Risk of parasite-induced predation: an experimental field study on Townsend's voles (*Microtus townsendii*)

Harald Steen, Mary Taitt, and Charles J. Krebs

Abstract: Studies of causes of mortality in rodents, snowshoe hares, and red grouse show that many individuals succumb to predation and few die from starvation or parasite infestation. Predation may be the proximate cause of death and factors like parasite infestation and starvation could facilitate predation. Our aim was to test whether Townsend's voles, *Microtus townsendii*, experimentally rid of parasites are less likely to be killed by predators than are control voles. During two breeding seasons we treated half of the adults caught at Boundary Bay, British Columbia, Canada, with the anthelmintic Ivomec® and left the other half as controls. Voles were randomly assigned to the treatment and control groups. Predators killed 5 of 23 treated voles and 11 of 26 control voles in 1998. In 1997 predators killed 3 of 17 treated voles and 2 of 18 control voles. Vole density was higher in 1998 than in 1997 and predation was the main cause of mortality in both years. Survival of control voles was lower in 1998 than in 1997, indicating lower predation pressure in 1997 than in 1998. Botfly prevalence and intensity were low and similar in both years. We speculate that the lack of treatment effect in 1997 could have been due to lower overall parasite prevalence in that year than in 1998, resulting in relatively higher susceptibility of voles to predation. Parasite-induced predation reduced monthly survival by 17% in 1998.

Résumé : Les études sur la mortalité des rongeurs, des lièvres d'Amérique et des lagopèdes d'Écosse démontrent que beaucoup d'animaux sont victimes de la prédation, mais que peu meurent d'inanition ou à la suite d'une infestation de parasites. La prédation est la cause immédiate de la mortalité, mais des facteurs tels que les parasites ou la privation de nourriture pourraient faciliter la prédation. Nous avons tenté de vérifier si les campagnols de Townsend, *Microtus townsendii*, débarrassés de leurs parasites expérimentalement, sont moins vulnérables aux prédateurs que des campagnols témoins. Nous avons traité à l'Ivomec®, un helminthique, la moitié des campagnols adultes capturés à Boundary Bay, Colombie- Britannique, Canada, et utilisé l'autre moitié comme témoins. Dans les deux groupes, les campagnols ont été choisis au hasard. Les prédateurs ont tué 5 des 23 campagnols traités et 11 des 26 campagnols témoins en 1998. En 1997, 3 des 17 campagnols traités et 2 des 18 campagnols témoins ont été victimes des prédateurs. La population de campagnols a été plus dense en 1998 qu'en 1997 et la prédation a été la principale cause de mortalité les deux années. La survie des campagnols témoins a été moins élevée en 1998 qu'en 1997, ce qui signifie que la pression de prédation a été moins forte en 1997. La prévalence et la gravité des infections d'oestres, ont été faibles et semblables les 2 années. Nous croyons que le traitement administré en 1997 a été inefficace parce que la prévalence des parasites cette année-là a été faible comparativement à celle de 1998, ce qui a entraîné une augmentation relative de la vulnérabilité des campagnols à la prédation. La prédation facilitée par le parasitisme a réduit de 17 % la survie mensuelle en 1998.

[Traduit par la Rédaction]

Introduction

Predation is the immediate cause of death in many rodents, snowshoe hares (*Lepus americanus*), and red grouse (*Lagopus lagopus scoticus*) (rodents, Boonstra et al. 1990; Mihok et al. 1988; Norrdahl and Korpimäki 1995; Reid et al. 1995; Steen 1994, 1995; Steen et al. 1997; snowshoe hares, Keith et al. 1993; Murray et al. 1997; grouse, Hudson et al. 1992; Smith and Willebrand 1999; Thirgood et al. 2000). If predators were the proximate and not the ultimate cause of death, one might erroneously discard all hypotheses regarding population regulation that do not explicitly incorporate predation (i.e., based on food quality and availability, e.g., Haukioja et al. 1983; Laine and Henttonen 1983; genetics, Chitty 1967; parasites, Elton 1924; social behaviour, e.g., Charnov and Finerty 1980; Lambin and Krebs 1991) and conclude that populations are regulated by predation. Other factors, like starvation and parasitism, may act on populations through increased susceptibility to predation and not cause mortality per se (Murray et al. 1997). Since predation is the immediate cause of death, a test of a hypothesis that does not incorporate predation must use the probability of being killed by a predator as the response variable, rather than the

Received 15 November 2001. Accepted 25 June 2002. Published on the NRC Research Press Web site at http://cjz.nrc.ca on 23 August 2002.

