

Modelling the dynamics and control of stoats in New Zealand forests

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N.D. Barlow and M.C. Barron

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ABSTRACT

The impact of sterilisation and culling control of stoat (*Mustela erminea*) populations was evaluated using models of increasing complexity. The first was a simple logistic model with continuous births and deaths; the second included a more realistic birth pulse rather than continuous births; and the third included a birth pulse and age structure. For beech forest (*Nothofagus* spp.) habitats, the birth pulse models distinguished between mast, crash and normal years, each year having a different intrinsic rate of increase, r_m , which was parameterised from trap-catch indices. The second model best predicted the large variation in stoat abundance observed in beech forest. Using this model, little difference was predicted in the proportional reduction of stoat density under culling or sterilisation control. Under continuous control, sterilisation was slightly more effective at reducing peak (summer) stoat density; however under pulsed control, culling was marginally more effective than sterilisation. Control of either kind was much more effective against populations in non-beech forests than against those in beech forests, essentially because of the former population's lower r_m value. The second birth pulse model was also used to predict the likely dynamics of canine distemper virus (CDV) in stoat populations. CDV was not predicted to persist as an endemic disease in New Zealand stoat populations. This was primarily due to the birth pulse structure which precluded the continuous recruitment of susceptible individuals required to maintain the disease within a host population.

Keywords: stoat, *Mustela erminea*, population modelling, sterilisation, culling, biological control, canine distemper virus, beech mast, pest control, New Zealand

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1. Introduction

Stoats (*Mustela erminea* L.) were introduced to New Zealand in the late 1800s to control rabbits. They spread rapidly to forests and now represent both the most widespread mustelid species and the most significant predator of a number of New Zealand's most threatened bird species. Stoat control will have to be ongoing if some endemic species like kaka (*Nestor meridionalis*) and kiwi (*Apteryx* spp.) are to survive on the mainland (McLennan et al. 1996; Wilson et al. 1998). To date, control has relied on labour-intensive trapping, but a 5-year research programme was instigated in 2000 by the Department of Conservation (DOC) to improve both short-term and long-term control, in the first case by developing more effective baits, lures and traps, and in the second by exploring higher-risk options such as fertility control and biological control (Murphy & Fechney 2003).

It is timely to question the likely efficacy of different control options in ecological terms, should the technology be capable of delivering them. Population models have been developed to help answer these questions. Three models are used, of progressive complexity and realism. The first and simplest is a single-species logistic model, in which births and deaths are assumed to be continuous. This allows simple but approximate analytical solutions for the long-term impacts of controls like sterilisation and culling. The second model is a more realistic birth pulse model, in which all rates, including controls like culling or sterilisation, are continuous except births, which occur as discrete events once a year. This more realistic model provides estimates for the transient impacts of culling and sterilisation over time following the start of control, as well as more refined estimates of the long-term effects. In particular, for beech forest habitats (*Nothofagus* spp.) it distinguishes mast years and the years immediately following mast years ('crash years') from other 'normal' years, each having different stoat parameters. Mast events occur every 3–5 years (Wardle 1984), and the associated irruptions of birds, mice and invertebrates provide a sudden increase in food resulting in a density-independent change in stoat vital rates (King 1983; King et al. 2003). The third model is a birth pulse one with age structure, the main aim of which is to validate the simpler, non-age-structured model. In all cases the models are habitat-specific, involving populations in both podocarp/broadleaf and beech forests. The primary data source is trapping records over periods of up to 5 years in a variety of habitats. Stoat populations were simulated using the above three models to answer the question: is sterilisation likely to be a superior method to culling for control for stoat populations?

Canine distemper virus (CDV) has been identified as a possible biological control agent for stoats (O'Keefe 1995; Norbury 2000) and a vaccine strain of CDV is currently being prepared to investigate the lethality and transmissibility of these strains in stoats and ferrets (Murphy & Fechney 2003). Mustelidae are known to be particularly susceptible to CDV (Appel 1987) but there has only been one case of CDV-associated death reported for stoats in the literature (Kymer & Epps 1969), so the understanding of CDV epidemiology in stoats is negligible. Epidemics in other carnivore populations are characterised by rapid disease dynamics and high mortality rates of affected individuals (Appel &

Summers 1995). An outbreak of CDV in black-footed ferrets (*Mustela nigripes*) eliminated the only known wild population of this species (Williams et al. 1988). Large reductions in local populations caused by CDV epidemics have also been recorded for lions (*Panthera leo*; Roelke-Parker et al. 1996), Caspian seals (*Phoca caspica*; Kennedy et al. 2000), African wild dogs (*Lycaon pictus*; MacDonald et al. 1992), and raccoon dogs (*Nyctereutes procyonoides*; Machida et al. 1993). We used a birth pulse model with masting events (Model 2) as the basis for a disease/host model of the Anderson/May type (Anderson & May 1981) to predict the likely dynamics of canine distemper virus (CDV) in stoat populations in New Zealand beech forest. Whilst this exercise was purely exploratory, there being no data on the pathology or epidemiology of CDV in stoats, it is anticipated that the models will be improved when parameter estimates become available and that they will also be used as the basis to evaluate other pathogens as potential biocontrol agents.

2. Models to compare stoat control by culling versus steritisation

2.1 POPULATION MODELS

2.1.1 Model 1: logistic model

The model for culling is:

$$\frac{dN}{dt} = r_m N \left(1 - \frac{N}{K} \right) - cN \quad \text{Eqn 1}$$

where dN/dt = rate of growth of the population, r_m = intrinsic rate of increase, K = equilibrium density or carrying capacity, c = instantaneous culling rate and N = the current population density. There is no evidence for density-dependent processes controlling fertility in stoats (McDonald & Larivière 2001). However, Powell & King (1997) did find a significant negative relationship between first-year survivorship and stoat density in New Zealand beech forest suggesting density dependence in mortality rates. Assuming it is the mortality rather than the birth rate that is density-dependent, for sterilisation the model becomes (Barlow et al. 1997):

$$\frac{dN}{dt} = N \left(b(1-Q) - d - \frac{r_m N}{K} \right) \quad \text{Eqn 2a}$$

$$\frac{dQ}{dt} = (1-Q)(s - bQ) \quad \text{Eqn 2b}$$

where b = instantaneous birth rate, Q = proportion of sterile females, d = density-independent instantaneous death rate, s = instantaneous sterilisation rate and dQ/dt = rate of change of the proportion of sterile females. Note that $r_m = b - d$. The derivation of equation 2b is given in Barlow et al. (1997), it represents a situation in which mating is polygamous and either females alone

or both sexes are sterilised, or a monogamous or harem mating system where either sex alone is sterilised. In all of these cases, the proportion of females not reproducing corresponds exactly or approximately to the probability of sterilisation, whichever sex this applies to. Here, we have assumed that it is the female stoats that are sterilised, but like all the other instantaneous rates, the sterilisation rate is expressed as a per capita rate. Other assumptions of the model are that sterilisation is lifelong, that sterilised females have the same mortality rates as fertile females and that all young are born fertile.

By setting dN/dt and dQ/dt to zero in equations 1 and 2 and solving for N^*/K (where N^*/K are equilibrium densities with, and without control, respectively), the final, equilibrium reductions in population size can be predicted for various intensities of control, as indicated in Table 1.

TABLE 1. RELATIONSHIPS BETWEEN PROPORTIONAL REDUCTION IN POPULATION DENSITY (p), ASSUMING POLYGAMOUS MATING.

Where $p = 1 - N^*/K$ and N^* and K are equilibrium densities with and without control respectively, c is the instantaneous rate of culling, s is the instantaneous rate of sterilisation, and Q is the proportion of females sterilised.

VARIABLE	ESTIMATE
Proportional reduction (p) given c	c/r_m
Proportional reduction (p) given s	s/r_m
Proportion sterilised (Q) given s	s/b
Proportional reduction (p) given Q	bQ/r_m
Q for eradication ($p = 1$)	r_m/b
Sterilisation rate (s) to give proportional reduction p	$r_m p$
Level (Q) to give proportional reduction p	$r_m p/b$

Stoat population parameters are: b = instantaneous birth rate, r_m = intrinsic rate of increase. Modified from Barlow et al. (1997).

2.1.2 Model 2: birth pulse model

This model included death rate, sterilisation and culling in continuous time, and births of fertile females as a discrete yearly event, thus:

$$\frac{dN}{dt} = N \left(-d - \frac{r_m N}{K} - c \right) \quad \text{Eqn 3a}$$

$$\frac{dQ}{dt} = s(1 - Q) \quad \text{Eqn 3b}$$

and at the birth pulse

$$N' = N(1 + (1 - Q)P) \quad \text{Eqn 3c}$$

$$Q' = \frac{Q}{1 + (1 - Q)P} \quad \text{Eqn 3d}$$

where N and Q are the values before the pulse, N' and Q' are the new values after the pulse, and P is the productivity or the number of female offspring produced per female per birth pulse. Results for culling and sterilising at different rates were obtained by simulation, comparing equilibrium densities without control

with equilibrium densities in the presence of control and with the rate at which populations declined following the start of control. For beech forests the model was run with a repeated sequence of 4 years corresponding to a mast year, a crash year and two normal years, using the parameters appropriate to each year type. Simulation used a weekly (1/52 year) time step and Euler integration. In the birth pulse models, 'births' take place at the time young stoats become independent from their mother. Hence reference to the 'birth pulse' is actually describing the large pulse in juvenile recruitment to the stoat population in December/January when young stoats disperse from their natal den.

2.1.3 Model 3: age-structured model

This model operated in the same way as Model 2, but juveniles aged 0-1 years were kept separate after the birth pulse and merged with adults at the next birth pulse. Adult survival was assumed to be density-independent but juvenile survival to be density-dependent based on data from the literature. In this respect the model differed from the previous two, in that virtually all the parameters were estimated from the literature rather than from the regressions of r against N_t .

2.2 PARAMETERS FOR POPULATION MODELS

The available data on stoat trap catches over more than one 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 (see Appendix 1). The data were then analysed by regressing the exponential rate of increase ($r = \ln(N_{t+1}/N_t)$)¹ on the first year's density index, N_t , for beech and non-beech habitats separately, then the data for beech were divided into mast years, 'crash years' immediately following mast years, and other years and analysed by stepwise multiple regression with dummy intercepts and slopes for mast years and crash years. For example, the dummy intercept for mast years took the value 0 for all other years and 1 for data corresponding to mast years. The variable would not be selected if there was no significant difference in intercepts, but if there were, the coefficient of the dummy represents an addition to the intercept for that set of data, namely mast years. Dummy slope variables were the dummy intercept (0 or 1) multiplied by N_t ; if significant, they represented an addition to the slope for the appropriate data set. In all cases the density indices represented maximum values per year (i.e. based on peak trap catches, generally in summer/early autumn) and, therefore, included the new recruits for that year.

For the birth pulse model, the intrinsic rate of increase, r_m , was partitioned into birth and death rates using additional data from the literature, and for the age-structured model, death rate was further divided into adult death rate and density-dependent juvenile death rate, again using values from the literature (Powell & King 1997).

