be built into predictions in such a way that the risk associated with alternative stoat management strategies can be considered directly. A model that attempts to do this for mohua in southern beech forest is described in the next section.

## 3.2 MOHUA

Elliott (1996a) elaborated an earlier model of stoat predation on mohua in southern beech forests, by incorporating the effect of periodic irruptions in stoat density and environmental, demographic, and stoat-related variation in fledgling production and the survival of fledglings and adults. The focus of Elliott's modelling was the influence that the frequency of stoat irruptions, carrying capacity (number of breeding pairs) for mohua, and the number of broods produced by mohua had on mohua population viability. Viability was measured as the probability that a mohua population would persist for the 100 years over which simulations ran. The frequency of stoat irruptions was set using a probability-based approach, which at each irruption determined the period until the next. Two density-dependent functions were used to limit mohua population growth: (1) a habitat-related constraint that limited the number of adult birds that could successfully form breeding pairs, and (2) a decline in overwinter survival of fledglings with increasing adult density.

Elliott's (1996a) model provides useful insights into the effect of stoat predation on the viability of single- and double-brooding mohua populations of different

size, and the effect that stoat control had on mohua viability. A weakness of the model (general to most stochastic models of population viability) is that measuring viability as the probability of population persistence implies that all factors that potentially threaten the population are encompassed within the demographic and environmental variation attributed to demographic rates, and the factors that are varied between model iterations (e.g. frequency of stoat eruptions, population carrying capacity etc.). For example, Elliott's modelling predicted that the probability of mohua population persistence increased dramatically when the interval between stoat irruptions exceeded about 5 years. However, the effect that variation in the frequency of stoat irruptions had on minimum or average mohua density was not considered. Given that smaller populations are inherently more exposed to extinction through stochastic mishaps than are larger populations (i.e. chance events leading to reduction of demographic rates below the range considered by the demographic and environmental variation applied in these models), this sort of information will be at least as important as the probability of mohua persistence (Caughley 1994; Caughley & Gunn 1996). Another limitation that Elliott's approach to modelling shares with that of Basse et al. (1999) is that the dynamics of the stoat population are treated implicitly. In Basse et al.'s model, stoat density is considered to be either constant or to vary only according to the reduction in density that control achieves. In Elliott's (1996a) model, variation in stoat density is included by varying the demographic rates that are influenced by stoat predation (fledging success and breeding female survival over summer), according to the stochastic frequency of stoat irruption. While this does not prevent these models representing the potential effects of stoat control on prey population dynamics, it assumes that variation in stoat density, independent of the effects of control, can be accurately monitored or predicted. While stoat eruptions can be predicted to at least some extent in beech forest systems, the timing of fluctuations in stoat density for populations in non-beech forests and the mechanisms driving these fluctuations remain largely unknown.

## 3.3 ALTERNATIVE MODELLING APPROACHES

Choquenot (unpubl. data) adapted the demographic approach employed by Elliott (1996a) to (1) develop a more mechanistic model for predicting fluctuations in stoat density in beech forests, and (2) consider how stoat control influenced **extinction risk** for mohua. Choquenot (unpubl. data) used this isocline or prey-dependent model to explore the effect of various stoat control strategies (based on different control efficacy and the use of environmental 'triggers' for implementing control) on the relative cost-effectiveness of control over simulations lasting 300 years. The model was based on a quantitative model linking semi-periodic high seedfall events in beech forests with variation in mouse density (Choquenot et al. 2000), the numerical response of stoats to changes in mouse density estimated by King (1983) (Fig. 10b), and estimates of the relationship between stoat density and mohua fledging success and summer adult female survival (Elliott 1996b; O'Donnell et al. 1996). Extinction risk for mohua was based on a modification of Burgman et al.'s (1993) concept of quasi-extinction probability, that estimates the probability that a threatened population will decline below some minimum threshold density. Quasi-extinction probabilities are a particularly useful way of considering how management

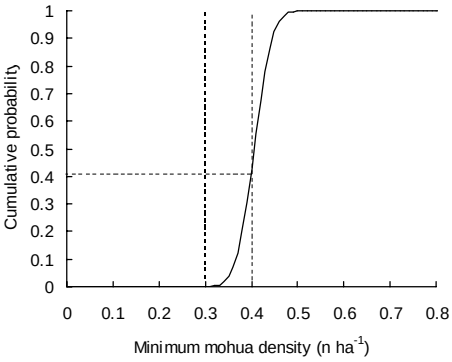


Figure 11. Cumulative probability of the density of mohua in beech forest falling below given levels when stoats density is reduced by an average of 90% every 2 years. The distribution was derived from 5000 iterations of a stochastic model of stoat and mohua dynamics in which stoat density is driven by changes in mouse density in response to periodic high seedfall events, and the dynamics of mohua are driven by a stochastic demographic model that incorporated the effects of stoat predation. The two dashed lines show the probability of mohua density falling below 0.3 and 0.4 per hectare.

influences the risk of a threatened species undergoing extinction, where the dynamics of the managed system vary stochastically. The concept is premised on the idea that because small populations are inherently at greater risk of extinction than larger populations (Caughley 1994), any management intervention that reduces the probability that the density of a threatened population will fall below some critical level, achieves some measurable conservation benefit. Hence, the benefit from any management intervention must be defined by the critical density above which a threatened population should be maintained, and an acceptable probability that this outcome will be achieved. Requiring conservation benefit to be defined in this way makes the concept of population viability much more tangible than does the probability of population persistence employed by other viability modelling approaches, including that of Elliott (1996a).

For example, conducting multiple iterations of some specified stoat management strategy, using Choquenot's (unpublished) model of the effect stoat control has on mohua populations, allows a cumulative probability distribution of minimum mohua density to be constructed. Figure 11 shows the cumulative probability of mohua density falling below a range of densities, when stoat control removes (on average) 90% of stoats, and is conducted every 2 years. This distribution can be used to establish the probability that the control strategy modelled would maintain a mohua population above any specified density. For example, under the modelled stoat control regime, the probability of mohua density falling below 0.3/ha is zero, while the probability of mohua falling below 0.4/ha is 0.42. Hence, if 0.3 mohua/ha was considered a critical lower density for the long-term viability of the species, the modelling suggests that this control strategy would successfully protect mohua. However, because controlling stoats every 2 years is an expensive undertaking and a zero probability of mohua falling below this critical density is very conservative, less-intensive control strategies might prove to be more cost-effective for mohua conservation. Figure 11 shows the cumulative probability distribution for minimum mohua densities, for stoat control conducted at fixed intervals of 1–10 years. If the interval between control operations is increased from 2 to 3 years, the probability of mohua density falling below 0.3/ha increases from zero to 0.63. Is a probability of 0.63 too high? If the critical density of 0.3/ha had been set for some biologically valid reason rather than being arbitrarily selected as it has been here, such a high risk of mohua falling below this level would not be desirable. However, the example serves to illustrate the point that this modelling approach allows both a biological decision

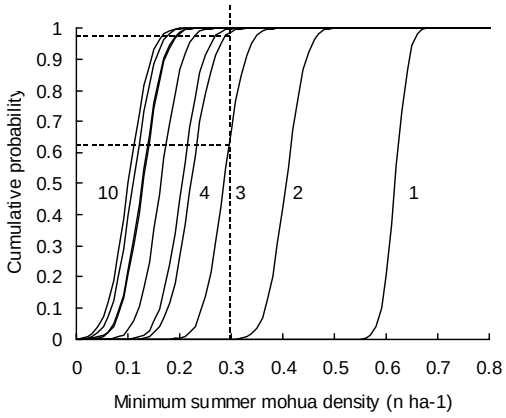


Figure 12. Cumulative probabilities of the density of mohua in beech forest falling below given levels when stoats density is reduced by an average of 90% every 1–10 years (numbers beside some distributions indicate years between stoat control operations). The distributions were derived from 5000 iterations of a stochastic model of stoat and mohua dynamics in which stoat density is driven by changes in mouse density in response to periodic high seedfall events, and the dynamics of mohua are driven by a stochastic demographic model that incorporated the effects of stoat predation. The dashed lines show the probability of mohua density falling below 0.3/ha when stoats are controlled every 3 or 4 years.

on critical population size, and a maximum acceptable risk of a population falling below that acceptable size, to be combined in setting a conservation outcome for stoat control. Presumably the critical minimum sizes and risks that would be set for real populations would reflect the relative importance of the threatened species, the relative significance that the population in question had for the viability of the species, the exposure each population had to other potential sources of extinction, and the opportunity costs (i.e. how much management on other species or populations is foregone to undertake more-intensive stoat control to benefit the population in question) that would accrue at progressively lower levels of risk. By developing families of probability distributions for a range of potential stoat control strategies, the strategy that achieves specified conservation outcomes most cheaply can be identified (Fig. 12).