H. Steen[1,2] and C.J. Krebs. Department of Zoology, The University of British Columbia, 6270 University Boulevard, Vancouver, BC V6T 1Z4, Canada.
M. Taitt. B.C. Open University, Mathissi Place, Burnaby, BC V5G 458, Canada.

[1]Corresponding author (e-mail: harald.steen@bio.uio.no).
[2]Present address: Division of Zoology, Department of Biology, University of Oslo, PB 1050 Blindern, 0316 Oslo, Norway.

 DOI: 10.1139/Z02-115

Table 1. Detailed treatment record for the 2 years.

	Number of days between treatments			Number of treatments		
Year	Median	25–75% quartiles	Min.–max.	Median	25–75% quartiles	Min.–max.
1997	8.25	7–11.7	6.7–35	3	2–4.8	1–7
1998	11.5	8.5–17	6–31	2	2–3	2–6

Note: We use median values, since a mean will obviously be affected by extreme values.

less precise change in population density. If any of the above hypotheses is correct, regulation of small-rodent populations may depend on interactions between processes on different trophic levels rather than on a single trophic level, as with snowshoe hares (Krebs et al. 1995). Our aim is to test if voles experimentally rid of parasites have a lower probability of being killed by predators than control voles do.

Townsend's voles (*Microtus townsendii*) at Boundary Bay south of Vancouver, British Columbia, Canada, show annual fluctuating population dynamics with variable amplitude. The area is dominated by tall grass with scattered bushes. There are birds of prey and mustelids in the area. The larvae of a cuterebrid dipteran, the botfly (*Cuterebra* sp.), also infects the voles. Peak prevalence of botfly larvae occurs in late August. Botflies lay their eggs on vegetation and these hatch when they come in contact with a heated object like an animal's skin (McAlpine et al. 1987). The larvae enter the body through any opening, grow to 2 cm in length and 1 cm in width, and end up under the skin, often around the testicles and anus. When they mature they emerge through an air hole and pupate on the ground. Multiple infections of botfly larvae are common in Townsend's voles. In an observational study on Townsend's voles, Boonstra et al. (1980) found that individuals infected with botflies (*Cuterebra grisea*) had lower survival and lower reproductive rates than uninfected voles. They also concluded that botflies had a detrimental effect on population growth. The methods available at the time did not allow for examination of the cause of death and Boonstra et al. (1980) assumed that animals which disappeared had died. Since those authors could not separate migration and death caused by parasite-induced predation, their results are uninformative regarding the immediate cause of death in these voles.

We tested the hypothesis that parasite-infected voles are more prone to predation than voles treated with Ivomec® (Merck AgVet), an anthelmintic commonly used to rid animals of murine mites (Baumans et al. 1988) and botflies (Ostlind et al. 1979), as well as nematodes (Murray et al. 1996). If parasites increase the probability of Townsend's voles being killed by predators, we predict higher survival rates in Ivomec-treated voles than in control voles, and we expect to see lower prevalence and intensity of botfly larva in treated than in control voles.

Methods

Trapping and treatment

Our study was conducted in semi-natural grassland at Boundary Bay (49°04′N, 123°00′W) in 1997 and 1998. We trapped Townsend's voles during the expected peak of botfly larva prevalence from 18 July to 8 October in 1997 and from 7 July to 3 September in 1998. In spring 1997 we established a grid with three rows of 23 Longworth traps spaced 15 m apart to maximize the number of animals caught. To increase the number of animals a further 20 traps were added in 1998 at a site about 300 m east of the original grid. Adding 7.5 m to the edge of the perimeter of the trapping grids gave trapped areas of 1.62 and 2.1 ha in 1997 and 1998, respectively. In both years we trapped following a robust design (Pollock et al. 1990), with primary sessions once a week that consisted of three secondary trapping sessions. In each primary trapping session the traps were set at dusk the first day, checked the following morning before sunrise and the following afternoon, and closed the next morning before sunrise. If a hot day was expected, we closed the traps during the day.