¹ \ln = natural log, \log_e

The regressions were:

$$\begin{aligned} \text{Beech: } r = \ln(N_{t+1}/N_t) &= 0.60 - 0.27N_t + 0.79 D_1 - 0.99D_2 \\ P &= 0.001 \quad P = 0.037 \quad P = 0.013 \\ R^2 &= 0.70; F = 19.1; df = 3,20; P = 0.001 \end{aligned}$$

where D_1 is the dummy intercept for mast years and D_2 that for crash years. This regression (Fig. 1) gave a slightly higher R^2 value than one with the dummy intercept for mast years but a dummy slope for crash years. The overall relationship for beech forests, irrespective of year, is:

$$\begin{aligned} \text{Beech overall: } r &= 0.87 - 0.38 N_t \\ R^2 &= 0.44; F = 19.3; df = 1,22; P < 0.001 \end{aligned}$$

$$\begin{aligned} \text{Non-beech: } r &= 0.23 - 0.79N_t \\ R^2 &= 0.18; F = 5.5; df = 1,19; P = 0.03 \end{aligned}$$

This relationship is shown in Fig. 2. All the above relationships are summarised in Table 2, and yield the basic population parameters r_m and K in Table 3. The intrinsic rate of increase, r_m , is the maximum value of r (i.e. the regression intercept, corresponding to $N_t = 0$) and the carrying capacity or equilibrium density, K , is the value of N_t which makes $r = 0$. This is the intercept divided by the slope.

Figure 1. 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 = normal year ($r = 0.60 - 0.27N_t$), dashed line = mast year ($r = 1.39 - 0.27N_t$), dotted line = 'crash year' following a mast year ($r = -0.39 - 0.27N_t$). See Section 2.2 for regression statistics.

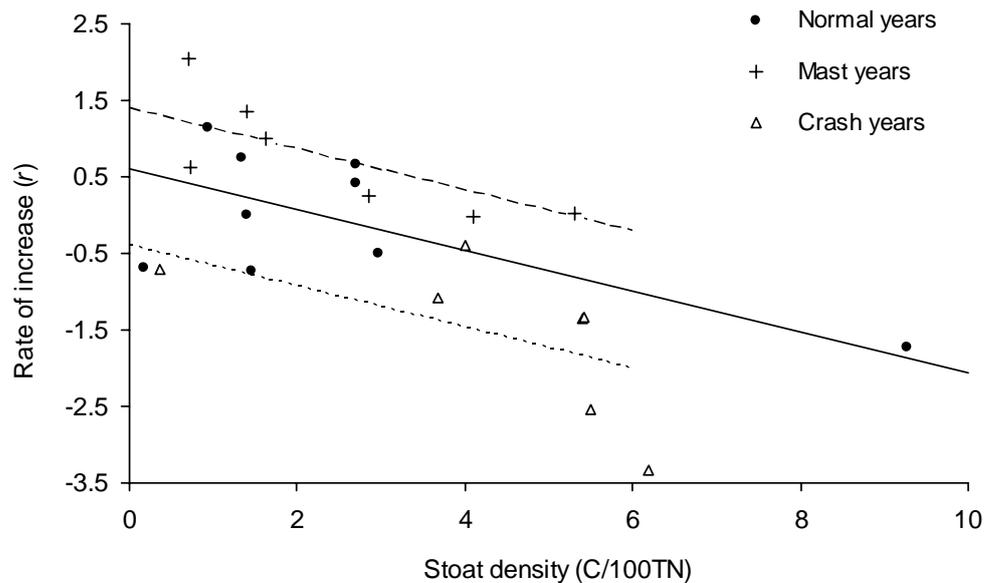
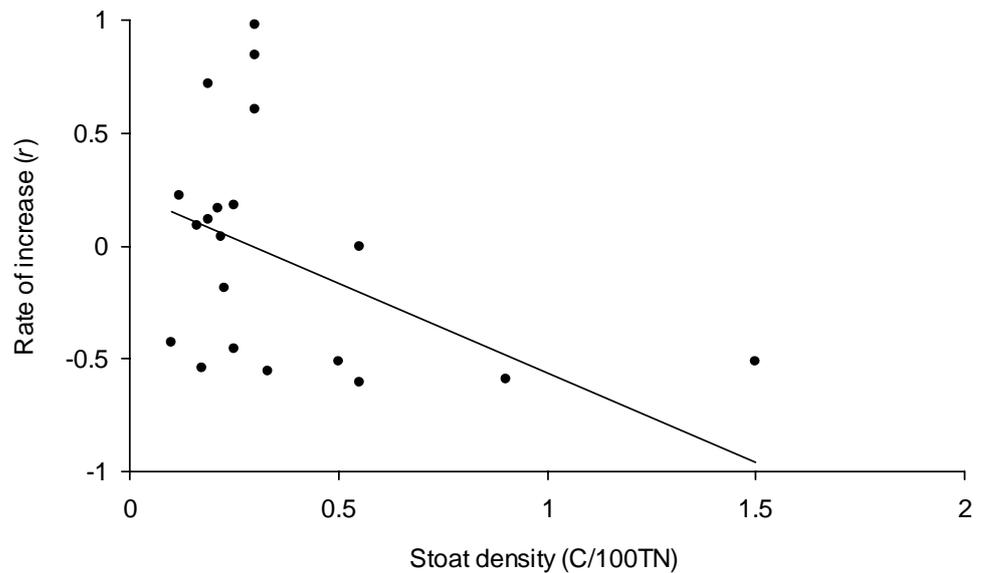


TABLE 2. RELATIONSHIP BETWEEN THE INTRINSIC RATE OF INCREASE (r) AND STOAT DENSITY (N_t), AS DETERMINED BY LINEAR REGRESSION* AND USING CATCHES PER 100 TRAP-NIGHTS FROM APPENDIX 1 AS DENSITY INDICES.

FOREST/YEAR TYPE	DENSITY RELATIONSHIP
Beech, overall	$r = 0.87 - 0.38 N_t$
Beech, mast	$r = 1.39 - 0.27 N_t$
Beech, normal	$r = 0.60 - 0.27 N_t$
Beech, crash	$r = -0.39 - 0.27 N_t$
Non-beech	$r = 0.23 - 0.79 N_t$

* Shown in Figs 1 and 2.

Figure 2. 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 ($r = 0.23 - 0.79N_t$; see Section 2.2 for regression statistics).



The linear regression method used to estimate the intrinsic rate of increase and the strength of density dependence acting on the rate of increase is somewhat simplistic and statistically dubious (McCallum 2000). However, given the lack of data on fecundity and mortality rates in relation to population density, it was the only method available. Process error due to the effects of habitat and mast seeding on the population growth rate was minimised by including these variables in the regressions, but it can be seen from Fig. 2 and from the low R^2 statistic ($= 0.18$) for the depicted relationship, that there is still a lot of unexplained variation in population growth rates in non-beech habitats. The intrinsic rate of increase estimated for non-beech habitats ($r_m = 0.23$; 95% CI = $(-0.13, 0.59)$) is thus not very robust, and perhaps the only meaningful statement we can make is that the population rates of increase are generally lower than those in beech habitats. In contrast, much of the variation in the rate of increase in beech habitats was explained by population density ($R^2 = 0.70$) if dummy variables to account for the differing rates of increase in mast and crash years were included. The data point corresponding to $N_t = 9.3$, $r = -1.7$ in Fig. 1 was identified as having high leverage. However, repeating the regression analysis without this data point resulted in very similar regression constant and

TABLE 3. POPULATION DATA FOR STOATS.

FOREST/YEAR TYPE	r_m	b	d	K	PRODUCTIVITY	ANNUAL SURVIVAL	DENSITY COEFFICIENT
Beech, mast	1.39	1.72	0.33	5.15	4.60	0.72	-0.27
Beech normal	0.60	1.35	0.75	2.22	2.86	0.47	-0.27
Beech crash	-0.39	0.36	0.75	-1.44	0.43	0.47	-0.27
Beech overall	0.87	1.48	0.61	2.29	3.40	0.54	-0.38
Non-beech	0.23	0.98	0.75	0.29	1.67	0.47	-0.79

r_m = intrinsic rate of increase, b = instantaneous birth rate, d = minimum (density-independent) instantaneous death rate, K = equilibrium density index (C/100TN), productivity = females/female/year, annual survival = finite survival rate/year, density coefficient = the constant estimated for N_t in the regression of r against N_t .

coefficient estimates to the original analysis ($r = 0.62 - 0.29N_t + 0.81D_1 - 0.93D_2$; $R^2 = 0.68$, $df = 3,19$; $P < 0.001$), so we are reasonably confident in the estimates of r_m based on the available data.

In Table 3, r_m was partitioned into birth and death rates as follows. Since fecundity is determined in the year prior to giving birth, the mean number of corpora lutea per female in a non-mast year was taken as the physiological maximum productivity in a mast year and was 4.6 females/female/year (Powell & King 1997). This productivity translates to an instantaneous birth rate of $\ln(1 + \text{productivity}) = 1.72/\text{yr}$. The density-independent death rate is calculated by subtraction of r_m from the birth rate, which is $1.72 - 1.39 = 0.33/\text{yr}$. For normal years the death rate is estimated from the life table of Powell & King (1997: table 8), which suggests an average survival rate of 0.47/yr over the first 3 years of life, hence an instantaneous death rate of $-\ln(0.47) = 0.75/\text{yr}$. The birth rate is r_m plus the death rate, or $0.6 + 0.75 = 1.35/\text{yr}$. Crash years are characterised mainly by a reduction in birth rate, so death rate was assumed to remain the same at 0.75 and the birth rate to be r_m plus the death rate or $-0.39 + 0.75 = 0.36/\text{yr}$. For the overall beech forest relationship, the death rate was assumed to be the weighted average of those in a mast year, crash year and two normal years ($= 0.61/\text{yr}$), and the birth rate to be r_m plus the death rate ($= 1.48/\text{yr}$).

In non-beech forests the death rate was assumed to be the same as in normal years in beech forests (0.75/yr), giving a birth rate of $0.23 + 0.75 = 0.98/\text{yr}$. The productivities and yearly survival rates in Table 3 are calculated from the instantaneous rates (productivity = $\exp[\text{birth rate}] - 1$, and survival = $\exp[-\text{death rate}]$). K values in the birth pulse model (Model 2) were adjusted downwards ($\times 0.6$ in beech forests, $\times 0.69$ in non-beech forests) to reflect the fact that they were based on peak numbers but applied in the model over the whole year, covering the peak and subsequent decline; the scaling factors were chosen to give simulated peaks equal to observed K values.

Unlike Models 1 and 2, the age-structured model used independent data from the literature rather than the regressions of r on N_t ; this includes the density-dependence. Adult survival rate was assumed to be 0.55/yr (Powell & King 1997: table 8) and juvenile survival to be density-dependent based on Powell & King (1997: fig. 8). Appendix 2 reproduces the data from Powell & King (1997), and regression of log-transformed survival rates against stoat density gave the linear relationship:

$$\ln(\text{survival}) = -0.82 - 0.65 N_t$$

$$R^2 = 0.71; F = 20.4; df = 1,7; P = 0.003$$

which translated to:

$$\text{1st year survival} = 0.44 \exp(-0.65 N_t)$$

where N_t is the density of stoats during the birth summer of the juveniles. This was implemented in the model by assuming that juvenile survival followed this relationship in summer but was 100% during the rest of the year. The relevant value of N_t was taken to be the mean density to date during summer.

The above data relate to the models as follows. The simple logistic model used the regression relationships for beech forest (overall) and non-beech forest, as in Table 2, together with the birth and death rates for the overall beech forest and non-beech forest habitats in Table 3. For the birth pulse model, the beech

forest data were those for specific years (Table 3), which were applied in a repeated sequence of mast, crash and two normal years. The age-structured model used these same parameters plus the separate values for adult survival and juvenile density-dependent survival, and was applied only to beech forest.

