Predator-induced breeding suppression and its consequences for predator–prey population dynamics

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SUMMARY

Recent empirical evidence demonstrates that certain small mammals suppress breeding in response to strong predation pressure, the interpretation being that non-breeding individuals have a better chance of avoiding predation than those in a reproductive state. A separate strand of recent research has sought to explain empirical observations of cycling in small mammal (especially vole) populations of Fennoscandia as being due to their interaction with predator populations. Predator-induced breeding suppression (PIBS) is likely to have an effect on these predator–prey dynamics, and here we present a model designed to investigate this effect. We find that a strong enough level of PIBS acts to stabilize predator–prey cycles, and that weaker levels reduce the amplitude and increase the frequency of existing oscillations. These effects are explained in terms of the intrinsic mechanism driving the predator–prey cycles. We do not find circumstances where PIBS acts to destabilize an intrinsically stable interaction. We identify the features of breeding suppression that have the strongest effect on dynamic stability (e.g. quick recovery to breeding condition as predation pressure reduces), and hence pinpoint areas where future empirical research is required.

1. INTRODUCTION

The population cycles of small mammals have fascinated population ecologists for several decades (see Krebs *et al.* (1995) for a recent overview). Attempts to explain these cycles have historically focused on factors intrinsic or extrinsic to the population which, at high population density, induce delayed density-dependence of the sort likely to promote cycling. ‘Intrinsic’ theories focus on specific sorts of stress that may become magnified at high population density (e.g. social strife, competition for food, etc.), whereas ‘extrinsic’ theories focus mainly on density-dependent predation (Krebs & Myers 1974; Taitt & Krebs 1985).

The last 30 years have seen a great deal of debate, and relatively little consensus on the validity of theories, based on extrinsic or intrinsic factors as the ultimate explanation for population cycling (Krebs *et al.* 1995). However, recent work provides relatively strong evidence that predation is an important determinant of population cycling in at least some systems. For instance, Korpimäki (1993) and Norrdhal & Korpimäki (1995) claimed to have identified density-dependent predation on rodents of the sort needed to regulate populations. Hanski *et al.* (1993) described a predator–prey model that predicts quantitatively the cycling of voles in Fennoscandia. Krebs *et al.* (1995) provided a rare, large-scale predator manipulation experiment that supports the idea that mammalian predators drive (at least to some extent) the cycling of

snowshoe hare (*Lepus americanus*). On the other hand, there have been important new theoretical developments regarding intrinsic factors and population cycling (e.g. Boonstra 1994; Lomnicki 1995), although Ostfeld *et al.* (1993) found little experimental evidence for the sort of stress assumed by such models.

We focus here on predation and its effects on population cycles. Our focus is not so much on the direct (lethal) effects of predation, but on recent speculation regarding the way in which such cycling is influenced by a particular anti-predator response, i.e. predator-induced breeding suppression (PIBS).

Several recent papers report that the presence of predators can induce a marked reduction in reproduction in several boreal mammals (Lima 1996). Ylönen (1989) first reported that bank voles (*Clethrionomys glareolus*) strongly suppress reproduction upon exposure to the scent of mustelid predators. Similar degrees of PIBS have been observed in other laboratory experiments on bank voles (Ylönen *et al.* 1992; Ronkainen & Ylönen 1994; Ylönen & Ronkainen 1994), other *Clethrionomys* voles (Ylönen *et al.* 1992; Heikkilä *et al.* 1993), and *Microtus* voles (Koskela & Ylönen 1995). Korpimäki *et al.* (1994) also demonstrate PIBS in bank voles under field conditions. The mechanism behind this breeding suppression may be predator-induced behavioural changes in mating (Ylönen & Ronkainen 1994; Ylönen 1994) or feeding (Heikkilä *et al.* 1993) and/or physiological changes involving the oestrous cycle (Koskela *et al.* 1996). The

adaptive basis of PIBS, as argued by Ylönen (1994; see also Oksanen & Lundberg 1995), lies in the possibility that non-reproductive females experience enhanced survival during the high-predation portion of a population cycle, after which their reproductive efforts would be more successful. However, Kokko & Ranta (1996) suggest that PIBS may be optimal only under rather restrictive conditions.