Voles were ear-tagged and sexed upon first capture. Tag numbers were randomly assigned to individuals. Voles with an even-numbered tag were assigned to the treatment group and received one drop of Ivomec solution (Injection 10 mg ivermectin/mL, Merck AgVet) behind the ear (Baumans et al. 1988; Ostlind et al. 1979). Throughout the study Ivomec treatment was applied upon recapture, and to avoid Ivomec poisoning we treated the voles maximally once a week (Ostlind et al. 1979) (see Table 1 for details). Animals were handled in accordance with the principles and guidelines of the Canadian Council on Animal Care and were released immediately after treatment.

Voles weighing more than 30 g at first capture were fitted with a 3.5-g SS-2 transmitter with a mortality switch (Biotrack, Wareham, Dorset, U.K.). To find voles as soon as possible after death, we checked twice a week whether any of the transmitters was emitting a slow pulse, indicating that the animal was dead. When a vole's transmitter emitted a slow pulse, or "dead" signal, the animal was located. The location of the transmitter and whether it was lying on grass, by a raptor perch, or in a burrow or runway were recorded. The remains of the voles were brought to the laboratory to establish the cause of death (Steen 1994).

Determining the cause of death

When the mortality collar was found without a carcass and lying open on a mound 50–100 m from the location of the last fix (trapping area), we assumed that predation by a bird was the cause of death. If the mortality collar was found under cover or below ground with bite marks or blood on it, mustelid predation was assumed. Part or whole carcasses with bite marks and subcutaneous hematoma were also taken as evidence of mammal predation (Steen 1994, 1995; Steen et al. 1997). In cases where we were not certain of the cause of death we designated it "unknown". The intensive live-trapping revealed that only one vole lost its transmitter.

Survival modelling

Hypotheses concerning the relationship between intensity of parasite infection and mortality due to predation were tested using radio-collared voles only, and survival was estimated from the weekly trapping sessions. As we knew whether an animal was dead or alive at a trapping session, we used known-fate analysis implemented in the program MARK (White and Burnham 1999). The known-fate model implemented in MARK is a Cormack–Jolly–Seber (CJS) survival model in which the recapture rate is set to 1, and the statistical power is increased to detect differences by reducing the number of estimated parameters. It differs from a Kaplan–Meier survival estimator (Kaplan and Meier 1958) in that it estimates whether an animal survived the period between two trapping sessions and not its survival until the day it died. CJS models allow staggered entry and censoring of individuals that left the study population for reasons outside the scope of the study.

In the full model, survival varies independently through time in the treated and control animals and sexes. Reduced models are constrained by setting survival constant through time and may be different or equal between the treatment groups or sexes. To choose between models, we used Aikaike's Information Criterion (AIC_c) (Anderson and Burnham 1994), and the 2 years were treated separately because pooling the two datasets would be wrong, since this assumes that grass growth, breeding of raptors and other predators, and population dynamics of botflies and other parasites were at the same stage on the same date in the 2 years. Judging from botfly prevalence this was obviously not the case (Fig. 1).

Density estimation

Vole density was estimated using closed-population models. Since trapping followed a robust design, we were able to utilize the probability of being recaptured for the first time and recaptured within the 2 days (Huggins 1989, 1991; White and Burnham 1999). The most parsimonious model was chosen using AIC_c (Anderson and Burnham 1994). As known-fate analysis provides a more reliable estimate of survival rates and yields more statistical power for testing hypotheses, we used only the robust-design-based models to estimate density.

Results

Densities

The model that best fitted the 1997 data had constant and identical weekly survival rates for control and treated voles. Survival estimated by the best model for the 1998 data showed that weekly survival was higher in treated than in control voles. Weekly survival was constant throughout summer 1998 for both groups. In both years the probabilities of capture and resighting were the same for treated and control voles but time-dependent within and between primary and secondary trapping sessions. The best model for the 1998 data showed that the survival rates of both the treated and the control voles differed from those in 1997, but the probability of capture and resighting was the same. In 1997, Townsend's vole densities were between 15 and 19/ha during July and August (Table 2) and reached a maximum of 30/ha in late September (Table 2). In 1998, vole densities during July and August were between 15 and 31/ha (Table 2).

Prevalence

The prevalence of botfly larvae was low in both years, with a maximum of 18 and 12% infected in 1997 and 1998, respectively (Table 2). No vole had more than one botfly larva at any given time. We encountered no infections of the grey flesh fly (*Wohlfahrtia vigil*).