## 3.4 PRIORITY RESEARCH FOR DEVELOPMENT OF BETTER PREDICTIVE MODELS FOR THE IMPACTS OF STOAT CONTROL ON THREATENED PREY

Identification of the most efficient control strategies for stoats requires some capacity to predict the costs and effectiveness of the range of alternative strategies that existing and emerging control methods offer. However, prediction of cost-effectiveness for stoat control is complicated by (1) their irruptive dynamics, (2) the potential for complex interactions with other pest species (rodents and predators), and (3) the fact that their impacts on most extant prey species appear to be chronic rather than acute. With these difficulties in mind, the simulation approaches developed by Elliott (1996a) and extended by Choquenot (unpubl. data) to evaluate the cost-effectiveness of stoat control to enhance mohua viability appear to provide the most promising modelling framework for evaluating the efficiency of alternative stoat control strategies. However, the highly prescriptive management and policy advice that can be gleaned from these models reflects the quality of data available to predict variation in controlled and uncontrolled stoat density in beech forest, and to link this variation to the demography of mohua. It follows that, where data linking variation in stoat density to the demography of other beech-forest birds is available, similar analyses of the cost-effectiveness of alternative stoat control strategies can be undertaken. While there are good demographic data for several bird species potentially at risk in beech forest, robust estimates of the effect of stoat predation on demographic rates are available only for yellow-crowned parakeets (*Cyanoramphus auriceps*) in Fiordland (Elliott et al. 1996). Obtaining estimates of demographic rates across years of high and low stoat density for other beech-forest birds that are known or suspected to be in decline should be viewed as the highest priority research need for improving stoat management in this habitat. If these estimates were available, existing data on mechanisms driving stoat densities in beech forest, and of the efficacy of available control techniques, would be sufficient to evaluate the relative costs and effectiveness of most conceivable stoat management strategies.

The capacity to develop models that can predict the cost-effectiveness of stoat control in non-beech habitats (principally podocarp–broadleaf forest) is current limited by (1) an inadequate understanding of the mechanisms driving variation

in stoat density in this habitat (see Section 2.3), and (2) the lack of unqualified data linking variation in stoat density to demographic rates of prey at risk (see Section 3.1). Hence, priority areas for research to support development of these models should focus on (1) identifying and quantifying the mechanisms that drive variation in stoat density in podocarp–broadleaf forest, and (2) assessing the effect that stoat predation has on the demographic rates of key prey species inhabiting podocarp–broadleaf forests. Circumstantial evidence of a numerical link between rat and stoat density in podocarp–broadleaf forest was described in Section 2.3. Given the potential that an understanding of this linkage has for predicting variation in stoat density in this habitat, immediate research priorities should include:

- validation/testing of the association between rat and stoat density in the absence of rodent poisoning in podocarp–broadleaf forest
- identification of factors driving variation in rat density in podocarp–broadleaf forest
- estimates of the effects that changes in stoat density have on key demographic rates of prey species at risk in podocarp–broadleaf forest (e.g. kiwi, kaka, robin, kereru).

## 3.5 ADVERSE CONSEQUENCES OF STOAT CONTROL

While the primary aim of stoat control is to enhance the viability of native prey that are at risk, stoats consume many other species, some of which are important conservation pests in their own right. If stoat control leads to increases in these species, the direct benefits of reduced stoat predation on native prey have to be discounted to an extent depending on the indirect impacts of these other pests. For example, if stoat control to reduce impact on kiwi elevates the density of rats, the increased impact of rats on native birds such as kokako will reduce the net conservation benefit from stoat control. This phenomenon is called 'mesopredator release', and is particularly prevalent where control of introduced higher-order predators such as mustelids and cats (so-called 'superpredators') leads to increased densities of rodents in insular ecosystems (Soule et al. 1988). In New Zealand, the obvious mesopredators that could be released by stoat control would be rats, mice and weasels. While all three species are known to prey on native fauna, the effect of rats on native birds and of mice on invertebrates is perhaps the most likely conservation consequence of mesopredator release[1].

The evidence for and against stoat control 'releasing' rodents in New Zealand is equivocal. Both rat and mouse density are substantially higher in the Eglinton Valley, where stoats are controlled through trapping, than in the Hollyford Valley where stoats are not controlled. However, this comparison potentially confounds site and predation effects, and should be validated under more controlled circumstances. Murphy & Bradfield (1992) and Murphy et al. (1998)

[1] In strict definitional terms a mesopredator should share the same primary prey as a superpredator (Soule et al. 1988). However, in applying the concept to conservation problems, the generic effect control of a superpredator has for general predation by mesopredators is also called mesopredator release (Soule et al. 1988; Courchamp et al. 1999).

found that offtake of rats by stoats was proportional to rat availability, suggesting that stoats had, at most, a limited capacity to reduce rat density.

To comprehend fully the direct and indirect consequences of stoat control, the reciprocal influence stoats and rodents exert over each other's abundance, and the effect that changes in stoat and rodent density has on prey at risk need to be predicted. Courchamp & Sugihara (1999) and Courchamp et al. (1999) formulated a general model for predicting the consequences of mesopredator release for threatened prey populations. The model consists of three linked differential equations, that predict simultaneous variation in the abundance of prey, a mesopredator, and a superpredator. A diagram of the model is shown in Figure 13. While the growth of each population in Courchamp's model is under intrinsic (density-dependent) control rather than control through a predator's numerical response, the three populations are made to 'interact' by making the carrying capacity of the superpredator entirely dependent on the prevailing density of the mesopredator and prey, and the carrying capacity of the mesopredator partially dependent on the prevailing density of the prey. A more explicit form of interaction between these three populations would require functional and numerical responses to be elaborated for superpredators and mesopredators (Fig. 14).

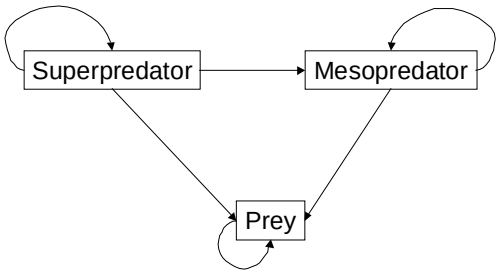


Figure 13. Diagram of the mathematical model of a superpredator–mesopredator–prey system, developed by Courchamp et al. (1999). Each box represents a population and arrows represent fluxes between and within populations. Straight arrows represent predation rates and curved arrows intrinsic growth rates.

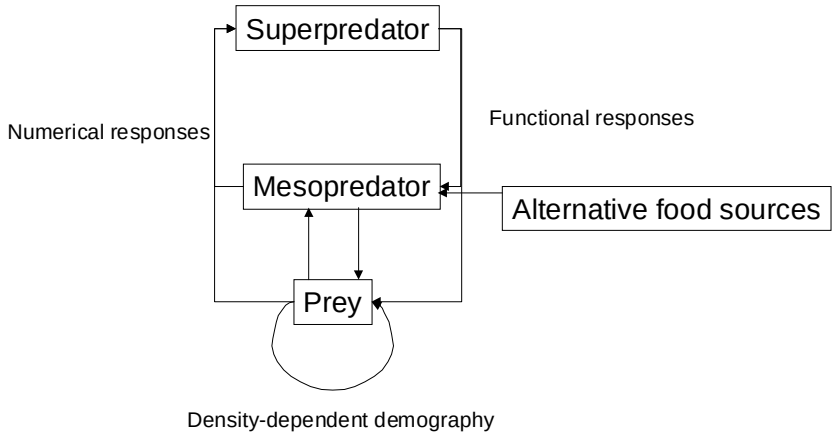


Figure 14. A more explicit model of a superpredator–mesopredator–prey system, which elaborates functional and numerical responses for superpredators and mesopredators.

To construct either model so that the net effect of stoat control on threatened prey populations can be predicted (i.e. the sum of reduced predation by stoats and increased predation through mesopredator release) would require numerical increases in the abundance of mesopredators and their offtake of threatened prey to be monitored on sites where stoats are controlled and sites where they are not controlled. Because of the interdependent dynamics of stoats and their rodent prey, such an experiment would need to be conducted through an irruptive oscillation in rodent and stoat density.

A more insidious potential consequence of stoat control is an increase in other predators such as weasels, cats, hedgehogs and ferrets through reduced

competition for available prey. Despite impressive elaboration to consider a vast range of contingent detail, all modelling frameworks for considering competition are based on the original models of Lotka (Tilman 1982). These models require direct estimation of competition coefficients using removal experiments. No explicit experiments have been undertaken to quantify competition between stoats and other predators, and the data from control programmes that regularly remove stoats and other predators are compromised through removal of all predators encountered. A similar experiment to that described to estimate the extent of mesopredator release would allow the degree of competition between stoats and other predators to be assessed. Data from such an experiment would allow the effect of stoat control on the abundance of other predators, and hence the net conservation benefit accruing from stoat control, to be predicted.