We focus in particular on suggestions that PIBS might influence the nature of predator-prey population cycling. Specifically, Ylönen (1994) and others suggest that PIBS hastens the crash phase in the cyclic population dynamics of boreal voles. Hik (1995) makes a similar argument for the potential importance of PIBS in snowshoe hare cycles, and further suggests that long-term breeding suppression by the hare might lengthen the recovery phase of a given cycle. We present a simple predator-prey population model that demonstrates that adaptive PIBS may indeed have these sorts of effects on predator-prey cycling. However, short-term (or flexible) PIBS generally has the effect of lessening or eliminating the cycles themselves (i.e. it promotes stability in the interaction between predator and prey), but this stabilization pressure is reduced if breeding suppression is long-term. Our model also highlights some important gaps in our present understanding of the biology behind PIBS.

2. THE MODEL

We consider the prey population to be divided into two subpopulations, one that breeds (B) and one that suppresses breeding (S). The breeding population is exposed to the predator population (P), but the suppressors are not. Predators capture breeding prey according to a simple Type II functional response and are subject to a constant per capita death rate (μ), so that the rate of change in the predator population is given by

$$\frac{dP}{dt} = \frac{\epsilon I_m B P}{B + B_0} - \mu P, \quad (1)$$

where ϵ is the efficiency with which prey are converted to predators, I_m is the maximum intake rate (the inverse of the handling time) and B_0 is the half-saturation constant that controls the shape of the functional response.

Individuals in the breeding population move to the suppressor population with a rate ϕ , which depends on both the breeding population size and the predator population size. We assume that $\phi(P, B)$ is given by

$$\phi(P, B) = \frac{\phi_m P}{P + P_0 + aB}. \quad (2)$$

This formulation assumes that the per capita rate of movement into the suppressor population increases with predator density. The maximum rate is given by ϕ_m , and the constant P_0 controls how quickly this maximum is reached with increasing predator density. If the parameter a is non zero, then the per capita rate of movement out of the breeding population is slower in larger breeding populations, because the predation risk to an individual is reduced by the dilution effect of being in a larger population. Indeed if a is large enough so that $aB \gg P_0$, then the per capita movement rate increases with the ratio of predators to breeding prey, rather than the absolute number.

For simplicity, we assume presently that the return movement, the per capita rate of movement from the

suppressor population to the breeding population, is a simple constant (α), so that the rate of change in the suppressor population is given by

$$\frac{dS}{dt} = \phi(P, B) B - \alpha S. \quad (3)$$

We assume that the fecundity of the breeding population is described by a simple logistic, so that

$$\frac{dB}{dt} = rB \left(1 - \frac{B}{K}\right) - \frac{I_m B P}{B + B_0} - \phi(P, B) B + \alpha S, \quad (4)$$

where r is the maximum population growth rate, and K is the carrying capacity.

3. RESULTS

Using standard methods (e.g. Nisbet & Gurney 1982) it is easy to show that this model has only one non-trivial steady state. This steady state is given by

$$B^* = \frac{\mu B_0}{\epsilon I_m - \mu}, \quad (5)$$

$$P^* = \left(\frac{\epsilon r B^*}{\mu}\right) \left(1 - \frac{B^*}{K}\right), \quad (6)$$

and

$$S^* = \frac{\phi(P^*, B^*) B^*}{\alpha}. \quad (7)$$

This steady state exists providing

$$K > \frac{\mu B_0}{\epsilon I_m - \mu}. \quad (8)$$

Consider first the limit where $\phi_m \rightarrow 0$, in this case $\phi(P, B) \rightarrow 0$ and there is no movement of individuals from the breeding to the suppressor population. As we would expect, the suppressor population steady state is zero, and the whole prey population exists in the breeding state. Now as ϕ_m is increased, we can see that B^* and P^* remain unchanged, but S^* increases in size. Thus the model predicts that as breeding suppression increases (ϕ_m increases) then the predator population size is unaffected but the prey population ($B^* + S^*$) increases, as does the fraction of the population in the suppressed state ($S^*/(B^* + S^*)$).