Predation rates

The field season was 1 month longer in 1997 than in 1998, and to compare predation rates between the years we pooled treated and control voles for each year and used only voles that died before 3 September in both years. Predators killed 5 of 35 voles in 1997 and 16 of 49 voles in 1998 (Table 3). Predation pressure, defined as the rate of predation on untreated (control) voles, was significantly higher in 1998 than in 1997 (2 of 18 voles in 1997; 11 of 26 voles in 1998; $p < 0.05$). Predators killed 9 of 44 voles during the whole 1997 season.

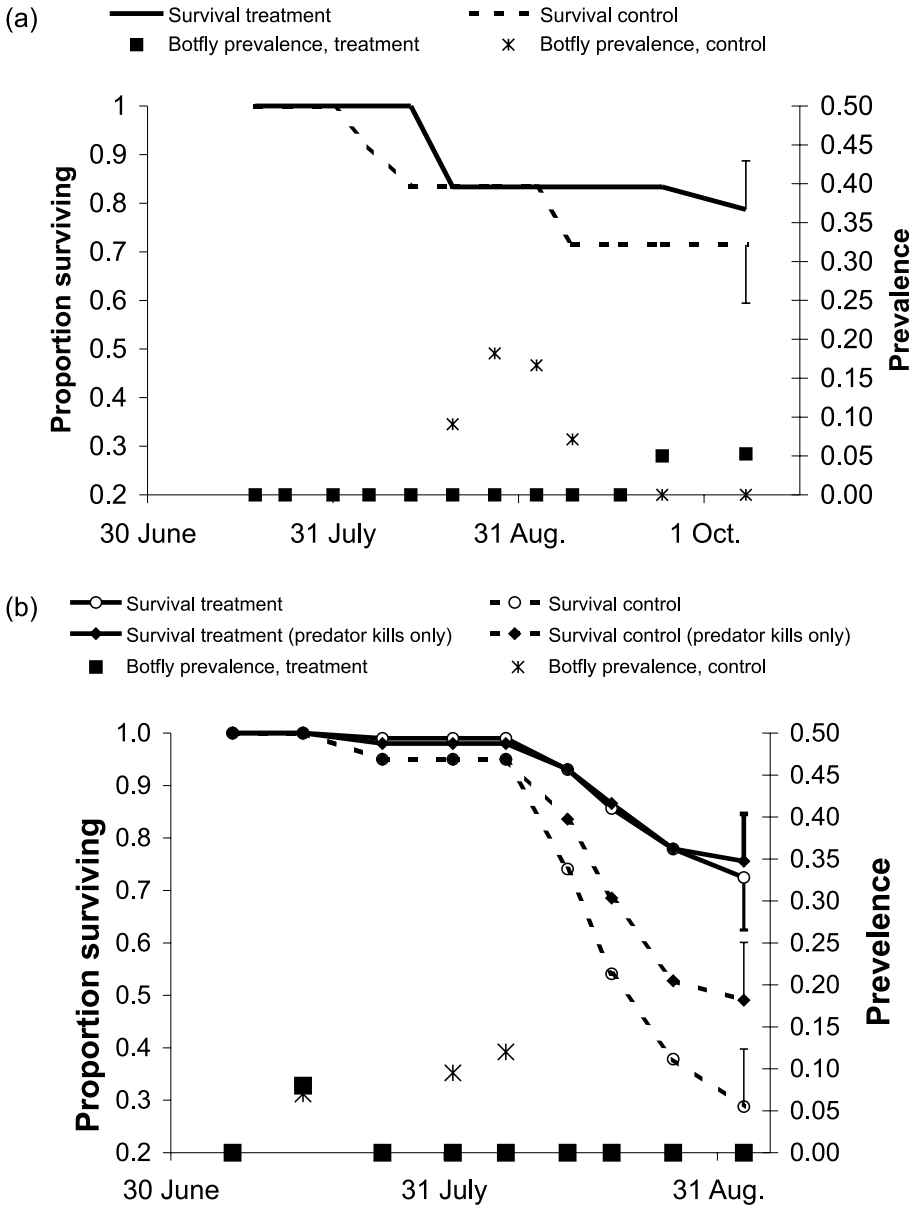
Survival

In 1997 we treated 26 voles and left 18 as controls. Predators killed 4 treated and 5 control voles, while 2 control voles and 1 treated vole died of unknown causes. The prediction that Ivomec-treated voles would be less susceptible to predation was tested using predator-killed voles only. Those that died of unknown causes or from human interference or disappeared were censored. The model with the lowest AIC_c estimated mean survival from 18 July to 8 October to be 0.73 (SE = 0.08; 92 days), with no difference between treated and control voles. To allow comparison of survival rates between the 2 years, cumulative survival rates are presented in Fig. 1. Since the cause of death was not known for only one animal, we did not test for differences in survival rate between treated and control animals when we did not censor animals that died of unknown causes.

In 1998 there were 23 treated and 26 control voles. Of these, predators killed 5 treated and 11 control voles, while 1 and 7 voles, respectively, died of unknown causes (Table 3). As with the 1997 data we used only predator-killed voles to test the hypothesis that parasites increase vulnerability to predation. Voles that died of unknown causes or from human interference or disappeared were censored. The mean probability of survival between 7 July and 3 September (58 days) was 0.50 (SE = 0.11) for control voles and 0.77 (SE = 0.09) for treated voles. Patterns of survival of treated and control voles were parallel through time, and this model was almost significantly better than a model with identical but time-dependent survival (log-likelihood ratio (LR) test, $p < 0.07$; Fig. 1). The estimated survival rate from 7 July to 3 September (58 days) including voles that died of natural causes was 0.72 (SE = 0.1) for treated voles and 0.28 (SE = 0.09) for control voles, and survival patterns were parallel through time. The data fitted the model significantly better than the next best model, in which there was no difference between treated and control voles (LR test, $p < 0.003$; Fig. 1).