# 4. Modelling stoat control

## 4.1 MODELS FOR EFFECTS OF CULLING AND NON-DISSEMINATING FERTILITY CONTROL

Few attempts have been made specifically to compare the effects of sterilisation and culling on wildlife populations using models. However, a number of models have considered fertility control alone and its effects on specific populations. Sturtevant (1970) plotted the theoretical effects of a single 70% cull and 70% sterilisation on a feral pigeon population, while Knipling & McGuire (1972) formulated a difference-equation model for sterilisation of rats, which was subsequently applied to the brushtail possum (*Trichosurus vulpecula*) by Spurr (1981). These and other species-specific studies of sterilisation were reviewed by Bomford (1990). More recent work of this kind includes models for sterilisation of feral horses (Garrott 1991; Garrott & Siniff 1992), foxes (Saunders & Choquenot 1995; Pech et al. 1997), white-tailed deer (Seagle & Close 1996); and badgers (White et al. 1997).

In terms of general, strategic models, Stenseth (1981) formulated simple mathematical models for birth and death processes in localised populations linked by migration. He used these to predict the optimal control strategy for a population based on its demography, specifically whether it is *r*-selected or *K*-selected. Although not specifically designed to compare culling with sterilisation, Stenseth's models led to the conclusion that a reduction in reproductive potential is likely to be the optimal method of control for populations with a high mortality rate and therefore a high population turnover rate. This was similar to a conclusion of Hone (1992) based on examination of the relationship between the rate of increase of populations and their fecundity and survivorship. Caughley et al. (1992) considered the effects of dominance and social structure on the impact that sterilisation of females has on the realised productivity of a population. They showed that productivity was generally reduced to a lesser extent than the proportional sterilisation rate, and that in one case sterilisation actually enhanced productivity.

Barlow et al. (1997) examined the relative effectiveness of sterilisation and culling and, unlike previous studies, they considered different assumptions about mating systems and the way density-dependence acts in the populations. Furthermore, they considered the effects of control, not just on the population's rate of increase (e.g. Stenseth 1981; Hone 1992) but also, more importantly and of more practical relevance, its effects on their average abundance. Specifically, the following were assumed: (1) mating systems represented by polygamy, 'harem'systems and monogamy; (2) sterilisation applied to either or both sexes; (3) linear density-dependence acting through mortality or recruitment (defined as fecundity plus juvenile mortality); (4) continuous, logistic population growth modelled by differential equations; (5) continuous rather than episodic controls (for example, through trapping or the use of poison, chemosterilant or immunocontraceptive baits, or by a micro-organism capable of causing immuno-contraception); (6) that sterilisation is life-long and does not affect behaviour; and (7) that all young are born fertile. Results were presented in terms of (1) the extent of control required to achieve a given level of population suppression, and (2) the effect of a given level of control on the rate of population decline and final level of suppression. Each of these was assessed for culling and for two sterilisation/mating scenarios that give significantly different outcomes, namely,

Scenario 1: (a) polygamous mating with females or both sexes sterilised, or
(b) monogamous mating and either males or females sterilised, or
(c) 'harem' systems, in which a number of females mate exclusively with each dominant male, and either males or females are sterilised;

Scenario 2: (a) monogamous mating with both sexes sterilised, or
(b) 'harem' systems with both sexes sterilised.

The more complex social systems considered by Caughley et al. (1992) could be included in the model by expressing them in terms of the degree of polygamy or monogamy they imply. Thus, family group structures (e.g. foxes and badgers) may be closer to monogamy than polygamy, whereas the reverse may be true for 'harem' systems with polyoestrous females that breed with subdominants if the dominant male is sterilised (e.g. feral horses).

A third alternative to scenarios 1 and 2 is polygamous mating and sterilisation of males. However, this is likely to be ineffective in reducing density (Barlow et al. 1997) and was not considered further in the models.

The models in Barlow et al. (1997) suggested that the mating system has a considerable effect on the relative efficacy of sterilisation, and that previous models for sterilisation may have overestimated its impact by assuming idealised monogamous mating. In general, and except for populations with monogamous mating or 'harem' systems and both sexes sterilised, culling gives a more rapid reduction in density than does sterilisation. However, the long-term degree of suppression obtained with the same levels of control applied are likely to be similar. Populations with density-dependent mortality will be reduced by sterilisation more quickly than those regulated by density-dependent recruitment, and the effect of a given proportion sterile on the steady-state density is greater in the first case than in the second. However, the effect of a given continuous sterilisation rate (e.g. baiting effort) on density is the same in both cases.

Hone (1992) suggested a need further to explore the interactions between fertility control and other control methods. The Barlow et al. (1997) paper provided a first step, by comparing culling with sterilisation, although it did not consider their interaction. The reasons for this were, firstly, that if one strategy is in some way superior to the other, it is unlikely that a combination will be superior to both, given the same conditions. Secondly, effects of both strategies on the rate of increase ($r$) tended to be linear in the models, and in this sense additive. This contrasts with Garrott (1991) and Hone (1992, 1994), who drew attention to a non-linear relationship between fecundity and $r$, in the first case from a study of feral horses and in the second from age-specific life-tables approximating to those for tahr (*Hemitragus jemlahicus*) and feral pigs. The difference is a simple consequence of the difference in the models. Barlow et al. (1997) assumed continuous breeding and considered proportional reductions in fertility to apply to the instantaneous birth rate, $a$, rather than to fecundity at the birth pulse. In contrast, Garrot (1991) and Hone (1992) used birth-pulse models and related $r$ to fecundity. Since $r$ is linearly related to the instantaneous birth rate, $a$, and since fecundity = $\exp(a)$, then clearly $r$ varies as log(fecundity). More specifically,

$$r \approx \ln [1 + (1 - Q) F] - b \quad (2)$$

where $F$ is fecundity per head of the population (= $\exp(a)$) per unit time (i.e. per birth pulse), averaged over all ages, $Q$ is the proportion of females sterile and $b$ is the instantaneous mortality rate, again averaged over all ages. Given density-dependence, the equation is:

$$r \approx \ln [1 + (1 - Q)\exp(f(N)] - g(N) \quad (3)$$

where $f(N)$ is the relationship between fecundity and density and $g(N)$ that between mortality and density. Equation 2 approximates Hone's (1992) detailed age-specific life-tables, although in this case more meaningful values of $F$ and $b$ are obtained by incorporating the substantial first-year mortality (see Hone's (1992) Table 1) within a reduced value of $F$. In practice, the difference between this birth-pulse model and the continuous models used in Barlow et al. (1997) is small, particularly for average fecundities less than about 1 per head of the population. For instance, the finite rate of increase (i.e. ratio of densities from one birth pulse to the next) is about 3% greater for a birth-pulse model than for a continuous-breeding model with $F = 0.65$ ($a = 0.5$) and 50% of females sterile or not breeding. The relative difference declines to 2% as the proportion sterile increases to 80%.

As a result of the non-linear relationship between $r$ and fecundity, Hone (1992, 1994) suggested that sterilisation would be most effective when $r$ was already low, as in a species with low fecundity or a population that was already being controlled by other means (Hone 1992). While it is true that linear decreases in fecundity give progressively greater linear decreases in $r$ as both decline, a given proportional reduction in fecundity (e.g. due to sterilisation) gives approximately the same linear reduction in $r$, whatever the initial value of both (e.g. in the examples in Hone's Fig. 2), so in this sense the above conclusions do not hold. Moreover, a greater or lesser effectiveness of sterilisation does not preclude the possibility that culling would be more effective under the same circumstances.

Stenseth (1981) suggested that sterilisation was likely to be an optimum control strategy for populations with a high death rate, as did Hone (1992, Fig. 2 and conclusions). Barlow et al. (1997) indicate a slightly more specific conclusion,

that a high death rate relative to birth rate enhances the impact of a given level of sterilisation (Table 4, row 3). The reason is clear from Equation 2 but is most easily visualised by considering an even simpler equation, namely:

$$r = a - b \qquad (4)$$

If $b$ is large relative to $a$, it takes only a small proportional reduction in $a$ to make its value equal $b$ and $r$ equal zero. If $a$ is reduced to zero by sterilisation, the rate of decline is proportional to $b$ and is therefore greatest for species with high death rates. Again, this does not imply that sterilisation is the optimum method of control for species with these characteristics. On the contrary, culling will still give a greater initial rate of decline and the same long-term degree of suppression as sterilisation, for most mating/sterilisation systems (scenario 1) and irrespective of fecundity or the value of $r$. The greater impact of culling on the rate of decline is obvious: a 100% cull will instantaneously eliminate a population ($r \rightarrow -\infty$), whereas 100% sterilisation gives a maximum rate of decline of $-b$, whatever the mating system.