2.3 RESULTS FROM POPULATION MODELS

2.3.1 Model 1: effects of culling and sterilisation

The general results for effects of culling and sterilisation, given Model 1, the simple logistic, are shown in Table 1. Applied to stoats, the key results are:

Proportional reduction in density (p); $p = c/r_m = 1.15c$ (beech) and $4.35c$ (non-beech)

Proportional reduction in density (p); $p = s/r_m = 1.15s$ (beech) and $4.35s$ (non-beech)

Proportion of females sterilised (Q); $Q = s/b = 0.68s$ (beech) and $1.02s$ (non-beech)

where c and s are the instantaneous sterilisation or culling rates. For practical purposes, and given that most control methods are applied on a continuous basis, it is more useful to consider control in terms of percent of the population culled or sterilised per month. This monthly rate (e.g. the proportion culled per month) is given by:

$$\text{Proportion culled per month} = 1 - \exp(-c/12)$$

and the same applies to sterilisation rates. Expressing control in these terms, the relationships are approximately linear and are summarised as follows:

$$p = 14 \times \text{proportion culled or sterilised per month (beech forest)}$$

$$p = 52 \times \text{proportion culled or sterilised per month (non-beech forest)}$$

$$Q = 8 \times \text{proportion sterilised per month (beech forest)}$$

$$Q = 12 \times \text{proportion sterilised per month (non-beech forest)}$$

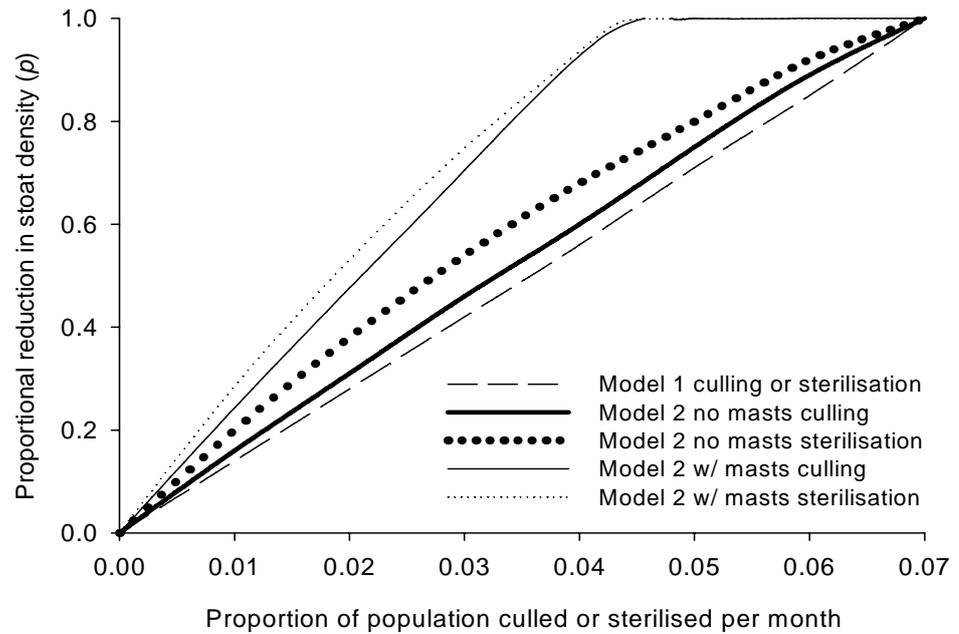
$$p = 1.7 \times Q \text{ (beech forest)}$$

$$p = 4.3 \times Q \text{ (non-beech forest)}$$

As these results show, the long-term reduction in equilibrium population density gained from sterilisation is the same as that from culling, if control is applied at the same rate (e.g. the same level of effective baiting or the same proportion of the population sterilised per month by baiting as is killed by trapping). Given the uncertainty in the estimate of r_m for non-beech habitats it is best not to place too much confidence in the quantitative predictions of the model for this habitat type. However if we accept that r_m is probably lower in non-beech compared with beech habitats, then control of either kind is much more effective against populations in non-beech forests than against those in beech forests, essentially because of this lower capacity for increase. In terms of sterilisation, the difference is because a given proportion of sterile females translates to a greater reduction in population density; the effort required to generate this proportion of sterile females, in terms of sterilisation rate per month, is slightly higher in beech habitats.

From the above relationships, culling or sterilising at a rate of 0.07/month in beech forests or 0.02/month in non-beech forests would, theoretically,

Figure 3. The effect of continuous culling or sterilising at different monthly rates on the proportional reduction (p) in stoat density in beech forest using different models.



eradicate a closed population of stoats. Whether or not eradication is possible in practice, the models suggest quite modest rates of removal or sterilisation have significant impacts on closed populations.

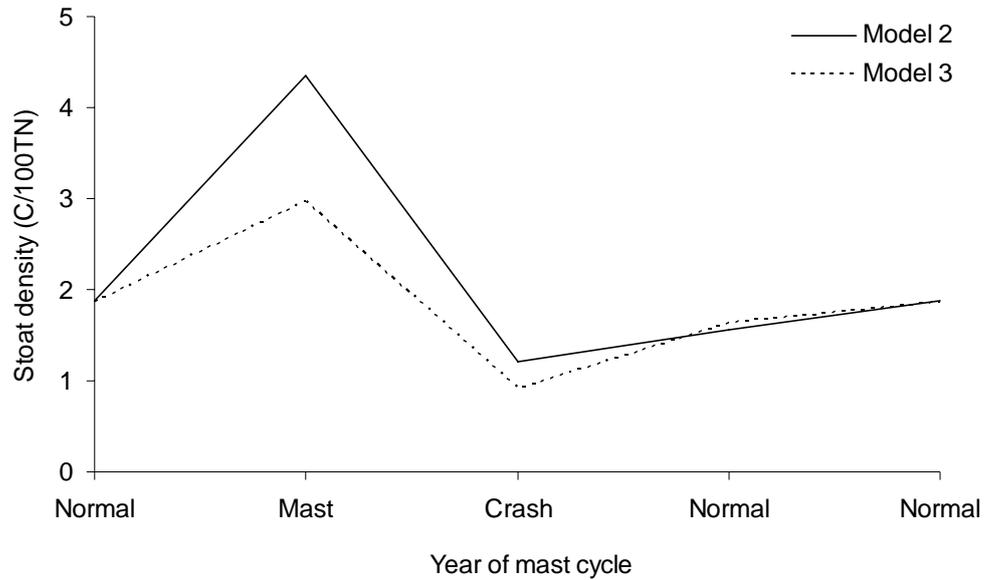
2.3.2 Model 1 v. Model 2: effects of birth pulse

Including a birth pulse (Model 2), rather than continuous births (Model 1), resulted in sterilisation being slightly more effective than culling at reducing peak (summer) stoat densities in (Fig. 3). This was because the effects of sterilisation are realised in summer, at the birth pulse, whereas the effects of culling occur continuously throughout the year. Therefore, for the same control rate, sterilisation is more effective than culling at reducing densities in summer, but as the year progresses, the additional constant mortality imposed by culling means that culling reduces winter and spring densities more, relative to sterilisation. Including a birth pulse introduced non-linearity in the relationship between the proportional reduction in host density and the control rate (Fig. 3). This was because there is a linear relationship between r_m , the intrinsic rate of increase (and hence the predicted population reduction) and the instantaneous birth rate b when births are continuous (Model 1). However, in the birth pulse model (Model 2) annual productivity was estimated as $\exp(b) - 1$, which means that r_m varied as $\ln(\text{productivity})$ (Barlow et al. 1997).

2.3.3 Model 2: effects of beech mast cycle

The trends in stoat numbers during a beech mast cycle predicted by Model 2, in the absence of control, are shown in Fig. 4. The model appears to mimic the large variation in stoat densities observed in the field reasonably well, including the approximately four-fold difference in stoat densities between mast and crash years. The effect of including a beech mast cycle in Model 2 was to decrease the average predicted population density for a given level of control

Figure 4. Predicted peak (summer) stoat density for Model 2 (birth pulse model) and Model 3 (age-structured model) for beech forests, over a 4-year mast cycle and in the absence of control measures.



(Fig. 3). This was because the intrinsic rates of increase, r_m , over the mast cycle were generally lower (except in a mast year) than those used in the overall model (Table 4). Since the proportional reduction in density at a given control rate varies negatively with r_m , a lower r_m results in lower population densities for a given continuous control rate. Sterilisation was slightly better than culling at reducing the summer peaks due to the birth pulse as discussed in Section 2.3.2 (Fig. 3), and this difference was more marked in a mast year when fertility was very high. However, continuous culling led to a more rapid reduction in stoat densities than did continuous sterilisation at the same rate (Fig. 5). This was because, with culling, individuals are instantly removed from the population but the effects of sterilisation are not realised until the following birth pulse. However, the difference in the rate of population decline between culling and sterilisation was not large, due to the relatively high intrinsic rate of increase of stoats (Barlow et al. 1997).

Figure 5. Predicted decline in relative stoat density under continuous control (at a rate of 0.04/month) based on Model 2 for a beech forest with mast cycles (vertical arrows indicate a mast year). Relative stoat density is calculated as N^*/K , where N^* and K are the equilibrium summer stoat densities in the presence and absence of control respectively.

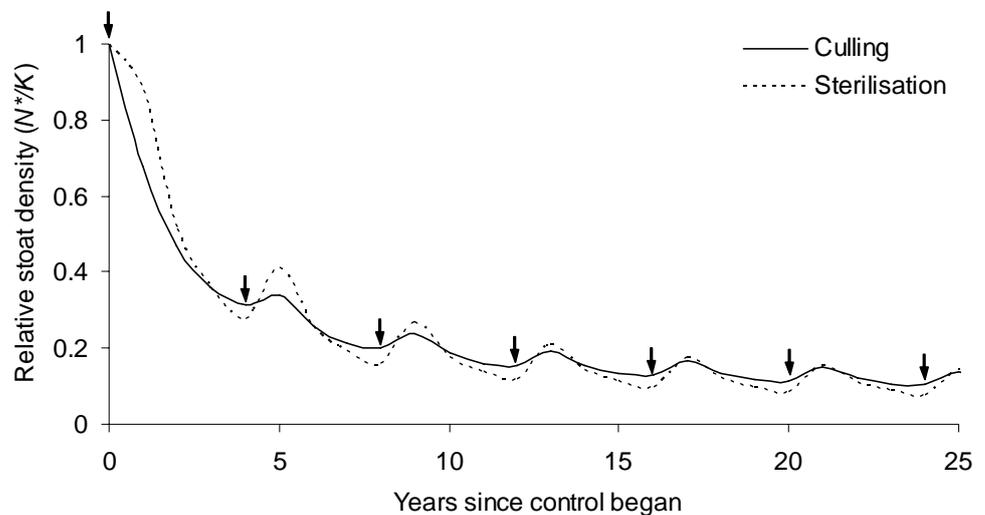


TABLE 4. INTRINSIC RATES OF INCREASE (r_m) AND PREDICTED REDUCTIONS IN POPULATION DENSITY UNDER CONTINUOUS CONTROL REGIMES USING THREE DIFFERENT MODELS FOR STOAT POPULATIONS IN BEECH FORESTS.

	r_m	PROPORTIONAL REDUCTION UNDER CONTROL REGIME (p):			
		CONTROLLING 0.02 OF POPULATION/MONTH		CONTROLLING 0.04 OF POPULATION/MONTH	
		CULLING	STERILISATION	CULLING	STERILISATION
Model 1	0.87	0.28	0.28	0.56	0.56
Model 2a overall	0.87	0.31	0.38	0.60	0.68
Model 2b with mast cycle	0.60, 1.39 ^M , -0.39 ^C , 0.60	0.48	0.53	0.92	0.94
Model 3	0.57, 0.94 ^M , -0.44 ^C , 0.68	0.65	0.67	1.00	1.00

Proportional reduction levels (p) are estimated as $p = 1 - N^*/K$ where N^* and K are equilibrium densities with and without control respectively. Estimates of N^* and K for the models that include a mast cycle (models 2b and 3) were calculated from the average stoat abundance over the 4 years of the mast cycle. M = maximum rate of increase from a normal to mast year, C = maximum rate of increase from mast to crash year.