We trapped few juveniles in either year. The large trap spacing could allow a female to give birth and raise her young without any young entering the traps. Hence, we will

Fig. 1. Cumulative survivorship curves for treated (solid line) and control (broken line) Townsend's voles (*Microtus townsendii*) in 1997 (*a*) and 1998 (*b*). Standard errors of estimated survival probability throughout the summer period are given as error bars for the last trapping session. Since our hypothesis predicts lower survival rates for the control voles than for the Ivomec-treated voles, and few voles died of unknown causes in 1997, we present survival curves based on predator-killed voles only. In 1998, quite large number of voles died of unknown causes and we have chosen to present a survival analysis that includes all deaths (○) in addition to survival based on predator kills only (◆). The prevalence of botfly larvae for the control group (*) and treated group (■) is shown.



not speculate on the effect of treatment on breeding success. There were no differences in body mass, or any difference in the proportion of reproductive females, between treated and control voles in either year.

Discussion

The probability of being killed by predators was lower (although not significantly) for Townsend's voles treated with the anthelmintic Ivomec than for control voles in 1998 but not in 1997. Although the Ivomec treatment was efficient in keeping the voles free of botfly larvae, the prevalence of botfly larvae (Table 2) was not high enough to allow any rigorous statistical analysis of their effect on survival.

The lack of a detectable effect of the treatment in 1997 may have been due to lower predator abundance or low parasite prevalence. Since predators killed 14 and 33% of the voles between mid-July and the beginning of September in 1997 and 1998, respectively, it is possible that the lack of a detectable treatment effect in 1997 was due to a low abundance of predators. Botfly prevalence was 18% in 1997 and 12% in 1998, and so cannot explain the lack of a treatment effect in 1997. Despite the low predation rates in 1997 we should have seen a similar trend in numbers killed by predators to 1998, but the observed trend was the opposite (Table 3). It

Table 2. Total numbers of individuals trapped during the 2 days, according to sex and treatment individuals.