In terms of the long-term (steady-state) effects of given levels of culling and sterilisation, if the population is to be eliminated, the culling rate $c$ or the sterilisation rate $s$ must exceed $r$ for most mating/sterilisation combinations (i.e. scenario 1: polygamous mating with females sterilised or monogamy/harem systems with either sex sterilised). For example, intensive trapping at a rate in excess of $r$ was successfully used to eradicate brushtail possums from Kapiti Island, New Zealand (Cowan 1993). As Table 4 shows, a given sterilisation rate yields a higher proportion sterile with recruitment density-dependent than with mortality density-dependent, because the lower mortality in the first case means a smaller loss of sterilised animals. On the other hand, the effect of a given proportion sterile on population density is smaller in the first case than in the second (Table 4), because the absolute impact of a reduction in birth rate is discounted by the fact that density-dependent recruitment reduces the birth rate itself. In spite of this, the proportion sterile required to cause complete suppression is the same whatever the nature of density-dependence (= $r/a$). These two opposing effects of density-dependence cancel each other out when considering the effect of the instantaneous sterilisation rate, as opposed to the proportion sterile, on population density. Irrespective of the density-dependence,

TABLE 4. FROM BARLOW ET AL. (1997), RELATIONSHIPS BETWEEN PROPORTIONAL REDUCTION IN POPULATION DENSITY ($p = 1 - N/K$ WHERE $N$ AND $K$ ARE EQUILIBRIUM DENSITIES WITH AND WITHOUT CONTROL), INSTANTANEOUS RATE OF STERILISATION ($s$) AND PROPORTION STERILE ($Q$ = PROPORTION OF FEMALES NOT REPRODUCING), FOR MATING SCENARIO 1 AND DIFFERENT TYPES OF DENSITY-DEPENDENCE. $a$ = MAXIMUM INSTANTANEOUS BIRTH RATE, $b$ = MINIMUM INSTANTANEOUS DEATH RATE, $r$ = MAXIMUM INSTANTANEOUS (INTRINSIC) RATE OF INCREASE.

| | DENSITY-DEPENDENT MORTALITY | DENSITY-DEPENDENT RECRUITMENT |
|---|---|---|
| Suppression ($p$) given $s$ | $s/r$ | $s/r$ |
| Level of sterilisation ($Q$) given $s$ | $s/a$ | $s/(b + s)$ |
| Suppression ($p$) given $Q$ | $aQ/r$ | $(a/r - 1)Q/(1 - Q)$ |
| $Q$ for 100% suppression | $r/a$ | $r/a$ |
| Rate ($s$) to give suppression $p$ | $rp$ | $rp$ |
| Level ($Q$) to give suppression $p$ | $rp/a$ | $rp/(b + rp)$ |

the proportion by which density is reduced is $s/r$. The models suggest that the critical sterilisation rate for (theoretical) elimination of the population may be less than $r$ for monogamy/harem systems in which both sexes are sterilised (scenario 2). In this case, too, a control rate of less than $r$ results in a more rapid suppression and greater long-term reduction in density than does culling.

In terms of the rate of control required to provide a given level of population suppression, the results are again the same for culling or for sterilisation for most mating/sterilisation systems (scenario 1). For monogamy/harem systems and both sexes sterilised (scenario 2), the control effort required is less than that for either culling, or for sterilisation under scenario 1. The reduction in control effort is because of an additional 'intrinsic' reduction in the birth rate due not to the imposed sterilisation regime but to the proportion of fertile females failing to reproduce because a proportion of their monogamous partners are also sterile.

Other sterilisation models have suggested more optimistic results from sterilisation as a method of control. However, this appears to be due to an assumption of idealised monogamy within these modelled populations (e.g. Sturtevant 1970; Knipling & McGuire 1972). In the case of Knipling & McGuire (1972), the monogamy is not explicit but is dictated by the assumption that mating with a sterile male prevents mating by a fertile one. Barlow et al. (1997) suggest that such models may considerably overestimate the efficacy of sterilisation for populations which are not strictly monogamous. On the other hand, idealised polygamy, as modelled here, assumes unlimited random matings within the population, a scenario which is as unrealistic as idealised monogamy for most natural populations. On this basis and in agreement with Bomford (1990), Barlow et al. (1997) concluded that sterilisation of males alone is likely to be ineffective in reducing population size. Real mating systems can be expected to fall somewhere between these two extremes. However, Caughley et al. (1992) demonstrated that most mating systems and social systems involving dominance hierarchies will tend to further reduce the effectiveness of actual sterilisation regimes in comparison with quantitative predictions from simple population models.

Bomford (1990) criticised previous models of sterilisation and culling for not including potential compensatory changes in natality, mortality, immigration, and dispersal, following a control operation. Even now, few fertility control models appear to consider density-dependence explicitly, although Saunders & Choquenot (1995) addressed the question in relation to foxes by considering hypothetically the extent to which different forms and degrees of possible compensation in the populations could offset reductions in $r$ due to sterilisation. Barlow et al. (1997) included such changes through density-dependent responses in recruitment and mortality functions. Moreover, their basic models are sufficiently general to be applicable to populations with a wide range of density responses, from density-independence to more complex density-dependence such as the $\theta$-logistic response (Gilpin et al. 1976).

Other forms of compensatory changes are clearly possible, such as increased breeding success of non-sterile animals, or reduced mortality for sterilised individuals. The density-dependence may also be more complex. For example, if juvenile survival depends on adult rather than total density, sterilisation will be considerably more effective since it leaves the adult density unchanged in the short term. Hence, not only are fewer young born but their density-dependent survival remains initially low. Finally, age-structure may have a considerable

influence on the above conclusions from non-age-structured models (N.D. Barlow, unpubl. data).

In summary, the simple population models in Barlow et al. (1997) allow prediction both of the effects of given intensities of sustained culling and sterilisation on population size, and of the intensities of control required to achieve given levels of suppression. The models suggest that sterilisation is less effective than culling at reducing populations in the short term. In the long term, the effect depends on the sterilisation regime and mating system. In most cases sterilisation and culling give the same long-term suppression, but for a monogamous population with both sexes sterilised, the level of suppression is considerably greater than for culling or for other mating system and sterilisation regimes. In the short term, sterilisation will be more effective in reducing density if populations are regulated by density-dependent mortality than if they are regulated by density-dependent recruitment, whereas the impact of culling is unaffected by the nature of density-dependence.

Considering the impact of a given, sustained proportion sterile ($Q$) on population density, the proportion ($P$) by which the density is suppressed below $K$ is given by:

$$P = 1 - (1 - aQ/r)^{1/\theta} \qquad (5)$$

where $\theta$ describes the non-linearity of any density-dependence ($\theta = 1$ corresponds to linear or logistic density-dependence). As Equation 5 indicates, a given proportion sterile has the greatest effect on population density when the birth rate ($a$) is high relative to $r$. Conversely, a low ratio for $a/r$ implies that a high level of sterilisation is necessary to achieve a given suppression of density. To eliminate a population (theoretically) requires a proportion $r/a$ of breeders to be sterile. Non-linearity in density-dependence ($\theta > 1$) will also reduce the impact of a given proportion sterile on population density, causing the relationship between $P$ and $Q$ to be non-linear and concave-up: initial increments in the proportion sterile will have a small effect on population density but this will rapidly increase as the sterilisation level increases (see below).

Equation 5 represents a logistic-type model with density-dependence acting on mortality. Populations regulated by density-dependent mortality will tend to be reduced more quickly by sterilisation than those regulated by density-dependent recruitment, though the final level of suppression achieved is the same in both cases. However, a given proportion sterile will have a smaller impact on population density in the case of density-dependent recruitment. The reason is that, in this case, the birth rate is itself reduced at high density, so a proportional reduction in birth rate will have a smaller absolute effect than if mortality is density-dependent and birth rate density-independent. A significant pre-reproductive period in an animal's development also reduces the impact on population density of a given proportion of adults sterile (N.D. Barlow, unpublished).

Finally, social effects and mating systems have a considerable impact on the efficacy of sterilisation. Monogamous mating or 'harem systems', and polygamous mating represent opposite extremes, with sterilisation most effective in the former, particularly when targeted at dominant animals or both sexes. Caughley et al. (1992) examined a variety of different dominance hierarchies and found that, in most cases, productivity was likely to be reduced to a lesser extent than the proportional sterilisation rate.