Using the Ruth-Hurwitz criteria (e.g. Nisbet & Gurney 1982), we can also (after considerable algebraic labour) show that the non-trivial steady state is locally stable providing

$$\begin{aligned} & \frac{I_m B_0 P^*}{(B_0 + B^*)^2} - r \left(1 - \frac{2B^*}{K}\right) + \alpha \\ & + \left(\frac{\phi_m P^*}{P^* + P_0 + aB^*}\right) \left(\frac{P^* + P_0}{P^* + P_0 + aB^*}\right) > 0. \end{aligned} \quad (9)$$

Consider again the simplifying case where there is no breeding suppression: i.e. no movement into or out of the suppressor state ($\alpha \rightarrow 0$, $\phi_m \rightarrow 0$). In this case, the last two terms in equation (9) tend to zero, and the simplified stability condition can be rearranged to give

$$K < B_0 \left(1 + \frac{2\mu}{\epsilon I_m - \mu}\right). \quad (10)$$

This demonstrates the classical paradox of enrichment shown by many simple resource-consumer models, where enriching the primary productivity of the system (increasing K) leads to destabilization (Rosenzweig 1971).

It is easy to see that allowing use of the suppression state (by increasing ϕ_m) acts to increase the size of the last term in equation (9), because P^* and B^* are unaffected by ϕ_m . This last term is always positive, so increasing the use of the suppression state by increasing ϕ_m is always stabilizing. In view of this it may seem paradoxical that decreasing α is also always destabilizing, because α is the inverse of the average length of time that individuals spend in a visit to the suppressor state. However, one can see that if individuals remained in the suppressor state for a long time then this effectively introduces a delay into the system, because the numbers suppressing breeding are closely related not to the current predation risk but to the predation risk at previous times. The introduction of such delayed regulation is a well-known agent of instability (May 1974). However, if individuals return quickly to the breeding state (perhaps only to return to suppression once again because predator numbers are still high), then the number of suppressors at any one time will be well correlated with current predation risk, which should lead to greater stability (see also below). Hence increasing α is always stabilizing. These effects are illustrated clearly in figure 1*a*. At very low values of ϕ_m , we see the paradox of enrichment effect. However, as ϕ_m is increased, the critical value of K required to produce instability increases. This effect is very strong and one can see that there is a threshold value of ϕ_m , beyond which no amount of enrichment will destabilize the system. Increasing α reduces the size of the area of instability. As increasing ϕ_m increases stability, we would expect that increasing the parameters P_0 or α , which both decrease the movement rate between breeding and suppressing states, would act to increase the area of instability. These effects are illustrated in figures 1*b* and *c*.

Figure 2 demonstrates the effect that breeding suppression has on predator-prey oscillations when the strength of breeding suppression is insufficient to stabilize the equilibrium. A common feature of predator-prey oscillations is that the predator population grows to such a level that it causes a crash in the prey population, and this is followed by a steady decline in the predator population. Eventually the prey recover and (in the absence of high predator population) grow rapidly. This increase in prey then causes a recovery in the predator population, and the cycle begins again (see figure 2*a*). However, with breeding suppression, the breeding population is able to recover more quickly from a crash, because it is bolstered by individuals returning from the suppressor population. Thus breeding suppression will cause an increase in cycle frequency and a decrease in the amplitude of the predator oscillations, because the predator population no longer has time to fall to such low values before the prey recover (compare figures 2*a*, *b*). The breeding population no longer climbs to such high values because it loses individuals that move

to the suppressor population. Although the breeding population still crashes to low values, the total prey population remains higher with breeding suppression because of the individuals protected in the suppressor state. Hence the amplitude of the prey oscillations also decreases. The stronger breeding suppression becomes, the more effect it has on cycle amplitude and frequency, until eventually sufficiently strong breeding suppression always leads to stability.

We have repeated the simulations in figure 2 for a wide range of different parameter values and have found the general conclusions given above to be maintained. We have also investigated the effect that various modifications to the model structure have on the effectiveness of breeding suppression as an agent of stability, and will now briefly summarize these results.

The original model assumed that there was no mortality among those suppressing breeding. If such mortality is added, then this works against the stabilizing effect of breeding suppression. The reason for this can be understood from the explanation of figure 2; the stronger this added mortality is, the less able the breeding population is to recover from population crashes. However, even when this mortality is very strong, breeding suppression still has some stabilizing effect, because the flow of individuals into this state reduces the size of breeding prey population peaks.