	1997											1998								
	18 July	23 July	31 July	6 Aug.	13 Aug.	20 Aug.	27 Aug.	3 Sept.	9 Sept.	17 Sept.	24 Sept.	8 Oct.	7 July	15 July	24 July	1 Aug.	7 Aug.	19 Aug.	26 Aug.	3 Sept.
Treated group																				
Females	7	8	9	11	12	11	10	9	9	11	10 (1)	10 (1)	7	9	10	12	12	11	10	10
Males	0	1	1	2	4	4	4	6	8	9	9	9	4	4 (1)	6	9	10	9	9	6
Subadults	4	3	0	0	0	0	0	0	1	4	3	0	0	3	0	8	0	1	0	0
Control group																				
Females	5	7	6	5	4	4 (1)	4 (1)	4 (1)	6 (1)	6	6	6	3	5 (1)	8	10	12 (1)	10	7	4
Males	0	0	0	1	3	4	5 (1)	6 (1)	4	4	4	4	4	8	8	11 (2)	13 (2)	10	7	4
Subadults	2	5	2	0	1	0	0	0	0	4	8	14	2	1	1	5	6	4	5	7
Estimated no.*	25	27	24	30	25	26	25	28	35	44	48	47	48	31	35	66	64	52	44	35
–95% CI	20	24	18	19	24	23	23	25	29	38	40	43	20	30	33	59	56	45	38	32
+95% CI	47	48	69	76	48	50	44	53	55	70	73	58	104	34	39	74	72	60	54	39
Vole density (no./ha)	15	17	15	19	15	16	15	17	22	27	30	29	23	15	17	31	30	25	21	17

Note: All adult females and males carried a transmitter. The date indicates the first day of each primary trapping session E. Numbers in parentheses show the number of individuals with visible botfly larvae.
*Estimated using closed-capture models.

is therefore possible that the effect of the treatment in 1998 was due to the removal by the Ivomec of some other parasite(s) that were not present in 1997. The between-year variation in rates of parasite-induced predation shows that while parasites are not the only factor responsible for the variation in vole survival, they can increase predation by 26% during July and August. If the mortality due to predation was evenly distributed over the summer, then parasites increased predation, on average, by 17% per month during the breeding season

Botfly prevalence at the study site averaged 15% between 1971 and 1976 (no yearly breakdown was reported; Boonstra et al. 1980) and ranged from 49 to 96% between 1988 and 1991 (X. Lambin, personal communication). Vole densities in our study ranged from 15 to 30/ha in 1997 and from 15 to 31/ha in 1998, while densities were between 100 and 400/ha between 1989 and 1991 (Lambin 1994). Our study was therefore done on a low-density population, which may explain the low botfly prevalence.

A parasite-infected vole carrying a transmitter might have relatively higher probability of being killed by a predator than a parasite-infected vole without a transmitter. Transferring this result to voles not carrying a transmitter would clearly exaggerate the difference between treated and non-treated voles. Dividing a group of animals without a transmitter into treatment and control groups could control for this. Unfortunately, densities were too low to allow the use of such a experimental protocol. In case of such an interaction, our results would only be valid for transmitter-carrying voles, and the generality of our findings is dependent on the magnitude of the interaction. Carrying a transmitter per se does not seem to increase mortality in voles (Johannesen et al. 1997; Steen 1995).

The variable effect of parasites on survival observed in the present study is expected on the basis of other studies. There are two studies in which the authors treated animals in their natural environment with an anthelmintic and monitored their probability of being killed by a predator. Incubating red grouse hens that were treated with an anthelmintic had a lower probability of being located by hunting dogs, which suggests that parasites increase the probability of being killed by predators (Hudson et al. 1992). Ivomec-treated snowshoe hares had a lower probability of being killed by predators relative to control hares in one year but not in another (Murray et al. 1997). In addition, Murray et al. (1997) showed experimentally that there is an interaction between food availability and the effect of a high parasite load on survival. The interaction between food and parasites is probably not very important in our system, since the monthly survival rate in our study was similar to the survival rates from a study where densities were 3–13 times higher. The monthly survival rate of voles at Boundary Bay during spring–summer in 1990 and 1991 was 0.52, with densities between 100 and 400/ha (Lambin 1994; Lambin and Yoccoz 1998), while during the present study, vole densities were between 15 and 30/ha, monthly survival rates of untreated voles being 0.88 and 0.51 in 1997 and 1998, respectively.

During Lambin's (1994) study, botfly prevalence was about 3–6 times higher and vole densities about 3–13 times higher than in our study. During the present study, vole densities and botfly prevalence were comparable between years. Despite

Table 3. Numbers of Townsend's voles (*Microtus townsendii*) fitted with a radio collar and the numbers that died and causes of death in the treatment and control categories between 18 July and 8 October in 1997 and between 7 July and 3 September in 1998.