## 4.2 MODELS FOR DISSEMINATING FERTILITY CONTROL

A recent review of biological control of vertebrates, including models, is provided by Hickling (in press). There are still few published models for disseminating immunocontraceptive systems. The first (Barlow 1994) deliberately simplified the immunological and physiological issues that determine the probability that an animal infected with the immunocontraceptive vector is successfully sterilised. In doing so, it made this one of the key variables and addressed the fundamental question: what is the effect of a given success rate for sterilisation of animals infected with the vector, and what success rate is required to achieve desired levels of population reduction?

Barlow (1997) expanded this model from its original treatment of sexually transmitted, non-pathogenic viral vectors, to include non-sexually transmitted vectors, a broader consideration of the effects of pathogenicity and recovery, changes in host density-dependence, and the effect of the original sexually transmitted vector but with a lag in sterilisation from one transmission event (mating) to the next. This was primarily in the context of possums, but consideration was briefly given to rabbits as a contrasting species with a much higher intrinsic rate of increase.

Including the effect of a vector at an equilibrium prevalence $y$, which causes an additional pest mortality of $\alpha$, into the above equation for suppression due to sterilisation gives:

$$P = 1 - [1 - (y/r)(aQ + \alpha)]^{1/\theta} \quad (6)$$

Equation 6 shows that, in addition to the effects discussed above under non-disseminating fertility control (Equation 5), suppression is enhanced by high disease prevalence and a high mortality rate from the vector.

The models showed that, for a sexually transmitted herpes-type viral vector in possums (Barlow 1994), the percent suppression of possum density related non-linearly to the percentage of infected animals sterile at mating; it requires a high level of sterilisation to achieve high levels of suppression (as indicated by Equation 6). Failure to attain these targets yields much poorer control. Nearly 80% of animals carrying the vector must be sterile at mating to give 100% (theoretical) suppression, with a contact rate of 1. The figure declines to 66% as the contact rate tends to infinity. Mathematically, this is equivalent to $r/a$, the proportion of the population that must be sterile to reduce density to zero (see above), given that prevalence of the vector is 100% in the absence of mortality or recovery and for high contact rates. These are difficult targets to meet, given that the contact rate includes the probability that an infected animal is infectious and that the percentage of infected animals successfully sterilised relies on the presence of sustained or repeated high levels of antigens within the average infected (not infectious) host, as well as an excellent immunocontraceptive response. The non-linearity in the suppression versus percent-sterile relationship is almost entirely due to the assumption of non-linear density-dependence in the possum (Barlow 1991, 1994), such that $\theta = 3$. As indicated above, in the context of the effect on density of a given proportion sterile, this effect is greater for linear (logistic) than for non-linear, asymmetric density-dependence. In the same way, the outcome of vectored immuno-

contraception is more encouraging if the density-dependence is linear. The proportion of infected animals that must be sterile to give 100% suppression remains the same, but no longer is the level of suppression obtained so critically dependent on reaching these high targets: there is a considerably greater reduction in density associated with any given proportion of infected animals successfully sterilised.

Even a modest mortality caused by the vector (10% per year) greatly enhances the effect, suggesting that a pathogenic agent is probably more effective than an immunocontraceptive one in this case. The same would not be true for a species with a higher $r$ value, such as rabbits, for reasons given above: the higher the rate of increase, the more effective sterilisation becomes relative to culling or mortality. Conversely, very modest recovery rates from the vector (5% per year) prejudice success and no significant suppression is possible. When mortality and recovery are combined, the effect is closer to that for recovery alone than for mortality, and the impact of immunocontraception is reduced compared with that in the absence of both processes.

For a non-sexually transmitted vector, less suppression is possible, even with the most optimistic assumptions about levels of sterilisation achievable given infection. The reason is the difference in realised contact rates as population density declines. With non-sexual transmission the contact rate is assumed to decline linearly with density, whereas for sexual transmission it is assumed to remain constant (see above). The real situations for both transmission routes probably lie between these extremes, but not so much as to dramatically enhance the results for non-sexual transmission. Again, adding mortality from the vector enhances suppression except at the highest levels of sterilisation, and recovery from infection suppresses the effect.

Returning to sexual transmission, the effects of two changes in the basic model were examined: a very significant increase in maximum contact rate (from 1 to 4 per year); and a year's delay between infection with the vector and effective sterilisation. Increasing the contact rate had a non-linear effect, dramatically increasing the level of suppression but only with high percentages sterilised. The lag from infection to sterilisation had exactly the reverse effect. This is because of the mortality of sterilised individuals during the year between infection and reproduction. Viewed in terms of the proportion that must be sterilised in order to give a theoretical 100% suppression, this is $r/a$ if the sterilisation is immediate (as above), but $(r/a)(1 + b)$ if there is a 1-year lag. At low population densities the death rate is $b$, so the necessary proportion sterile must be inflated by $1/(1 + b)$ to allow for mortality over the year between the proportion becoming sterile and the effect on birth rate.

Whatever type of vector is employed, immunocontraception is slow to take effect, at least in possums. This does not invalidate its use. Rather, it suggests that there may be a need for integrated control, based on intensive control to obtain a rapid reduction in density once the immunocontraceptive vector is established in the population, combined with vectored immunocontraception to act as a subsequent 'maintenance control' and prevent the population from recovering (Barlow 1994). The intensive control could be through conventional methods or a much more intensive but short-term sterilisation programme. Clearly, optimum design of an integrated control regime demands a good knowledge of the epidemiology of the vector.

In summary, success requires that the genetically engineered vector persists in the population, that it attains a high prevalence, that this translates to a high proportion of animals sterilised, and finally that this leads to significant suppression of the population. Persistence is enhanced by the (local) absence of competing vector strains or a higher basic reproductive rate for the new strain if a wild-type already exists. High prevalence is also associated with a high basic reproductive rate, but is prejudiced by the spatial patchiness that appears to characterise many diseases. The probability that an infected host is sterilised is a physiological issue, but the translation from a given proportion sterile to a suppression of density is an ecological question. Sterilisation is most effective relative to other control options when the intrinsic rate of increase is high. A given proportion sterile yields greatest suppression when the birth rate is high relative to $r$, density-dependence acts on mortality rather than recruitment, density-dependence is linear rather than non-linear, and there is no pre-reproductive period.

In terms of the overall effect, and using possums as an example, a sexually transmitted vector is likely to be superior to a non-sexually transmitted one, both because the contact rate is higher at low densities and because multiple matings following sterilisation may increase the contact rate, the basic reproductive rate, and hence the vector's competitive ability. Even with a sexually transmitted vector, at least 66% of infected possums must be sterile at mating for complete suppression and this increases by a further 10% if there is a year's lag, from one mating to the next, between infection and sterilisation. If density-dependence is non-linear and these targets are not met, the level of population suppression achieved drops dramatically. The impact of immunocontraception is significantly enhanced if the vector causes limited mortality of the host, but is compromised to varying extents by either the existence of an immune class of hosts or spatial aggregation of the vector. The latter depresses overall prevalence, hence its overall effect on the host (Barlow 1991). To ascertain its practical significance would ideally require field measurements of spatial variation in the vector's prevalence. However, it is possible that stochastic models of the host/vector interaction, containing an explicit spatial component, could yield some useful insights into the likelihood of patchiness developing in the local distribution of disease, and the consequent impact of such patchiness on the populations (G. Hood, pers. comm.).

The targets identified by the models for immunocontraception of possums are qualitatively similar to those applying to any population and can be expressed relatively simply. To reduce possum populations to very low levels, at which density-dependent mortality is insignificant, the birth rate (0.305) must be reduced to match the minimum or density-independent death rate (0.105). This represents a 66% reduction in the birth rate, achieved if 66% of females are sterile at mating. If a microbial vector such as a herpes virus has a prevalence of, say, 90%, then it must successfully sterilise $100 \times 66/90 = 73\%$ of females carrying the vector. More seriously, for vectored immunocontraception to succeed in virtually eliminating the population, the prevalence of the vector must at least equal the required reduction in the birth rate, namely 66%. To reduce the population by 50% rather than (theoretically) eliminate it still requires a vector prevalence of at least 57%, since this is the birth rate reduction required to equal the higher death rate at 0.5 $K$ rather than zero density (Barlow 1994).

Although complete suppression of pest populations may be difficult to achieve through immunocontraceptive control of fertility alone, even partially successful immunocontraception may contribute usefully to integrated control by eroding part of the pest population's compensatory potential. As a biological control it also obviates the need for repeated application, the adverse impact of immigration on localised traditional control, and the animal welfare and environmental problems associated with trapping and poisoning.