The original model also assumed that individual fitness in the breeding population depended only on the size of the breeding population and not on the size of the prey population as a whole. If we alter the model so that the logistic model for the growth of the breeding population is changed to

$$rB\left(1 - \frac{B+S}{K}\right), \quad (11)$$

then, although we lose analytic tractability, extensive numerical simulations show that the essential behaviour of the model is unaffected.

The original model also assumed that the per capita rate at which individuals return to breeding from suppression (α) is a simple constant, independent of the current predation risk. We modified the model, in order to make α a dynamically varying function. Two forms were investigated:

$$\alpha(P) = \alpha_0 \exp(-uP), \quad (12)$$

and

$$\alpha(B, P) = \alpha_0(\phi_m - \phi(B, P)), \quad (13)$$

where α_0 and u are constants. In both cases, making α dynamic rather than static increased the stability effect of breeding suppression. Again the reasons can be inferred from the explanation given of figure 2; making α dynamic allows the use of breeding suppression to be tailored to current predation risk, rather than predation risk in times past. In other words, a dynamic α causes individuals to return to breeding as soon as predator numbers drop, hence speeding up the breeding populations' recovery from a crash and so enhancing stability.

Figure 1. For legend see opposite.

Figure 2. The changes in population values over time for two integrations of equations (1)–(4). In (a) there is no breeding suppression ($I_m = 0$); in (b) there is breeding suppression but insufficient to stabilize the predator–prey oscillation ($I_m = 0.04$). $K = 5$ and all other parameter have the default values used in figure 1. Solid lines denote breeders; dotted lines denote predators; heavy line denotes suppressors.

4. DISCUSSION

One might intuitively expect that predator-induced breeding suppression (PIBS) would accentuate predator–prey cycling (e.g. Ylönen 1994; but see Oksanen & Lundberg 1995), but the opposite is more

likely to occur: PIBS tends to stabilize otherwise oscillatory predator–prey dynamics. Other models suggest that adaptive antipredator behaviour will generally act to stabilize predator–prey population dynamics (Ives & Dobson 1987; Ruxton 1995), but few models have actually addressed this general issue

Figure 1. The stability boundary of the model obtained by solving equation (9). Population dynamic parameter values are the same as used in simpler models by Abrams (1996) and Ruxton (1995): $\epsilon = 0.1$, $r = 1$, $B_0 = 1$, $I_m = 1$, $m = 1$. The parameters controlling breeding suppression are (unless otherwise stated): $\alpha = 0.01$, $a = 1.0$, $P_0 = 0.1$; (a) shows that increasing α decreases the area of parameter space where the model is unstable; (b) and (c) show that increasing a or P_0 increase the area of instability. However in all cases, one can always find a critical value of I_m , beyond which the system is always stable.

(Lima 1996), although Dehn (1994) provides a good example. The underlying reason for the stability-enhancing effects of PIBS lies in the assumption that 'suppressed' individuals are relatively invulnerable to predation. These individuals, in effect, buffer the population from extreme degrees of predation, and are ready to breed when predation drops to lower levels. Our results thus bolster the conclusion by Abrams & Walters (1996) that the presence of invulnerable prey can act to stabilize otherwise unstable systems. A key difference in approach, however, is that the extensive modelling of Abrams & Walters rests on the assumption that transitions to and from the invulnerable state are density independent, whereas we assume that the suppression state is used by prey topically, with transition rates being dependent on both the predator and prey densities. In any case, such effects can also be seen as a special case of the well known tendency for refuges to enhance stability in predator-prey systems (McNair 1986; Sih 1987; Ruxton 1995).

When the level of PIBS is inadequate to stabilize a predator-prey system, its general effect on predator-prey cycles is to lower their amplitude and increase their frequency, relative to a situation lacking PIBS. As mentioned earlier, Ylönen (1994), Hik (1995) and others suggest that PIBS may hasten the crash phase in a prey population cycle. Our results suggest that such an effect may well occur. However, PIBS also acts to reduce both the degree of population decline and the time required for the prey population to re-establish positive growth. This latter effect runs counter to Hik's (1995) suggestion that PIBS in snowshoe hare would also act to deepen the decline and lengthen the recovery phase of the prey cycle. We cannot, however, dismiss Hik's suggestion as unlikely, because he also notes that PIBS in snowshoe hare may be relatively long-lasting (perhaps exceeding one or two years). As seen in the previous section, increasing the length of time in a suppressed state (i.e. decreasing α) acts to reduce the strength of the stabilization caused by PIBS.