2.3.4 Model 2: levels of control required to achieve specific population reductions

To cause a 50% reduction in mast year densities, the control rate had to be 0.021/month through culling or 0.018/month through sterilisation. To achieve an 80% reduction in mast year densities, the control rate had to be 0.034/month through culling or 0.031/month through sterilisation. Assuming control began in a mast year then the time taken for stoat densities to decline by 50% was the same under both culling and sterilisation and was the length of two mast cycles, approximately 8 years. Under the higher control rates the time taken for stoat densities to be reduced by 80% was three mast cycles or approximately 12 years. If control ceased, then the time taken for the stoat population to recover to pre-control levels depends on the level of control achieved. Thus for 50% reduction, recovery to pre-control peak densities took only one mast cycle. For an 80% reduction, the predicted recovery time was two mast cycles. Populations that were under sterilisation control took a little longer to recover than those that were culled, due to some sterilised females remaining in the population, but this difference was small and less than a year. In these population models, recovery is due to local recruitment only, the effects of immigration are not included.

2.3.5 Model 3: effects of age structure

King (1983) reported that for the Eglinton Valley in Fiordland the majority (85–92%) of the stoats trapped in a mast year were young born that season, but in a crash year this percentage is much lower (32%). The age structure of the summer stoat populations predicted by Model 3 was similar to that observed in the field, namely 82% young in a mast year and 30% in a crash year. However, the predicted fluctuations in stoat abundance over the course of a mast cycle were not as large in magnitude as those observed in the field (Fig. 4). This was because the population's capacity for increase under Model 3, as indicated by the r_m values, was generally lower than that under Model 2 which did predict

fluctuations in stoat abundance adequately (Table 4). These low rates of increase were a result of the strong density-dependent juvenile mortality assumed in the model. Low rates of increase meant that the predicted levels of control achieved by culling or sterilisation were higher than those predicted by Model 2 (Table 4), although sterilisation was still predicted to be slightly more effective than culling at reducing summer stoat densities. Because Model 3 did not adequately capture the changes in population abundance observed in the field, the effects of different control regimes predicted by this model were not explored any further.

2.3.6 Model 2: pulsed control

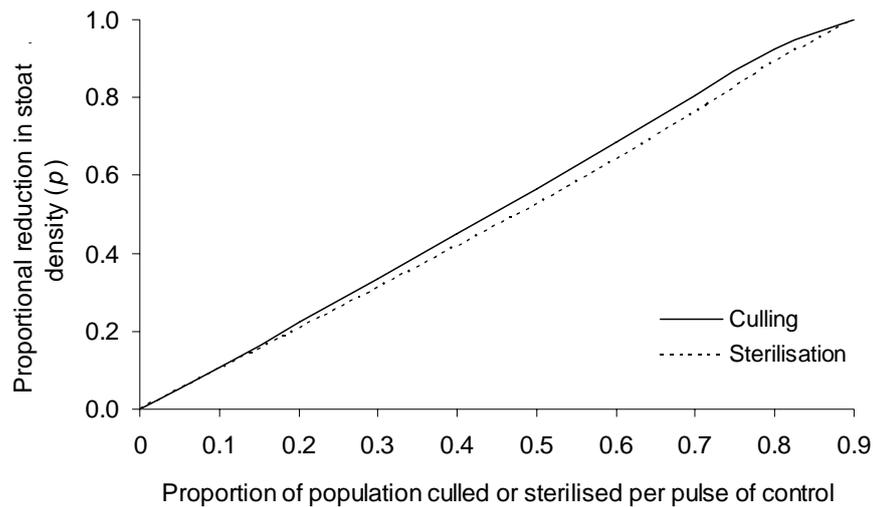
The control strategies discussed above assumed continuous culling or sterilisation efforts throughout the year, but in the field continuous control is difficult to sustain due to logistical and financial constraints. These results give the outcomes of pulsed control strategies to illustrate the optimum time to control within a year and within a mast cycle. A 'pulse' of control was simulated as 3 months of continuous control in spring, summer, autumn or winter using Model 2 with a mast cycle.

With annual pulsed control, culling in spring gave the highest reduction in average summer densities, followed by a summer or winter cull equivalently. Culling in autumn was the least effective, though the difference between control then and control in spring was small. For example, if culling took place at a rate of 0.20/pulse, stoat densities in a mast year were reduced by 39% and 44% for autumn and spring culls respectively. This difference was because autumn stoat densities are still relatively high and thus there is the potential for any removal by culling in autumn to be compensated for due to the strong density-dependent mortality acting at this time. In contrast, in spring, densities are low, so density-dependent mortality is minimal and the potential for compensation is less. The level of control achieved under sterilisation was not dependent on the timing of control because the effects of sterilisation were not realised until the birth pulse, and the model assumed that mortality rates were equal for sterilised and fertile stoats. For the same level of control, sterilisation proved more effective than culling at reducing summer stoat densities as was the case under continuous control. If stoats were controlled annually at a rate of 0.20/pulse, the model predicted a 53% reduction in mast stoat densities under sterilisation (in any quarter) versus the 44% reduction predicted from culling in spring.

If control was restricted to one pulse every 4 years then, not surprisingly, the best time within the mast cycle to reduce the mast birth pulse was in a mast year. Conversely, the worst year to control stoat populations was in a crash year. With the increased time between pulses of control, culling became slightly more effective than sterilisation at reducing mast year stoat densities. This was because under annual pulsed control there were still some sterilised animals left in the population from the previous year's control effort which, combined with the animals sterilised in the current year, enhanced control; but if control was applied only once every 4 years there was no such cumulative effect as all sterilised animals from the previous control effort had died. However, this difference between sterilisation and culling in terms of the

proportional reduction in stoat density was minimal: controlling at a rate of 0.20/pulse in the spring of a mast year gave a 22% reduction in stoat abundance under culling and a 21% reduction under sterilisation. Controlling at a rate of 0.40/pulse in the spring of a mast year gave a 45% and 42% reduction in stoat densities under culling and sterilisation respectively (Fig. 6). As depicted in Fig. 6, substantial reductions in population density were difficult to achieve under four-yearly pulsed control: the relationship between the proportional reduction in density and the control rate was almost one to one so for a 100% reduction in density, 90% of the population had to be removed at each pulse.

Figure 6. The proportional reduction in peak (summer) stoat population density in beech forests associated with different control intensities, given a 3-month pulse of control in the spring of a mast year (i.e. control only once every 4 years), based on Model 2.



2.4 DISCUSSION OF POPULATION MODELS

These simple population models have indicated that there is little difference in the effectiveness of sterilisation, compared with culling, in reducing mast year stoat populations if these controls are applied continuously at the same rate. Sterilisation was marginally better at suppressing summer densities than culling when control was applied continuously. This was because stoat density was assessed in summer after the birth pulse when the effects of sterilisation from the previous year had been realised. If control was pulsed every 4 years rather than continuous, culling was marginally better than sterilisation at reducing peak density because the proportion sterilised and hence the reduction in fecundity was less than under continuous sterilisation due to the loss of sterilised individuals from the population from the previous sterilisation effort. The parity of sterilisation and culling in suppressing stoat density is similar to the findings of Barlow et al. (1997) which were based on a continuous logistic model and their conclusions appear to hold true for birth pulse models and when there is large year-to-year variation in vital rates. The faster rate of decline in stoat density under culling compared with sterilisation was not as pronounced as that predicted for possum (*Trichosurus vulpecula*) populations (Barlow et al. 1997) because stoat populations have much higher death rates than do possums ($d = 0.7$ v. $d = 0.1$), so the maximum rate of decline under sterilisation ($= -d$) was greater for stoat populations and closer to the maximum rate of decline under culling ($c = 1$) (Barlow et al. 1997).

Given this predicted similarity in the effectiveness of culling and sterilisation, the choice between control methods might boil down to technical or social considerations. The models presented here were based on a non-disseminating form of sterilisation, which in practice would probably be a bait-delivered chemosterilant or immunocontraception. Hence, the cost and effort required to deliver these baits would be similar to those for conventional toxic baits but would also suffer the same problems such as patchy delivery and bait aversion (Bomford 1990; Norbury 2000). The mode of sterilisation assumed in the models was the disruption of implantation. The long period (9–10 months) of delayed implantation in stoats represents a larger window of opportunity for effecting female sterilisation than does the short period of oestrus and fertilisation (< 1 month) (Norbury 2000). However, if sterilisation affected fertilisation rather than implantation, we would expect sterilisation to be less effective than culling at reducing summer stoat densities. This is because a proportion of the young born each year would escape sterilisation (since they are fertilised in the nest before weaning and possible exposure to a sterilant) and thus the following year's potential productivity would still be high.

Perhaps the most glaring omission of detail from these models, and one which is likely to affect the relative effectiveness of culling compared with sterilisation, is the lack of immigration into the populations. Because stoats are often territorial, a sterilised female stoat maintaining her home range could effectively reduce recruitment from both local and external sources, giving sterilisation an advantage over culling, which leaves empty territories which are quickly recolonised. Caughley et al. (1992) and White et al. (1997) suggested that sterilisation could be compensated for by a change in the social structure of the targeted population allowing subordinate females, that would not otherwise breed, to have offspring. We would not expect this to happen in stoat populations because almost all female stoats caught in the wild are fertilised (King 1990), suggesting that total productivity will be linearly related to the proportion of unsterilised females as was assumed in the model. Characterising the correct social structure of the population under study is important, as previous work has shown that the breeding system of the modelled species can have a large influence on the predicted efficacy of sterilisation (Caughley et al. 1992; Barlow et al. 1997). Here, the breeding system assumed for the stoat models was 'Scenario 1' of Barlow et al.'s (1997) classification system. This covers polygamous mating where the females are sterilised and 'harem' systems in which a number of females mate exclusively with each dominant male and the females are sterilised. In spring, male stoats roam their extended home ranges, mating with multiple females and no pair bonds are formed (King & Murphy in press), so this assumption seems justified.

The results of this modelling exercise should be interpreted with some caution because the data on which they were based are limited and there are no field data available to validate the effects of sterilisation. The limitations of the models are discussed below with a view to identifying the types of data required to further refine the models and improve confidence in their predictions. The first critical assumption was that trap catch indices represent absolute density, i.e. that there is a linear relationship between captures per 100 trap nights and true stoat density and that this relationship does not change over time. Quantifying stoat abundance is notoriously difficult due to their low density,

small body size, and large home ranges (McDonald & Harris 1999; King & Murphy in press). The use of trap catch indices can lead to biases in estimates of stoat abundance because trappability can vary with sex, age (McDonald & Harris 2002; King 2003), and with season (McDonald & Harris 2002). There is also evidence that trap catch density indices underestimate stoat densities in summer because prey is plentiful and stoats might be less attracted to traps at this time of year (Alterio et al. 1999; King 2003). Assuming this underestimation is constant from year to year and across different locations, this would mean that the estimate of the strength of density dependence (the slope of the regression of r vs N_t) was larger than the true value, though the estimated value of r_m would be the same. Conversely, if we take into account the fact that trapping removes a certain proportion of the population, and assuming this removal is simply an additional density-independent mortality, this would mean that the strength of the density dependence would be estimated correctly but the value of r_m would be underestimated. Criticism could also be levelled at the method used to detect density dependence (r vs N_t), which assumes a particular form of density dependence (non-linear, with no delay) and is statistically dubious. More sophisticated tests for density dependence are available (e.g. Dennis & Taper 1994) but they require long time series (> 15 years) and are still affected by measurement error. Furthermore, changes in abundance can identify density relationships but will not reveal much about the mechanisms of density dependence. Here, we have assumed that density dependence operates on mortality because there is evidence that juvenile survival is negatively correlated with total stoat density (Powell & King 1997). There is no evidence for density dependence in fertility (McDonald & Larivière 2001), this being primarily determined by food supply (Powell & King 1997). We agree with McDonald & Larivière (2001) that much work needs to be done on identifying the nature and strength of any density dependent mechanisms in stoat populations. This is essential for improving the management of stoat populations as compensatory responses can greatly modify the impact of control.