## 4.3 MODELS FOR BIOLOGICAL CONTROL USING PATHOGENS

Until very recently, and with the notable exception of myxomatosis, biological control by pathogens has been largely focused on insects, both in theory and in practice. The models tend to be rather different from those for pathogens in vertebrates since there is typically no 'recovered and immune' class of insects. The few models published for biological control of vertebrates (see Hickling, in press) include the following systems: myxomatosis, the most comprehensive of which is Dwyer et al. (1990); the nematode *Capillaria hepatica* as a control for mouse outbreaks in Australia (McCallum & Singleton 1989); rabbit haemorrhage disease (RHD) and rabbits in New Zealand (Barlow & Kean 1998); and viruses for the control of cats on islands (Courchamp & Sugihara 1999). In spite of this limited literature, some lessons can also be taken from the vastly greater number of models for control of wildlife diseases, biological control of the host being the inverse of control of the disease. The immunocontraception model of Barlow (1994, 1997) also provides some clues about control by pathogens, notably the fact that when only a small degree of disease-induced mortality was assigned to the otherwise benign herpes vector, then control was radically improved and occurred much more quickly. Control by the right kind of pathogen is likely to be much more effective than control by immunocontraception, at least in theory. The practical problem remains, however, that the technology is likely to be less publicly acceptable. Considering models such as that for a chronic pathogen (bovine Tb in possums, Barlow 1991) and a more acute, virulent one (RHD in rabbits, Barlow & Kean 1998), suggests that an ideal pathogen for biological control is likely to be more like RHD than Tb. The latter has a low prevalence in the population and little effect on the host, apparently because of spatial patchiness or other heterogeneities in disease transmission. RHD also has a low prevalence, even during epidemics, but its impact is considerable, resulting in a 60–70% sustained reduction in host density. Possibly because of its greater transmission rate, patchiness of the disease seems to be much less of a constraint to its impact. The crucial difference between the two diseases is that RHD has both a high transmission rate and a high mortality rate, and also a moderate persistence in the environment (a half-life of about 2 weeks), which allows for the possibility of persistent biological control. Without this (as in the case of Tb), RHD would cause a massive initial knockdown of the host but fail to persist and would need to be repeatedly reintroduced. Consequently it appears that the ideal pathogen for biological control should have a high transmission rate, cause high mortality, and have some degree of environmental persistence. There may be other combinations of characteristics that give both high and persistent host

suppression, and this is an ideal area for further modelling to explore, without the need for additional data.

## 4.4 MODELS FOR STOAT DYNAMICS

It is clearly fruitless to develop models that have unrealistic data requirements. Consequently a useful first step is to determine the kind of data that are available and let this, together with the known biology and the management questions, determine the kinds of models that are required. For stoats, the majority of extant data consist of trap catches, which pose the immediate problem that the populations to which they apply are subject to removal. Nevertheless, if the trapping effort is reasonably constant, then to a first approximation the trap catches can be considered as indices of density of a population subject to a constant proportional removal rate (i.e. additional mortality). The latter will not affect the density-dependence, so models based on trap catches can be used to test for this and establish its magnitude. Furthermore, the models can potentially be used as a basis for management if the removal rate (density-independent mortality) due to trapping can be corrected for. This should be possible by comparing the trapping-based model's estimate of intrinsic rate of increase with that deduced from known reproductive rates, recruitment rates, and minimum mortality rates.

With this in mind, available data on stoat trap catches over more than 1 year were collated and analysed, for both beech and non-beech habitats. All data were converted to a common index of stoats caught per 100 trap nights, in several cases corrected for sprung traps but in the majority of cases uncorrected (Appendix 2). The data were then analysed by regressing the exponential rate of increase ($r = \ln(N_{t+1}/N_t)$) on the first year's density index, $N_t$, for beech and non-beech habitats separately. The regressions were then compared and used to parameterise a Ricker model for changes in stoat density indices from year to year. In all cases the density indices represent maximum values per year (i.e. based on peak trap catches, generally in summer/early autumn), and therefore include the recruits for that year. The regressions are:

Beech: $r = \ln(N_{t+1}/N_t) = 0.828 - 0.320N_t$ SE 0.0875 $R^2 = 0.36$, df 1,21, $P = 0.001$

Non-beech: $r = \ln(N_{t+1}/N_t) = 0.229 - 0.793N_t$ SE 0.3380 $R^2 = 0.18$, df 1,19, $P = 0.03$

suggesting values for the intrinsic rates of increase of 0.83/year and 0.23/year in beech and non-beech habitats respectively, and $K$ values (carrying capacities, expressed in units of trap catch/100 trap nights) of 2.6 and 0.29 respectively (i.e. the respective values of $N_t$ that make $r = 0$, namely 0.828/0.321 and 0.229/0.793). The Ricker models from these equations are:

Beech: $N_{t+1} = 2.30\, N_t \exp(-0.320N_t)$

Non-beech: $N_{t+1} = 1.26\, N_t \exp(-0.793N_t)$

These relationships are illustrated by the dashed lines in Figs. 15 to 18. The proportions of variance accounted for are not high, especially in the non-beech habitats, but this is perhaps not surprising given the rather eclectic data. Much

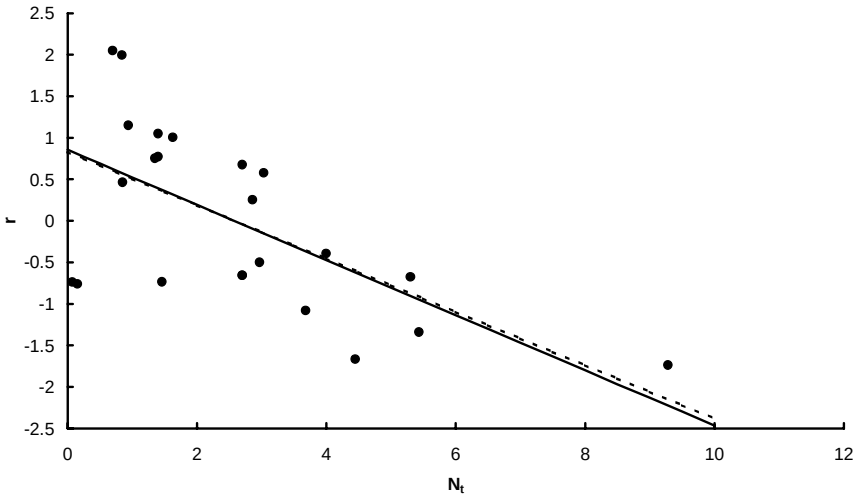


Figure 15. The relationship between stoat rate of increase/year ($r = \ln(N_{t+1}/N_t)$) and current year's density index ($N_t$, number caught/100 trap nights), for beech forest habitats. Solid line = combined regression for beech and non-beech habitats with common slope but different intercepts; dashed line = separate regression for beech habitats.

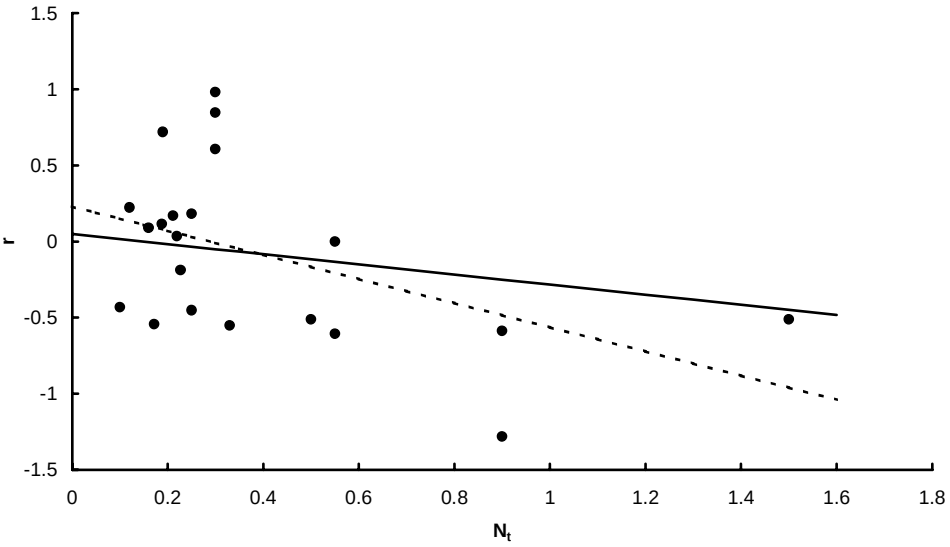


Figure 16. The relationship between stoat rate of increase/year ($r = \ln(N_{t+1}/N_t)$) and current year's density index ($N_t$, number caught/100 trap nights), for non-beech forest habitats. Solid line = combined regression for beech and non-beech habitats with common slope but different intercepts; dashed line = separate regression for non-beech habitats.