Two significant gaps in our present understanding of PIBS presently limit the precision of our analysis. First, there is very little information on the temporal extent of breeding suppression once such a state is entered. Recent physiological work on *Clethrionomys* voles (Koskela *et al.* 1996) suggests that PIBS may be difficult to reverse in the very short term. As mentioned above, Hik (1995) suggests that PIBS in snowshoe hare may be a relatively protracted affair. There also appears to be no information at all on the degree of flexibility one might expect in a suppressed animal's ability to re-enter the breeding population. As the lengths of the periods in a suppressed breeding state are predicted to have a strong influence on the stabilizing effect of PIBS, the lack of information on the temporal dynamics of this phenomenon is an important problem.

Second, the degree to which breeding suppression actually decreases predation is largely unknown. There is reason to believe that breeding generally exposes females to greater predation in general (Lima & Dill 1990; Magnhagen 1991) and in rodents in particular (Cushing 1985; Ylönen 1994), but the precise survival benefit of breeding suppression is unknown (see also

Lambin *et al.* 1995). If PIBS detracts only slightly from mortality, then its stabilizing effects on predator-prey dynamics will be minimal.

In closing, we note that the idea of breeding suppression also appears in many 'intrinsic' theories of predator-prey cycling (see Boonstra (1994) for a review). Rather than being induced by predators, this breeding suppression is induced by stresses associated with high population density. Simulation modelling suggests that this form of breeding suppression promotes population cycling, in that it provides a form of delayed density dependence that encourages cycling (see Lomnicki 1995). In this sense, PIBS and stress-induced breeding suppression may differ qualitatively in their effects on population cycling. In any case, many assumptions regarding the role of breeding suppression in these theories have yet to find solid empirical support, including the assumption that stress-induced breeding suppression leads to enhanced survival (e.g. Lomnicki 1995). Perhaps there is presently only one point of consensus in the study of population cycling in small mammals: that the degree to which intrinsic versus extrinsic factors influence population cycling remains one of the major unresolved issues in population ecology.

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REFERENCES

- Abrams, P. A. & Walters, C. J. 1996 Invulnerable prey and the paradox of enrichment. *Ecology* **77**, 1125–1133.
- Boonstra, R. 1994 Population cycles in microtines: the senescence hypothesis. *Evol. Ecol.* **8**, 196–219.
- Cushing, B. S. 1985 Estrous mice and vulnerability to weasel predation. *Ecology* **66**, 1976–1978.
- Dehn, M. M. 1994 Optimal reproduction under predation risk: consequences for life history and population dynamics in microtine rodents. Ph.D. thesis, Simon Fraser University, Canada.
- Hanski, I., Turchin, P., Korpimäki, E. & Henttonen, H. 1993 Population oscillations of boreal rodents: regulation by mustelid predators leads to chaos. *Nature, Lond.* **364**, 232–235.
- Heikkilä, J., Kaarsalo, K., Mustonen, O. & Pekkari, P. 1993 Influence of predation risk on early development and maturation in three species of *Clethrionomys* voles. *Ann. Zool. Fennici* **30**, 153–161.
- Hik, D. S. 1995 Does risk of predation influence population dynamics? Evidence from the cyclic decline of snowshoe hares. *Wildl. Res.* **22**, 115–129.
- Ives, A. R. & Dobson, A. P. 1987 Antipredator behaviour and the population dynamics of simple predator-prey systems. *Am. Nat.* **130**, 431–447.
- Kokko, H. & Ranta, E. 1996 Evolutionary optimality of delayed breeding in voles. *Oikos* **77**, 173–175.
- Korpimäki, E. 1993 Regulation of multiannual vole cycles by density-dependent avian and mammalian predation? *Oikos* **66**, 359–363.
- Korpimäki, E., Norrdahl, K. & Valkama, J. 1994 Reproductive investment under fluctuating predation risk: microtine rodents and small mustelids. *Evol. Ecol.* **8**, 357–368.
- Koskela, E., Horne, T. J., Mappes, T. & Ylönen, H. 1996 Does risk of small mustelid predation affect the oestrous