Once control ceased, the recovery rates predicted by the model were slower than those observed in the field (Griffiths 1999). This was because the models assumed a closed population, with recovery dictated by the time until, and the size of, the next birth pulse. In the field, empty stoat habitats are rapidly recolonised by dispersing stoats. For example, Murphy & Dowding (1994) found that an area in the Eglinton Valley was re-invaded within 2 months of trapping being discontinued. More data is needed on stoat dispersal and recolonisation rates, particularly in relation to area and resident stoat density (Barlow & Choquenot 2002), so that more realistic, spatial models can be developed to predict control outcomes in the presence of immigration.

Stoat populations in non-beech forests are generally more stable than those in beech forests and attain densities similar to, or lower than, those in a beech forest in a non-mast year (King & Murphy in press). However the causes of population variation are not as obvious as in beech habitats and the data used to parameterise the model showed only a weak relationship between the rate of increase and population density, with considerable unexplained variation ($R^2 = 0.11$). Because of this low predictability, the quantitative results of the non-beech model are circumspect, although the greater efficacy of a given control

rate in non-beech forest compared with a beech forest should hold because the rates of increase in a non-beech forest rarely approach those in a beech forest in a mast year.

The beech mast cycle and associated stoat population response to increased prey density has been greatly simplified in the beech mast models. The effects of periodic seedfall on ecological communities are very complex (Ostfeld et al. 1996) and it would be naïve to assume that we have encapsulated the indirect numerical response of stoats to mast seeding within a 4-year cycle of: medium, medium, high, low productivity. In reality there is variation in the timing and size of beech mast events (Wardle 1984; Allen & Platt 1990) which will influence the demographic response of rodent and stoat populations. In terms of the model predictions, a consistently shorter cycle will increase the overall rate of increase making control more difficult, whilst a longer cycle will have the opposite effect. Including stochastic variation in the timing of the mast cycle is outside of the scope of the current models, which are deterministic, but could conceivably be incorporated into future stochastic models if a transition matrix could be quantified which described the probability of moving from one state (mast, crash, normal) to the next given the current state and the time since the last mast seedfall. More problematic is variation in the effect of a mast seedfall on stoat population responses, since a high seedfall year and associated high rodent density does not necessarily result in increased stoat density as was found by Byrom (2004) at Craigieburn following a beech mast in 2002. The low stoat densities observed in that study were mirrored by a similar decline in stoat numbers over the same period in a different (braided river) habitat (Dowding in Murphy & Fechny 2003), suggesting that some other mortality factor was overriding the typical positive response to increased food supply. Identifying the cause of this decline would not only improve understanding of stoat population dynamics but could possibly lead to a novel control method.

3. Disease/host models for CDV in stoat populations

3.1 DISEASE/HOST MODELS

3.1.1 Model 4: disease/host model for a simple epidemic

The simplest model for an epidemic of a reasonably virulent disease is an SIR-type model (Anderson & May 1992), which has one equation for the change (dx/dt) in each of three host classes—susceptibles (S), infectives (I), and recovered and immunes (R). The model is:

$$\frac{dS}{dt} = -\beta IS \quad \text{Eqn 4a}$$

$$\frac{dI}{dt} = \beta IS - \alpha I \quad \text{Eqn 4b}$$

$$\frac{dR}{dt} = (1 - \delta)\alpha I \quad \text{Eqn 4c}$$

where, β is the disease transmission coefficient, α is the loss rate from the infectious class through death or recovery ($= 1/\text{infectious period}$), and δ is the proportion of infected animals dying from the disease. No new susceptible animals enter the population so the disease must eventually die out, and the remaining population will consist of a mixture of animals which have caught the disease and recovered to an immune state, and those which did not become infected and are still susceptible. If the disease-free density of the population prior to an epidemic (S_0) is K then the contact rate, or number of potentially infectious contacts made per day per infectious host in a population at K , is βK . This model is essentially the same as for phocine distemper virus (PDV) in North Sea seals (Grenfell et al. 1992). Disease transmission is assumed to occur directly between hosts and the number of new infections per unit time is βSI where β is effectively the proportion of susceptibles infected per infected host per day. This transmission mechanism is described as ‘mass action’ because it resembles the law of mass action in reaction kinetics (McCallum et al. 2001). The number of infectious hosts recovering per unit time is the density of infectious individuals multiplied by the loss rate from the infectious class (α) multiplied by the proportion surviving infection ($1 - \delta$). The effect of any latent period is subsumed within the transmission coefficient.

An alternative assumption is that transmission depends on the proportion of individuals infected rather than the density infected. In this case the number of new infections per unit time is $\beta SI/N$ and the transmission mechanism is referred to as ‘frequency-dependent’ (McCallum et al. 2001). Mass action transmission would be expected where individuals mix freely and contacts between them depend on their densities. Frequency-dependent transmission occurs when the rate at which animals make contact is independent of density: each susceptible host contacts the same number of other individuals per unit time so the number of infectious contacts depends on the fraction of these

individuals that are infected. For example, if mating represents the main infectious contact, then the number of such contacts is likely to be relatively independent of density and best represented by frequency-dependent transmission.

3.1.2 Model 5: disease/host population model

To predict the course of disease over time and assess its long-term impacts on the host population, the simple epidemic model must be combined with a model for population growth of the host, in this case the birth pulse population model (Model 2). This gives a model of the form:

$$\frac{dS}{dt} = -(d + fN)S - \beta IS \quad \text{Eqn 5a}$$

$$\frac{dI}{dt} = \beta IS - (d + fN)I - \alpha I \quad \text{Eqn 5b}$$

$$\frac{dR}{dt} = (1 - \delta)\alpha I - (d + fN)R \quad \text{Eqn 5c}$$

and at the birth pulse:

$$S' = S + ((N - I)P) \quad \text{Eqn 5d}$$

where β is the disease transmission coefficient, α is the loss rate from the infectious class through death or recovery (= 1/infectious period), and δ is the proportion of infected animals dying from the disease. N denotes the total population density (= $S + I + R$), d is the density-independent death rate, and f is the density-dependence coefficient = r_m/K . At the birth pulse (S') only susceptible and recovered animals breed with a productivity P , and all new births go into the susceptible class. The frequency-dependent transmission model is the same but with βIS replaced by $\beta SI/N$ in the first two equations.

3.2 PARAMETERS FOR DISEASE MODELS

CDV is transmitted via aerosol or direct contact (Appel 1987) so disease transmission was assumed to occur directly between susceptible (S) and infectious (I) individuals. Vertical transmission (from mother to offspring) was not included in the stoat model since transmission does not occur across the placenta in mink (*Mustela vison*) (Hagen et al. 1970). There are no data available on CDV in stoats so the following parameters are mostly gleaned from studies of CDV infection in domestic ferrets (*Mustela furo*). For ferrets exposed to CDV, Ryland & Gorham (1978) report times from infection to death of 12–14 days for ferret-adapted strains and 21–25 days for canine strains. Given that any CDV strain released against stoats will probably be passaged through stoats to decrease its virulence to dogs and increase its virulence to stoats, the more rapid time to death of 12–14 days was considered most likely. Taking an average time to death of 13 days minus a latent period of 6 days (Crook et al. 1958), gives an average infectious period of 7 days and thus a loss rate (α) from the infectious class of: $\alpha = 1/7 = 0.143/\text{day}$.

The transmission coefficient, β , is the most problematic parameter to estimate. It is possible to estimate β from experimental manipulations or fit it to time series of prevalence in the field (McCallum et al. 2001), but there are no such data to estimate β for ferrets or stoats. Arbitrary values of β were chosen for the exploratory disease/host models presented here, where the aim was to compare the effects of different transmission models and the effects of different times of CDV introduction on relative host suppression rather than make quantitative predictions. Note that the same value of β will result in a different number of new infections per unit time in the mass action compared with the frequency-dependent model, since in the former the number of individuals infected per unit time is βSI whereas in the later it is $\beta SI/N$. Proportional mortality rates of ferrets infected with CDV range from 0.90 to 1.00 (Dunkin & Laidlaw 1926; Ryland & Gorham 1978); a conservative proportional mortality of $\delta = 0.90$ was chosen as the model default. Although there is a latent or incubation period of approximately 6 days when the affected individual is infected but not infectious (see above), a separate latent class was not included in the model because this latent period was short enough that that the possibility of an infected individual dying from other causes before becoming infectious was minimal. Furthermore, the inclusion of an incubating class did not substantially alter the disease dynamics in the simple epidemic model. It was assumed that there was no loss of immunity i.e., no transfer from the recovered immune (R) to susceptible (S) class, since infection from morbilloviruses like CDV generally results in lifelong immunity in recovered hosts (Barrett 1999).

3.3 RESULTS OF DISEASE/HOST MODELS

3.3.1 Model 4: disease/host model for a simple epidemic

The basic reproductive rate (R_0) of a disease is the expected number of secondary infections produced within the infectious period of one newly introduced host (Anderson & May 1981). For the simple epidemic model with mass action (MA) disease transmission this is:

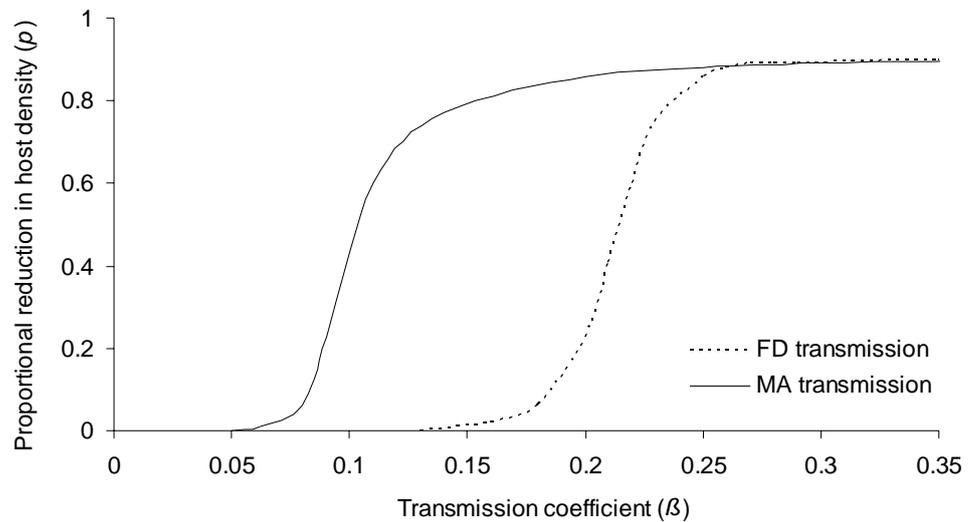
$$R_0 = \frac{\beta S}{\alpha} \quad \text{Eqn 6}$$

and for the corresponding model with frequency-dependent (FD) transmission, R_0 is:

$$R_0 = \frac{\beta}{\alpha} \quad \text{Eqn 7}$$

For an epidemic to proceed, the number of secondary infections produced by the original infectious host must be greater than one, therefore the condition $R_0 > 1$ must be met for initiation of an epidemic. If $R_0 = 1$ is substituted into equations 6 and 7 and solved for β , this gives an estimate of the minimum transmission coefficient (β_{\min}) required for disease maintenance. For the MA and FD models $\beta_{\min} = 0.062$ and 0.143 respectively, where $\alpha = 0.143$ and the initial density of susceptible individuals (S_0) is the equilibrium density of stoats in a beech forest, $S_0 = K = 2.29$. Figure 7 plots the proportional reduction in stoat density, p , against various values of β . Both models show no reduction in stoat density below β_{\min} , followed by increasing levels of p for higher values of

Figure 7. Results from a simple epidemic model for CDV (Model 4), showing the proportional reduction in host density for varying values of β , the transmission coefficient, for the MA and FD models, where $S_0 = 2.29$ and $\alpha = 0.143$.