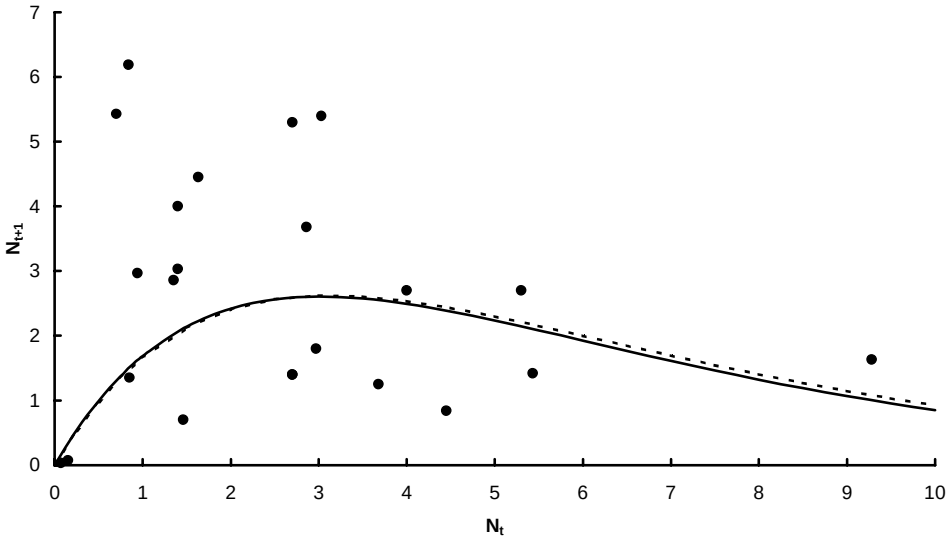


Figure 17. The relationship between stoat density index ($N$ = number caught/100 trap nights) in successive years ($t$ and $t$+1), for beech forest habitats. Solid line based on combined regression of $r$ (=$\ln(N_{t+1}/N_t)$) on $N_t$ for beech and non-beech habitats with common slope but different intercepts; dashed line = based on separate regression for beech habitats.

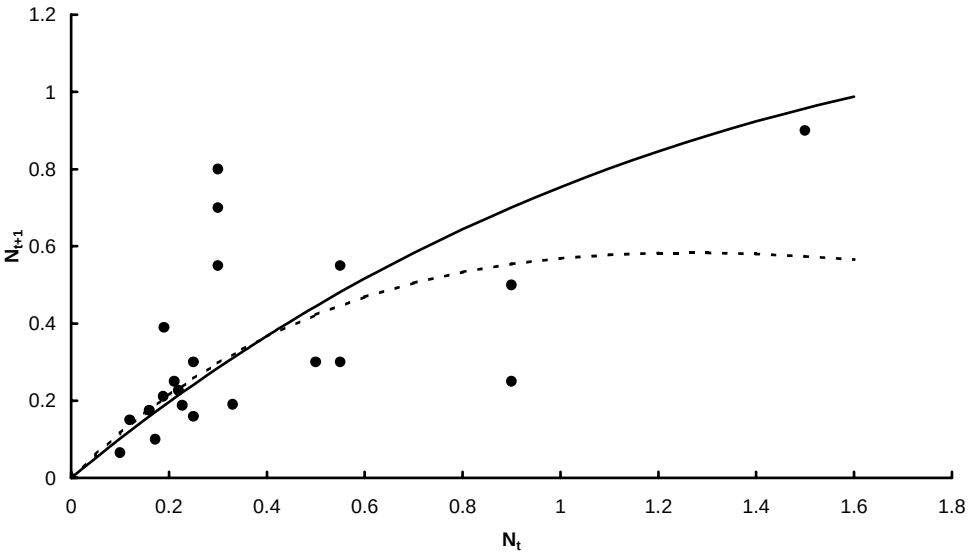


Figure 18. The relationship between stoat density index ($N$ = number caught/100 trap nights) in successive years ($t$ and $t$+1), for non-beech forest habitats. Solid line based on combined regression of $r$ (=ln($N_{t+1}/N_t$)) on $N_t$ for beech and non-beech habitats with common slope but different intercepts; dashed line = based on separate regression for non-beech habitats.

of the unexplained variance is undoubtedly due to variations in trapping intensity between sites, and the fact that some catches were corrected while others were not. However, there may also have been underlying causal variation between sites and years due to factors such as rat abundance, in the case of the non-beech habitats. In beech forests, a certain departure from the Ricker model would be expected, particularly at low densities, if the underlying pattern of population change is 3 years of low density followed by a fourth year of high density after a mouse outbreak (King et al., unpubl. data). The Ricker model can only mimic a 2-year pattern of density changes.

The slopes of the two regressions above are not significantly different, so the data for beech and non-beech habitats were combined and a dummy variable added that took the value of 0 for non-beech points and 1 for beech ones. Stepwise multiple regression was then applied, for $r$ (ln($N_{t+1}/N_t$)) against $N_t$ and the dummy variable, in order to test for differences in intercepts given a regression of common slope. The resulting regression was:

Combined beech/non-beech:

$$r = \ln(N_{t+1}/N_t) = 0.049 + 0.806(\text{dummy}) - 0.332N_t \quad R^2 = 0.31, \text{ df } 2,41, P < 0.001$$
$$\text{SE } 0.27 \qquad \text{SE } 0.072$$

The coefficient of the dummy variable was highly significant ($p$ = 0.005), indicating a significant difference in the intercept (= intrinsic rate of increase) between beech and non-beech habitats. The estimates are 0.855 (beech) and 0.049 (non-beech), the latter considerably lower than for the separate regression for the non-beech habitats. The resulting two Ricker models are:

Beech: $N_{t+1} = 2.35\ N_t \exp(-0.332N_t)$

Non-beech: $N_{t+1} = 1.05\ N_t \exp(-0.332N_t)$

The two regression relationships are shown by the solid lines in Figs. 15 and 16 and the two Ricker models by the solid lines in Figs. 17 and 18. Note that combining the data in this way suggests not only a very low $r_m$ value (intrinsic rate of increase) of 0.049, corresponding to a finite rate of increase of only 1.05/year in non-beech habitats, but less strong density-dependence than in the

model fitted separately to the non-beech data (0.332 compared with 0.793). *K* values from the combined regression are 2.6 for beech forests and 0.15 for non-beech.

## 4.5 RECOMMENDED APPROACHES FOR MODELLING STOAT DYNAMICS AND CONTROL

Three main modelling approaches appear appropriate for modelling stoat dynamics, control and impacts, and all would be worth pursuing. These are:

1. Single-species discrete generation density-dependence models (because they are simple, easiest to parameterise from sparse data, can form the basis for evaluation of controls, and can be developed immediately).
2. Single-species discrete generation density-dependence models with age-classes and perturbations (because they may give different results). These are the next simplest models, with density-dependence age-specific. They have the ability to mimic irruptions due to beech masting and mouse outbreaks, by assuming enhanced reproduction in one year (masting) followed by a year of suppressed reproduction and reduced first-year survival, at intervals that can be varied to suit local beech-masting conditions. Such models thus act as surrogates for explicit mouse- or rat-driven interactive ones, and are similar to and slightly simpler than the matrix model developed by Efford (M. Efford, pers. comm.).
3. Two-species fully interactive predator–prey models with age-classes and demography (separate stoat birth and death rates, intraspecific stoat density-dependence, dependence of stoat rates of increase on prey density, prey birth rate, death rate and density-dependence, and predation rate from stoats via an appropriate functional response). Again, these models may give different results in terms of predictions for stoat control (e.g. because they allow for density-dependence to be both direct and through competition for resources), and in that sense act as a test of the simpler models, but more importantly they are necessary to predict stoat impacts on prey, hence to establish stoat density targets for control.

In addition, all models should include net immigration if control policies are likely to be restricted to parts of contiguous stoat habitat. To obtain estimates for this requires a spatial model of dispersal, readily achieved using a simple grid structure that will allow net immigration rates to be estimated for controlled areas of different sizes. This enables an equation to be developed relating the immigration rate to the area and density differential. This area- and density-based immigration rate can then be added to the closed population models developed above. Such an approach has been successfully applied to possums and appears to be the simplest way of handling the problem of recolonisation (Barlow 1993).

It would also be useful to evaluate the adequacy of the discrete-generation Ricker models for predicting control, particularly in terms of within-year timing, against slightly more complex and realistic models in which mortality is continuous throughout the year but births are pulsed.

# 5. Recommendations for future research

We have divided our recommendations for research into those which would best support development of the predictive modelling frameworks we advocate, and those which are modelling tasks in themselves. This second series of recommendations has been further divided into tasks which could be conducted immediately, and those which will require new data.

Modelling should precede and accompany, rather than succeed, the field work if maximum benefit is to be gained from it. This is for two reasons. Firstly, one of the main benefits is to guide data collection and this is a continuing process; it cannot happen, and there is a risk of waste, if the data are gathered first. Secondly, some modelling can be done with virtually no data (e.g. evaluating characteristics of pathogens that would give successful biological control), and a considerable amount can be done with only few data (e.g. applying general models for control options specifically to stoats, based on simple population parameters already partially established from this preliminary study).