- cycle in the bank vole, *Clethrionomys glareolus*? *Anim. Behav.* **51**, 1159–1163.
- Koskela, E. & Ylönen, H. 1995 Suppressed breeding in the field vole (*Microtus agrestis*): an adaptation to cyclically fluctuating predation risk. *Behav. Ecol.* **6**, 311–315.
- Krebs, C. J. & Myers, J. H. 1974 Population cycles in small mammals. *Adv. Ecol. Res.* **8**, 267–399.
- Krebs, C. J., Boutin, S., Boonstra, R. *et al.* 1995 Impact of food and predation on the snowshoe hare cycle. *Science, Wash.* **269**, 1112–1115.
- Lambin, X., Ims, R. A., Steen, H. & Yoccoz, N. G. 1995 Vole cycles. *Trends Ecol. Evol.* **5**, 204.
- Lima, S. L. 1997 Stress and decision-making under the risk of predation: recent developments from behavioral, reproductive, and ecological perspectives. *Adv. Study Behav.* **27**. (In the press.)
- Lima, S. L. & Dill, L. M. 1990 Behavioral decisions made under the risk of predation: a review and prospectus. *Can. J. Zool.* **68**, 619–640.
- Lomnicki, A. 1995 Why do populations of small rodents cycle? A new hypothesis with numerical model. *Evol. Ecol.* **9**, 64–81.
- Magnhagen, C. 1991 Predation risk as a cost of reproduction. *Trends Ecol. Evol.* **6**, 183–186.
- May, R. M. 1974 *Stability and complexity in model ecosystems*. Princeton University Press.
- McNair, J. N. 1986 The effect of refuges on predator–prey interactions: a reconsideration. *Theor. Popul. Biol.* **29**, 38–63.
- Nisbet, R. M. & Gurney, W. S. C. 1984 *Modelling fluctuating populations*. Chichester: Wiley.
- Norrdahl, K. & Korpimäki, E. 1995 Mortality factors in a cyclic vole population. *Proc. R. Soc. Lond. B* **261**, 49–53.
- Oksanen, L. & Lundberg, P. 1995 Optimization of reproductive effort and foraging time in mammals: the influence of resource level and predation risk. *Evol. Ecol.* **9**, 45–56.
- Ostfeld, R. S., Canham, C. D. & Pugh, S. R. 1993 Intrinsic density-dependent regulation of vole populations. *Nature, Lond.* **366**, 259–261.
- Ronkainen, H. & Ylönen, H. 1994 Behaviour of cyclic bank voles under risk of mustelid predation: do females avoid copulations? *Oecologia* **97**, 377–381.
- Rosenzweig, M. L. 1971 The paradox of enrichment: destabilization of exploitation ecosystems in ecological time. *Science, Wash.* **171**, 385–387.
- Ruxton, G. D. 1995 Short term refuge use and stability of predator–prey models. *Theor. Popul. Biol.* **47**, 1–17.
- Sih, A. 1987 Prey refuges and predator–prey stability. *Theor. Popul. Biol.* **31**, 1–12.
- Taitt, M. J. & Krebs, C. J. 1985 Population dynamics and cycles. In *Biology of New World Microtus* (ed. R. H. Tamarin), pp. 567–620. Special Publication No. 8, American Society of Mammalogists.
- Ylönen, H. 1989 Weasels, *Mustela nivalis*, suppress reproduction in the cyclic bank voles, *Clethrionomys glareolus*. *Oikos* **55**, 138–140.
- Ylönen, H. 1994 Vole cycles and antipredatory behaviour. *Trends Ecol. Evol.* **9**, 426–430.
- Ylönen, H. & Ronkainen, H. 1994 Breeding suppression in the bank vole as antipredatory adaptation in a predictable environment. *Evol. Ecol.* **8**, 658–666.
- Ylönen, H., Jedrzejewska, B., Jedrzejewski, W. & Heikkilä, J. 1992 Antipredatory behaviour of *Clethrionomys* voles: ‘David and Goliath’ arms race. *Ann. Zool. Fennici* **29**, 207–216.

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