β , asymptoting to $p = 0.9$ when all susceptible stoats become infected so that the maximum population reduction is simply the disease mortality rate ($\delta = 0.9$). Both models show that high values of β are required to overcome the short duration of the infectious period and cause a substantial reduction in stoat density.

If the initial stoat density (S_0) varies, as it can do between mast and non-mast years, then the MA model predicts, for a constant value of β , different impacts on host density. For higher values of S_0 there will be a greater proportional reduction in host density whilst for lower values of S_0 , the reduction in host density will be smaller. This is because the reproductive rate of the disease in the MA model is dependent on host density so increasing the host density increases the disease reproductive rate. Also, substituting $R_0 = 1$ into equation 6 and solving for S shows that there is a threshold susceptible host density (S_T) below which an epidemic cannot proceed and above which it can:

$$S_T = \frac{\alpha}{\beta} \tag{Eqn 8}$$

In contrast, the equation for R_0 for the FD model (equation 7) does not involve the density of susceptible individuals, therefore the FD model predicts the same proportional reduction in stoat density (for a constant β) regardless of the initial density of susceptible individuals. This means, that given a fully susceptible initial host population and provided $\beta > \alpha$, a disease can become established regardless of the host population density, i.e. is no threshold density (S_T) for an epidemic to proceed under the assumptions of the FD model.

The course of a simple epidemic for CDV is plotted in Figs 8A and 8B for the MA model and Figs 8C and 8D for the FD model, where the transmission rate, β , has been scaled to give the same reproductive rate, $R_0 = 2.4$, for both models. The speed of the epidemic is similar for both models, this is because they both had a similar R_0 and the speed of an epidemic is positively related to $\alpha(R_0 - 1)$ (Anderson & May 1992). The FD model predicted a larger number of stoats infected than the MA model and consequently a larger reduction in host density. This is because the FD model assumes a contact rate that is independent of host density, i.e. that the number of potentially infectious contacts made per

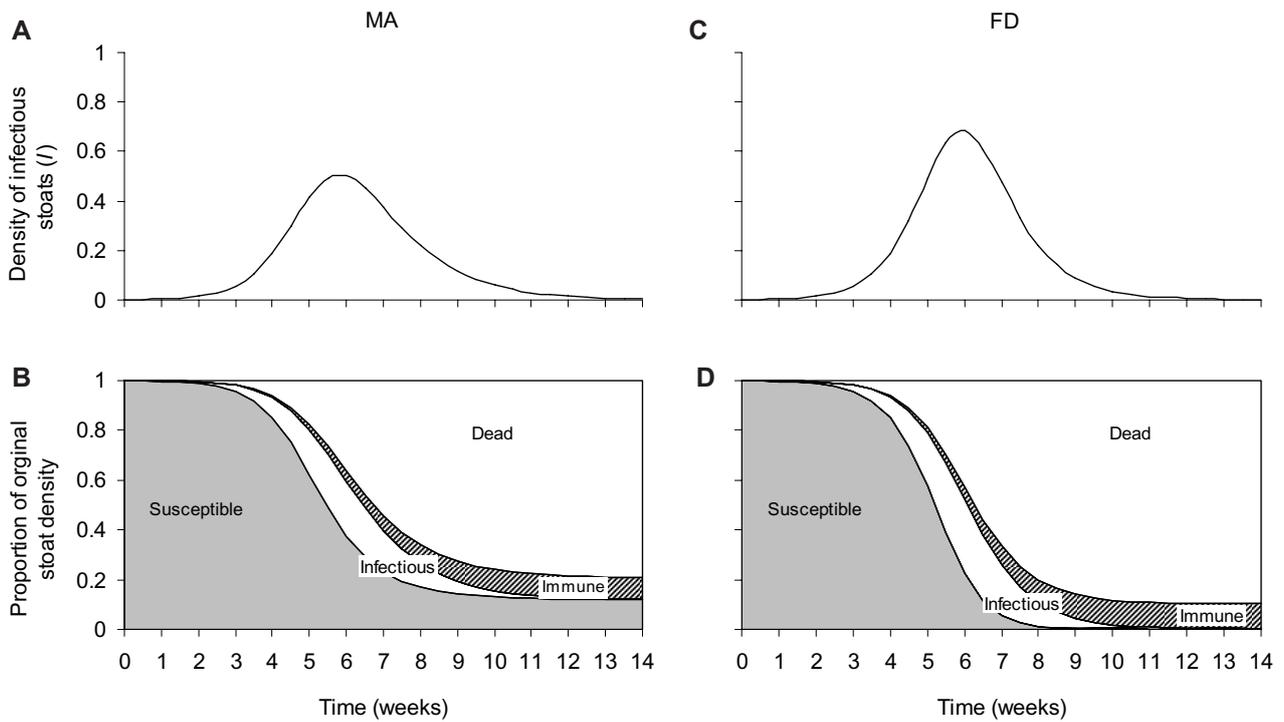


Figure 8. Results of a simple epidemic model for CDV (Model 4). Panels (a) and (c) show the density of infectious stoats, and panels (b) and (d) show the proportion of susceptible, infectious and immune stoats over the course of the epidemic. Panels (a) and (b) correspond to the MA model where $\beta = 0.15$, $\alpha = 0.143$, $\delta = 0.90$ and $S_0 = 2.29$; panels (c) and (d) correspond to the FD model where $\beta = 0.34$, $\alpha = 0.143$, $\delta = 0.90$ and $S_0 = 2.29$.

infectious host per unit of time remains the same regardless of host density. However, in the MA model, the contact rate is proportional to host density so that as host density starts to decrease due to disease-induced deaths, the contact rate and thus the number infected per day declines.

These simple epidemic models assume that other host population processes occurring do not affect the course of the epidemic. However, the influx of new, susceptible individuals through births or a decline in total population size through non-disease related deaths will affect the number of new infections per unit time. The effects of births and other sources of host mortality on the impact of CDV on stoat populations are explored in the following disease-host population models.

3.3.2 Model 5: disease/host population model

Much theory has been developed on the conditions for host population regulation by an endemic disease based on SIR-type models (e.g. Anderson & May 1981). However, these conditions were developed from equilibrium solutions for host density and disease prevalence which are probably not applicable to a CDV-stoat system for three reasons. Firstly, stoat breeding is strongly seasonal, occurring for a discrete period each spring, so that there is no constant influx of susceptibles as assumed by these continuous time models. Secondly, even between years, there is large variation in stoat density due to the effects of mast seeding, so that host density is very unstable, which is not conducive to disease persistence. Thirdly, the short disease duration and high

mortality rates assumed for CDV in stoats are characteristic of epidemic rather than persistent/endemic disease. It was, therefore, concluded that CDV was unlikely to persist as an endemic disease within local populations and that the logistic model with its continuous births was not appropriate to predict CDV epidemiology. Instead, the more realistic birth pulse model (Model 2), was used to investigate the timing of CDV introduction in relation to the mast cycle and birth pulse and the effects of compensatory mortality on the relative size of the epidemics. Also, the time taken for stoat populations to recover to densities at which they were susceptible to future outbreaks and the size of these future outbreaks was investigated.

The basic reproductive rate of the disease is similar to that of the simple epidemic, except that non-disease mortality also contributes to the loss of infectious stoats. Thus for the MA population model:

$$R_0 = \frac{\beta S}{\alpha + d + fN} \quad \text{Eqn 9}$$

and R_0 for the population model with FD transmission is:

$$R_0 = \frac{\beta}{\alpha + d + fN} \quad \text{Eqn 10}$$

where d is the density-independent, non-disease mortality, and f is the density-dependence coefficient = r_m/K . For the MA model, a high disease transmission rate is required to initiate an epidemic in late winter/early spring, before the birth pulse, as stoat densities are very low at this time of year. Assuming that an epidemic would not occur in the late winter/early spring following a crash breeding season because stoat densities are generally too low, but that an epidemic is possible at this time of year following a normal or mast breeding season, this gives an estimated minimum transmission rate of: $\beta_{\min} = 0.25$ (where $S_0 = N_0 = 0.6$, $d = 0.0021$ and $f = 0.0012$) for the MA model. The predicted epidemics for different disease introduction times are shown for both the MA (Fig. 9) and FD models (Fig. 10) using $\beta = 0.25$. Given that it is unknown what form or value the transmission function would actually take, it is best to interpret these results by comparing the relative rather than the absolute size of the epidemics produced by different disease introduction times.

For the MA model, if CDV was introduced in January after the birth pulse, the size and speed of the epidemic was determined primarily by the initial density of susceptible stoats. So the greatest reduction in relative stoat density occurred following a large birth pulse, in a mast breeding season (Fig. 9E) and the least reduction occurred following a crash breeding season (Fig. 9F). If CDV was introduced in November before the birth pulse, initially only low levels of infection resulted and the reduction in relative host density was minimal. However, once births occurred and new susceptible stoats entered the population, an epidemic was initiated where again the final size of the epidemic was determined by the size of the birth pulse (Figs 9A-C). Because the size of the epidemics was determined primarily by the size of the birth pulse, whether this was before or after the introduction of CDV, similar reductions in host density were predicted for both November and January introductions (compare Figs 9A to 9D, 9B to 9E, and 9C to 9F).

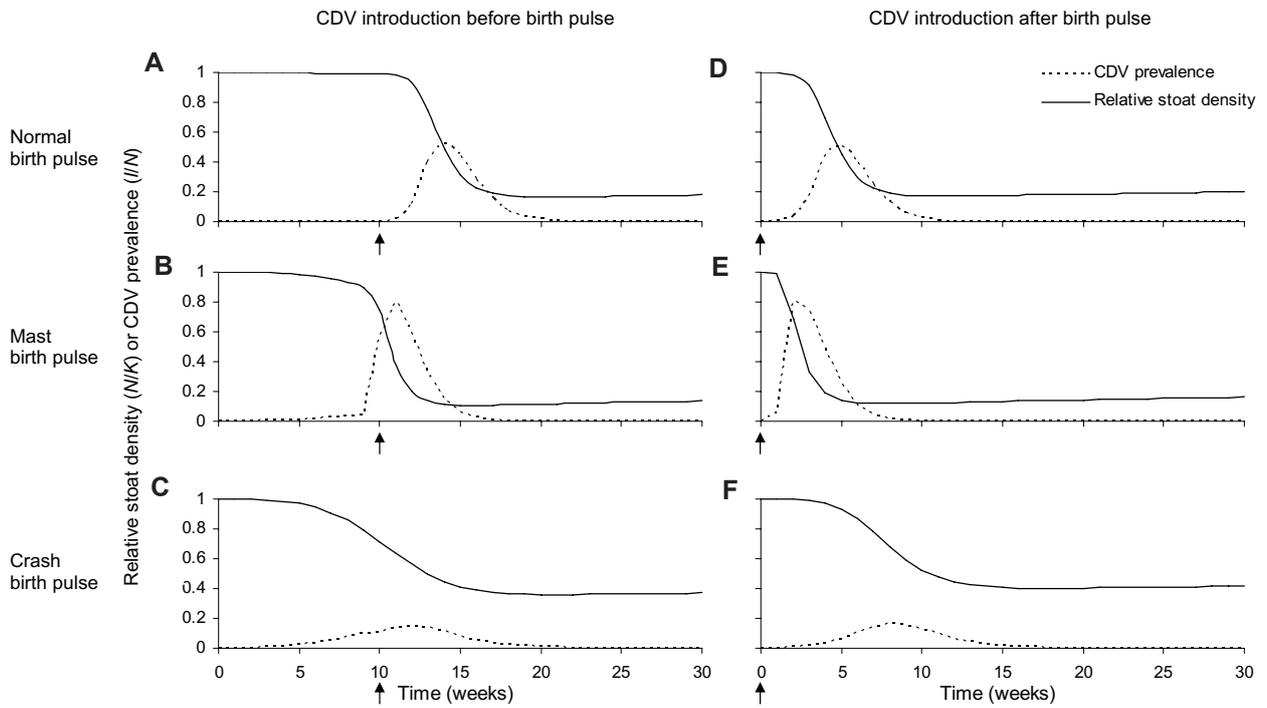


Figure 9. The size of the epidemic and the decline in relative host density caused by introducing CDV into a stoat population at different stages of the mast cycle, and before, panels (a)–(c), or after, panels (d)–(f), the birth pulse; based on the MA version of Model 5 where $\beta = 0.25$, $\alpha = 0.143$ and $\delta = 0.90$. Vertical arrows indicate the timing of the birth pulse.