The following specific modelling tasks are of high priority and can be undertaken immediately, without the need for additional data:

- Assess target rates of immunocontraception necessary to achieve different reductions in density.
- Assess whether non-disseminating fertility control could compare favourably with trapping.
- Assess the kinds of pathogens that would be most effective as biological control agents (in particular whether canine distemper could work and what additional data might be needed to assess this).
- Compare results of models of different levels of complexity and realism in terms of predictions of control effectiveness.
- Link models for stoat control to existing models of prey responses to control.

The following modelling tasks are of high priority and require more data:

- Use a simple spatial grid model to help translate data on stoat dispersal and recolonisation into a relationship between immigration rate, density differential, and area controlled, and add this to the non-spatial models.
- Improve simple non-interactive models for control effectiveness to develop more quantitative recommendations, based on more-detailed stoat and prey population dynamics data.
- Develop interactive models of stoat control for podocarp–broadleaf and mixed forests, with links to the demography of kiwi and other key prey species, in order to develop control recommendations based on analysis of viability of the prey under different control scenarios.

The following data needs are of high priority in relation to modelling requirements:

- Establishment of the density-dependent relationship between juvenile stoat survival and population density.

- Long-term (4-year) monitoring of non-trapped stoat densities (if reliable indices can be found or developed that can be related to actual density) in several sites, ideally of markedly different density and including non-beech habitats.
- Improved estimates of stoat dispersal and recolonisation rates in relation to density and area (e.g. by monitoring reinvasion into controlled areas of different sizes).
- Validation/testing of the association between rat and stoat density in the absence of rodent poisoning in podocarp–broadleaf forest.
- Identification of factors driving variation in rat and stoat density in podocarp–broadleaf forest.
- Experimental assessment of the effect of stoat control on rodent density and the density of potentially competing predators (cats, weasels, ferrets).
- More robust estimates of the efficacy, consistency, and cost of existing stoat control strategies.
- Additional population data for key species at risk, involving profiles of densities over time covering at least 4 continuous years and more than one site, which allow density-dependence to be identified and quantified, together with predation rates by stoats.
- Estimates of the effect of changes in stoat density on key demographic rates of prey species at risk in podocarp–broadleaf or mixed forest and selected other habitats (e.g. kiwi, kaka, robins, kereru).

# 6. Acknowledgements

This work was funded by the Department of Conservation. We are grateful to Christine Bezar and Wendy Weller of Landcare Research for editorial assistance, and to John McLennan for helpful discussions.

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# Appendix 1

## CAUGHLEY'S THREE-LEVEL INTERACTIVE MODEL

The model consists of three simultaneous differential equations, describing variation in the instantaneous rates of change (dV/dt, dH/dt, dP/dt) in the abundance of vegetation (*V*), herbivores (*H*), and predators (*P*) respectively:

$$\frac{dV}{dt} = r_{mV}V\left(1-\frac{V}{K_V}\right)-c_V H\left(1-e^{-d_V V}\right)$$

$$\frac{dH}{dt} = -a_H H + c_H H\left(1-e^{-d_H V}\right)- fP\left(1-e^{-d_P H}\right)$$

$$\frac{dP}{dt} = -a_P P + c_P P\left(1-e^{-d_{PN} H}\right)$$

Parameters are:

$r_{mV}$ Maximum rate of increase in vegetation biomass

$K_V$ Maximum vegetation biomass

$c_V$ Maximum rate of vegetation offtake by herbivores

$d_V$ Herbivore foraging efficiency

$a_H$ Rate of decline in herbivore abundance in the absence of vegetation

$c_H$ Rate at which the decline in herbivore abundance is ameliorated by increasing vegetation

$d_H$ Herbivore demographic efficiency

$f$ Maximum rate of herbivore offtake by predators

$d_P$ Predator foraging efficiency

$a_P$ Rate of decline in predator abundance in the absence of herbivores

$c_P$ Rate at which the decline in predator abundance is ameliorated by increasing herbivores

$d_{PN}$ Predator demographic efficiency

This formulation includes no social regulation of predators through interference or territoriality. One approach to modelling the combined effect of prey availability and prevailing predator density on rates of change in predator density would be to incorporate a function (*g*) that modifies the numerical response of the predator population to herbivore availability according to prevailing predator density:

$$\frac{dP}{dt} = -a_P P + c_P P\left(1-e^{-d_{PN} H}\right)+ g(P)P$$

While interferential models that purport to describe the dynamics of herbivore populations where individuals interfere with each other's capacity to graze on vegetation have been described (e.g. Caughley & Lawton 1981; Barlow 1985), the explicit form of *g* has not been determined for any predator population.

# Appendix 2

## AVAILABLE DATA ON STOAT TRAP CATCHES OVER MORE THAN 1 YEAR

PEAK STOAT NUMBERS ($N$ = NO. CAUGHT/100 CORRECTED TRAP NIGHTS, * INDICATES NUMBERS NOT CORRECTED) IN SUCCESSIVE YEARS ($t$ AND $t$+1). SITES ARE: 1 NORTHERN UREWERA (OTAMATUNA, DOC 1999a); 2 BOUNDARY STREAM (DOC 1999b); 3 MAPARA* (MURPHY ET AL. 1998); 4 MT MISERY* (WILSON ET AL. 1998); 5 TROUNSON* (DOC 1999c); 6 PUREORA* (EXOTIC FOREST, KING ET AL. 1996); 7 PUREORA* (UNLOGGED PODOCARP, KING ET AL. 1996); 8 PUREORA* (LOGGED FOREST ROAD EDGE, KING ET AL. 1996); 9 EGLINTON* (KING 1983); 10 HOLLYFORD* (KING 1983); 11 CRAIGIEBURN* (KING 1983); 12 MT COOK* (KING 1983); 13 EGLINTON* (DEER FLAT, MURPHY & DOWDING 1995); 14 MAPARA* (MURPHY & BRADFIELD 1992).

| NON-BEECH | | | | BEECH | | | |
|---|---|---|---|---|---|---|---|
| SITE | YEAR | $N_t$ | $N_{t+1}$ | SITE | YEAR | $N_t$ | $N_{t+1}$ |
| 1 | 97 | 0.330 | 0.190 | 4 | 75 | 2.700 | 1.400 |
| 1 | 98 | 0.190 | 0.390 | 4 | 76 | 1.400 | 4.000 |
| 2 | 97 | 0.160 | 0.175 | 4 | 77 | 4.000 | 2.700 |
| 3 | 90 | 0.219 | 0.227 | 4 | 78 | 2.700 | 5.300 |
| 3 | 91 | 0.227 | 0.188 | 4 | 79 | 5.300 | 2.700 |
| 3 | 92 | 0.188 | 0.211 | 4 | 80 | 2.700 | 1.400 |
| 3 | 93 | 0.211 | 0.250 | 4 | 81 | 1.400 | 3.030 |
| 3 | 94 | 0.250 | 0.159 | 4 | 82 | 3.030 | 5.400 |
| 5 | 97 | 0.172 | 0.100 | 9 | 75 | 1.460 | 0.700 |
| 5 | 98 | 0.100 | 0.065 | 9 | 76 | 0.700 | 5.430 |
| 6 | 83 | 1.500 | 0.900 | 9 | 77 | 5.430 | 1.420 |
| 6 | 84 | 0.900 | 0.500 | 10 | 75 | 9.280 | 1.630 |
| 6 | 85 | 0.500 | 0.300 | 10 | 76 | 1.630 | 4.450 |
| 6 | 86 | 0.300 | 0.800 | 10 | 77 | 4.450 | 0.840 |
| 7 | 83 | 0.550 | 0.300 | 10 | 78 | 0.840 | 6.190 |
| 7 | 84 | 0.300 | 0.700 | 11 | 74 | 0.850 | 1.350 |
| 7 | 85 | 0.700 | 0.000 | 11 | 75 | 1.350 | 2.860 |
| 7 | 86 | 0.000 | 0.350 | 11 | 76 | 2.860 | 3.680 |
| 8 | 83 | 0.900 | 0.250 | 11 | 77 | 3.680 | 1.250 |
| 8 | 84 | 0.250 | 0.300 | 12 | 75 | 0.940 | 2.970 |
| 8 | 85 | 0.300 | 0.550 | 12 | 76 | 2.970 | 1.800 |
| 8 | 86 | 0.550 | 0.550 | 13 | 90 | 0.152 | 0.071 |
| 14 | 89 | 0.120 | 0.150 | 13 | 91 | 0.071 | 0.034 |