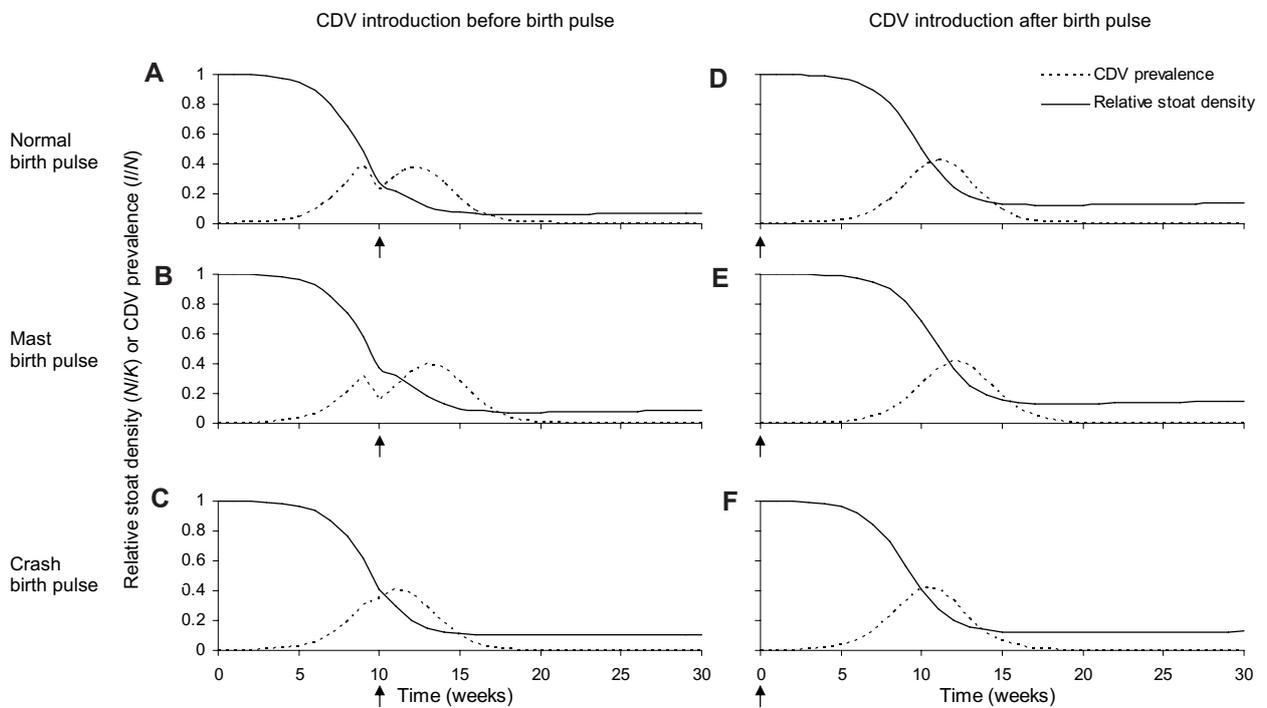


Figure 10. The size of the epidemic and the decline in relative host density caused by introducing CDV into a stoat population at different stages of the mast cycle, and before, panels (a)–(c), or after, panels (d)–(f), the birth pulse; based on the FD version of Model 5 where $\beta = 0.25$, $\alpha = 0.143$ and $\delta = 0.90$. Vertical arrows indicate the timing of the birth pulse.

For the FD model, if CDV was introduced in January after the birth pulse, the speed of the epidemic was slightly faster and the final reduction in host density was slightly larger at lower initial host densities (see Fig. 10F compared with Figs 10D and 10E). This was because strong density-dependent mortality following normal and mast breeding seasons compensated for the losses through infection but, following a crash breeding season, when stoat densities were low anyway, there was no such compensation. If CDV was introduced in November before the birth pulse, there was only weak density dependence operating at this time and epidemics produced at different stages of the mast cycle were initially very similar (Figs 10A–C). Once births occurred, this increased the proportion of susceptibles in the population in normal and mast years but not appreciably in a crash year. However, increases in the infection rate due to the increased proportion susceptible were offset by the increased density-dependent mortality following a large birth pulse. Thus it was at intermediate densities, following a normal breeding season, that the largest reduction in relative host densities occurred (Fig. 10A).

In summary, the MA model showed the most sensitivity to the variations in density caused by the mast cycle, so that the control rate was better if CDV was introduced in a peak stoat year. The influence of the initial density of susceptibles on the infection rate overrode any density-dependent compensation through other sources of mortality which is not surprising given that disease dynamics are much more rapid than density dependent mortality ($\beta \gg f$). In contrast, for the FD model, the initial density of susceptibles didn't influence the size of the epidemic but strong density-dependent mortality in peak stoat years did slightly reduce the impact of disease.

For both the MA and FD models, persistent (endemic) disease could not be generated even if the loss rate from the infectious class was halved ($\alpha = 0.07$) and/or very high values of the transmission rate (up to $\beta = 1$) were used. This was because of the birth pulse structure of the model which meant that there was not a constant influx of susceptibles to sustain the disease.

The time taken for stoat densities to recover from an epidemic is proportional to the level of control achieved so is the same as that predicted from the stoat population models (see Section 2.3.5). The recovery time to densities at which the population is susceptible to further CDV outbreaks is dependent on the values for the disease parameters. These values determine the initial reduction in stoat densities and the threshold density of susceptible stoats (MA model) or the threshold proportion of susceptible stoats (FD model) which the population must attain for a second epidemic to be initiated. In practice, the time to recovery is determined largely by the time until, and the size of, the next birth pulse. The influence of the number, or proportion, of immune stoats in the population is minimal because of the low recovery rate from CDV ($= 0.1$), the assumption that immune females produce susceptible offspring, and the high turnover rates in stoat populations. For a CDV epidemic initiated in November, where $\beta = 0.25$, $\alpha = 0.143$, and $\delta = 0.9$ as before, the FD model predicts that stoat populations will be susceptible to another CDV epidemic after approximately 1 year, at the next birth pulse, providing this is a normal or mast breeding season. If the post-epidemic birth pulse is a normal one a reduction in stoat density of approximately 55% is predicted; if the birth pulse is a mast one

then a reduction of 67% is predicted. However if the post-epidemic birth pulse is a crash one, the population will not be susceptible for another year. For the MA model, with the same parameter values, the recovery time will depend on the timing of the initial epidemic in relation to the mast cycle. If an epidemic occurs before a mast year it will only take approximately 1 year before stoat densities reach the threshold susceptible host density, $S_T = 0.58$, and another epidemic which reduces stoat populations by 20% is possible. If, however, a mast birth pulse is 2-4 years away it will take this long for the populations to reach the threshold density, but by then the density of susceptible hosts will be high enough (> 1.7 C/100TN) to cause a large epidemic which reduces stoat density by 80%.

3.4 DISCUSSION OF DISEASE/HOST MODELS

CDV was predicted to not persist as an endemic disease in stoat populations, primarily because of the birth pulse structure assumed in Model 5 which precludes the continuous recruitment of susceptibles required to maintain the disease within a host population. A birth pulse seems a reasonable approximation of the field situation, where births are synchronised by day length cues, thus the practical use of CDV would appear to be as an inundative rather than a classical biocontrol agent. It is possible that spatial interactions, such as ongoing recruitment of susceptible individuals to the population by immigration or the spread of CDV to, and re-infection from, neighbouring populations could provide a mechanism for disease persistence but the rapid dynamics predicted might preclude even this, since by the time the affected population has recovered to levels where it is susceptible again, the epidemic wave will have travelled far away and there will be no source for re-infection. The rapid dynamics were a result of the short infection period ($1/\alpha$) assumed in the model which was estimated from CDV infection in ferrets rather than stoats. If a suitable vaccine strain of CDV can be developed (Zheng in Murphy & Fechny 2003), then inoculation of captive stoats should provide estimates of the average infectious period and mortality rates of CDV in stoats and improved model predictions could be made. However, it will be very difficult to estimate the disease transmission rate from laboratory studies; the most robust way of estimating the transmission rate is from monitoring a natural epidemic (McCallum 2000).

It is also important to determine how the transmission rate, specifically the contact rate component (the number of potentially infectious contacts made per infected host per unit time), scales with host density in the field as this will determine whether transmission is density- (MA) or frequency-dependent (FD). The implications of the mode of transmission (MA or FD) are significant for the use of CDV as a biocontrol agent for stoat populations, which are generally at low densities. For example, if transmission is density dependent (MA), then this implies that there will be some host density threshold that needs to be exceeded before an epidemic can be initiated and this may preclude the use of CDV at times of the year, such as winter and spring, when stoat densities are below this threshold. This is problematic because, under the assumptions of MA transmission, the best time of year to initiate a CDV epidemic would be in

summer when stoat density is highest, yet the purpose of control is to prevent these high densities from occurring in the first place. On the other hand, if transmission was frequency dependent, then a CDV epidemic could be initiated in late winter to early spring when densities are low, reducing the number of potential breeders and thus peak summer densities. It is possible that contact rates between stoats are frequency- rather than density-dependent during the breeding season when the roaming males probably make the same number of contacts with females regardless of density, so provided the transmission rate is high enough ($\beta > \alpha$), the population would be susceptible to a CDV epidemic. It is worth noting that several studies of the epidemiology of wildlife diseases have found little empirical evidence to distinguish between the two different transmission functions (McCallum 2000; McCallum et al. 2001), indicating that the problem is not confined to the disease-host system presented here. CDV epidemics in wild populations of black footed ferrets and lions have shown evidence of a threshold host density for disease initiation (May 1986; Packer et al. 1999), which is suggestive of a MA transmission function. However, since the contact rate component of the disease transmission function depends on the social structure of the host population rather than being an attribute of the virus, we cannot assume the same transmission function for CDV in stoat populations.

An assumption implicit in the MA transmission function is that of homogeneous mixing, i.e. susceptible and infected hosts mix completely with one another so that contacts between them are random and in proportion to their respective densities. Populations are rarely homogenous in nature, often showing aggregation in response to underlying resource distributions. Heterogeneity can also be caused by individual variation in susceptibility to disease. Heterogeneity in the risk of becoming infected effectively lowers the disease transmission rate when averaged over different populations or over individuals of differing susceptibility. Barlow (2000) showed that it was necessary to include some heterogeneity of risk in possum-Tb models to mimic the disease dynamics observed in the field, namely high possum density in the presence of Tb and a low possum threshold density for Tb elimination. He speculated this heterogeneity could be due to spatial variation in habitat carrying capacity so that possum density in some habitats is above the threshold for disease persistence yet below it in others, which could explain the 'hot-spots' of high disease prevalence observed in the field (Barlow 2000). There is some evidence that stoat populations are aggregated in space (J. Christie, DOC, pers. comm. 2004), so it is likely that CDV infection would also be patchy in space. The models presented here also assumed that the stoat populations were fully susceptible before the introduction of CDV and that all individuals were equally susceptible. The results of a screening study of viral diseases, including CDV, potentially present in New Zealand stoat populations (McDonald in Murphy & Fechney 2003) should give some indication of how susceptible New Zealand stoat populations are to CDV.

Since the values of the transmission rate, β , used in the model were arbitrarily chosen, it is not worth dwelling on the quantitative predictions of the model. For a given β , the models showed that, under the assumptions of MA transmission, relatively lower population densities were achieved by releasing CDV in a mast year when stoat densities were highest. Compared with the MA

model, the predicted reduction in population density under the FD model was less sensitive to the initial density of susceptibles (S_0), but more sensitive to compensation in host mortality rates (f). These conclusions are encapsulated by equations 9 and 10 for the reproductive rate for the respective models. The type of model presented here will be useful for comparison and evaluation of potential disease biocontrol agents. Provided estimates for the infectious period ($1/\alpha$) and the disease mortality rate δ can be obtained, and the relative infectiousness of the diseases can be estimated (so that the β s can be scaled accordingly), then simple epidemic models could be used to determine the relative effectiveness of each potential biocontrol agent. In terms of selecting the best classical biocontrol agent, theory predicts that the highest levels of control will be achieved with diseases of intermediate pathogenicity (Anderson & May 1981). This is because if the mortality rate from infection is too high (large $\delta\alpha$), infected hosts die before effective transmission is achieved and the disease does not persist in the host population. In this case, the disease transmission rate would have to be very high to offset the large loss rates from the population and ensure persistence (i.e. to make the numerators of equations 9 and 10 larger than the denominators, giving $R_0 > 1$). Stoat populations in beech forests are so unstable that, for CDV at least, persistent disease in local populations is unlikely. It is possible that CDV could be maintained in an alternative host species such as ferrets which could provide a source of re-infection for stoat populations, although epidemic rather than endemic dynamics are likely to result from this re-infection due to the short infectious period of CDV. If a classical biocontrol agent is sought for stoat populations, it may be better to look for a pathogen that has a mechanism for persistence when stoat densities are very low such as a pathogen that has a free-living (outside the host) infective stage or one that causes latent (covert) disease in some individuals (Anderson & May 1981). On the other hand, the predicted epizootic behaviour of CDV in stoat populations may make it useful as an inundative biocontrol agent which can be released before an anticipated high stoat density year (e.g. after a mast seedfall) to produce an epidemic and rapidly reduce stoat populations. With inundative biocontrol there is no expectation that the disease will persist in the host population but this could prove advantageous in terms of slowing the evolution of host resistance to the disease since the period of disease-host association is brief.

4. General conclusions and recommendations for future research

The birth pulse model with different intrinsic rates of increase in mast, crash and normal years (Model 2), best predicted the large year-to-year variation in stoat abundance observed in beech forest. Using this model, little difference was predicted in the long-term proportional reduction of stoat density under culling or sterilisation control. Under continuous control, sterilisation was slightly more effective at reducing peak (summer) stoat density; however, under pulsed control, culling was marginally more effective than sterilisation. Control of either kind was relatively more effective against populations in non-beech forests than against those in beech forests, essentially because populations in non-beech habitats have a lower intrinsic rate of increase. However, more robust estimates of population parameters in non-beech habitats are required before we can make quantitative predictions for these populations.

The results of the models for both habitat types are only applicable to closed populations but, in reality, there is considerable dispersal between local stoat populations and this dispersal could change the relative effectiveness of sterilisation and culling control. For example, if immigration into a local population is dependent on local population density then sterilisation could be more effective than culling at reducing local population density. This is because it reduces recruitment both from local sources by reducing the birth rate and from external sources because the sterilised female is still present to maintain her territory and prevent immigration. In contrast, culling increases mortality but results in empty territories which are vulnerable to recolonisation from neighbouring populations. Therefore, the main recommendation for further research into stoat population dynamics is to incorporate spatial structure into the population models. It is envisaged that a spatial model would take the form of a grid of local populations which are linked by dispersal. To parameterise this type of model, data on stoat dispersal and recolonisation rates would be required.

Model 2 was also used to predict the likely dynamics of canine distemper virus (CDV) in stoat populations. The model assumed a short infectious period and a high disease mortality rate which produced a short-lived epizootic in the modelled stoat populations. CDV was not predicted to persist as an endemic disease in New Zealand stoat populations because strongly seasonal breeding precludes the continuous recruitment of susceptibles required to maintain CDV within the stoat population. Similarly, very low birth rates in crash years of the mast cycle would result in susceptible host densities below the threshold for disease maintenance. It is possible that the spread of CDV to, and re-infection from, neighbouring populations could provide a mechanism for disease

persistence and it would be useful to explore this possibility using a spatial population model.

Whether transmission is density- (MA) or frequency-dependent (FD) may be critical to whether CDV could be used to initiate an epizootic before the birth pulse when stoat densities are very low. However, as CDV is just one of many candidate biological control agents (McDonald & Larivière 2001), researching the transmission behaviour of a disease that may never be introduced could represent unwarranted effort. Instead, the simple models developed here could be used to make general comparisons among candidate biocontrol agents under both MA and FD transmission assumptions, and transmission mechanisms could then be investigated once the most promising candidates had been identified.

Quantitative model predictions are only as good as the data on which they are based. The following list identifies the types of data required to obtain better parameter estimates and further develop the models.

- Estimates of stoat dispersal and recolonisation rates in relation to stoat density and the size of the area controlled, to parameterise a spatial model.
- More long-term (> 4 years) time series of annual stoat densities, particularly in non-beech habitats, to obtain better estimates of the intrinsic rate of increase.
- Identification of the factors driving variation in stoat abundance in non-beech forests.
- Validation/testing of the negative relationship between juvenile stoat survival and population density.
- Identification of the relationship between trap-catch indices of density ($C/100TN$) and actual stoat density.
- Estimation of disease parameters for other potential biological control agents for stoats (e.g. Aleutian disease virus) to plug into model and compare with predicted CDV epidemiology.

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Sadly, the senior author Nigel Barlow died midway through this project and I (MB) would like to credit Nigel with doing most of the model formulation, and thank him for his inspiration and teachings in the discipline of population modelling in general. Nigel was posthumously awarded the Australasian Wildlife Management Society's prestigious Caughley Medal in 2003 for his outstanding contribution to wildlife management and ecology.

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Appendix 1

PEAK (SUMMER) STOAT DENSITY INDICES (N) IN SUCCESSIVE YEARS (t and $t+1$)

DATA FROM STUDIES WHERE STOAT TRAP CATCHES WERE MONITORED FOR MORE THAN ONE YEAR.

FOREST TYPE	SITE	YEAR	N_t	N_{t+1}
Non-beech	Northern Urewera* (Otamatuna, DOC 1999a)	97	0.330*	0.190*
Non-beech	Northern Urewera* (Otamatuna, DOC 1999a)	98	0.190*	0.390*
Non-beech	Boundary Stream* (DOC 1999b)	97	0.160*	0.175*
Non-beech	Mapara (Murphy et al. 1998)	90	0.219	0.227
Non-beech	Mapara (Murphy et al. 1998)	91	0.227	0.188
Non-beech	Mapara (Murphy et al. 1998)	92	0.188	0.211
Non-beech	Mapara (Murphy et al. 1998)	93	0.211	0.250
Non-beech	Mapara (Murphy et al. 1998)	94	0.250	0.159
Non-beech	Trounson (DOC 1999c)	97	0.172	0.100
Non-beech	Trounson (DOC 1999c)	98	0.100	0.065
Non-beech	Pureora (exotic forest, King et al. 1996)	83	1.500	0.900
Non-beech	Pureora (exotic forest, King et al. 1996)	84	0.900	0.500
Non-beech	Pureora (exotic forest, King et al. 1996)	85	0.500	0.300
Non-beech	Pureora (exotic forest, King et al. 1996)	86	0.300	0.800
Non-beech	Pureora (unlogged podocarp, King et al. 1996)	83	0.550	0.300
Non-beech	Pureora (unlogged podocarp, King et al. 1996)	84	0.300	0.700
Non-beech	Pureora (unlogged podocarp, King et al. 1996)	85	0.700	0.000
Non-beech	Pureora (unlogged podocarp, King et al. 1996)	86	0.000	0.350
Non-beech	Pureora (logged forest road edge, King et al. 1996)	83	0.900	0.250
Non-beech	Pureora (logged forest road edge, King et al. 1996)	84	0.250	0.300
Non-beech	Pureora (logged forest road edge, King et al. 1996)	85	0.300	0.550
Non-beech	Pureora (logged forest road edge, King et al. 1996)	86	0.550	0.550
Non-beech	Mapara (Murphy & Bradfield 1992)	89	0.120	0.150
Beech	Mount Misery (Wilson et al. 1998)	75	2.700	4.100
Beech M	Mount Misery (Wilson et al. 1998)	76	4.100	4.000
Beech C	Mount Misery (Wilson et al. 1998)	77	4.000	2.700
Beech	Mount Misery (Wilson et al. 1998)	78	2.700	5.300
Beech M	Mount Misery (Wilson et al. 1998)	79	5.300	5.400
Beech C	Mount Misery (Wilson et al. 1998)	80	5.400	1.400
Beech	Mount Misery (Wilson et al. 1998)	81	1.400	1.400
Beech M	Mount Misery (Wilson et al. 1998)	82	1.400	5.400
Beech	Eglinton (King 1983)	75	1.460	0.700
Beech M	Eglinton (King 1983)	76	0.700	5.430
Beech C	Eglinton (King 1983)	77	5.430	1.420
Beech C	Eglinton (King 1983)	80	5.490	0.430
Beech	Hollyford (King 1983)	75	9.280	1.630

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N = no. caught/100 uncorrected trap nights.

* Indicates numbers were corrected.

M = mast year, C = 'crash year' following a mast year.

Appendix 1 continued

FOREST TYPE		SITE	YEAR	N_t	N_{t+1}
Beech	M	10 Hollyford (King 1983)	76	1.630	4.450
Beech	C	10 Hollyford (King 1983)	77	4.450	0.840
Beech	C	10 Hollyford (King 1983)	80	6.190	0.220
Beech	M	11 Craigieburn (King 1983)	74	0.720	1.350
Beech		11 Craigieburn (King 1983)	75	1.350	2.860
Beech	M	11 Craigieburn (King 1983)	76	2.860	3.680
Beech	C	11 Craigieburn (King 1983)	77	3.680	1.250
Beech		12 Mount Cook (King 1983)	75	0.940	2.970
Beech		12 Mount Cook (King 1983)	76	2.970	1.800
Beech	C	13 Eglinton (Deer Flat, Murphy & Dowding 1995)	90	0.362	0.178
Beech		13 Eglinton (Deer Flat, Murphy & Dowding 1995)	91	0.178	0.089

N = no. caught/100 uncorrected trap nights.

* Indicates numbers were corrected.

M = mast year, C = 'crash year' following a mast year.

Appendix 2

JUVENILE DENSITY-DEPENDENT MORTALITY FUNCTION IN MODEL 3

First-year survival versus stoat density (trap catches per 100 corrected trap-nights (Stoats/100CTN)) in the birth summer used to estimate juvenile density-dependent mortality function in Model 3. Data are from Powell & King (1977).

STOATS/100CTN	1ST YEAR FINITE SURVIVAL RATE (s)	LOG SURVIVAL RATE, LN(s)
0.73	0.34	-1.07881
1.36	0.137	-1.98777
1.456	0.066	-2.7181
1.646	0.243	-1.41469
1.709	0.212	-1.55117
2.848	0.168	-1.78379
3.703	0.018	-4.01738
4.462	0.018	-4.01738
5.443	0.018	-4.01738