	Total number of voles		Number killed by predators		Number dead from unknown causes	
	Females	Males	Females	Males	Females	Males
1997						
Treated	11 (3)	6 (3)	3 (0)	0 (1)	0	0 (1)
Control	12 (3)	6 (0)	2 (1)	0 (2)	1	0 (1)
1998						
Treated	12	11	2	3	0	1
Control	12	14	4	7	3	4

Note: Since we continued trapping until 8 October in 1997, we give the number of new individuals, with the number killed between 3 September and 8 October in parentheses.

higher vole densities and higher botfly prevalence during 1990–1991, survival of voles was almost identical with that in 1998 and lower than in 1997. The interaction between parasites and predators obviously has a variable effect on the population dynamics of voles, and is probably not capable of regulating their population dynamics or causing regular population fluctuations.

Acknowledgements

The project was supported by a postdoctoral scholarship to Harald Steen from The Research Council of Norway and by research funds from the Natural Sciences and Engineering Research Council of Canada. We thank X. Lambin for providing his botfly data and S. Davidson, R. Goldsmith, N. Yoccoz, X. Lambin, and two anonymous reviewers for their helpful comments.

References

Anderson, D.R., and Burnham, K.P. 1994. AIC model selection in overdispersed capture–recapture data. Ecology, **75**: 1780–1793.

Baumans, N., Havenaar, R., van Herck, H., and Rooymans, T.P. 1988. The effectiveness of Ivomec and Neguvon in the control of murine mites. Lab. Anim. **22**: 243–245.

Boonstra, R., Krebs, C.J., and Beacham, T.D. 1980. Impact of botfly parasitism on *Microtus townsendii* populations. Can. J. Zool. **58**: 1683–1692.

Boonstra, R., Krebs, C.J., and Kanter, M. 1990. Arctic ground squirrel predation on collared lemmings. Can. J. Zool., **68**: 757–760.

Charnov, E.L., and Finerty, J.P. 1980. Vole population cycles: a case for kin-selection. Oecologia, **45**: 1–2.

Chitty, D. 1967. The natural selection of self-regulatory behavior in populations. Proc. Ecol. Soc. Aust. **2**: 51–78.

Elton, C.S. 1924. Periodic fluctuations in the number of animals: their causes and effects. Br. J. Exp. Ecol. **2**: 119–163.

Haukioja, E., Kapiainen, K., Niemalä, P., and Tuomi, J. 1983. Plant availability hypothesis and other explanations of herbivore cycles: complementary or exclusive alternatives. Oikos, **40**: 419–432.

Hudson, P.J., Dobson, A.P., and Newborn, D. 1992. Do parasites make prey vulnerable to predation? Red grouse and parasites. J. Anim. Ecol. **61**: 681–692.

Huggins, R.M. 1989. On the statistical analysis of capture–recapture experiments. Biometrika, **76**: 133–140.

Huggins, R.M. 1991. Some practical aspects of a conditional likelihood approach to capture experiments. Biometrics, **47**: 725–732.

Johannesen, E., Andreassen, H., and Steen, H. 1997. Effect of radiocollars on survival of root voles. J. Mammal. **78**: 638–624.

Kaplan, E.L., and Meier, P. 1958. Nonparametric estimation from incomplete observations. J. Am. Stat. Assoc. **53**: 457–481.

Keith, L.B., Bloomer, S.E.M., and Willebrandt, T. 1993. Dynamics of snowshoe hare population in fragmented habitat. Can. J. Zool. **71**: 1385–1392.

Krebs, C.J., Boutin, S., Boonstra, R., Sinclair, A.R.E., Smith, J.N.M., Dale, M., and Turkington, R. 1995. Impact of food and predation on the snowshoe hare cycle. Science (Washington, D.C.), **269**: 1112–1115.

Laine, K., and Henttonen, H. 1983. The role of plant production in microtine cycles in northern Fennoscandia. Oikos, **40**: 407–418.

Lambin, X. 1994. Sex ratio variation in relation to female philopatry in Townsend's voles. J. Anim. Ecol. **63**: 945–953.

Lambin, X., and Krebs, C.J. 1991. Can changes in female relatedness influence microtine population dynamics. Oikos, **61**: 126–132.

Lambin, X., and Yoccoz, N.G. 1998. The impact of population kin-structure on nestling survival in Townsend's voles, *Microtus townsendii*. J. Anim. Ecol. **67**: 1–16.

McAlpine, J.F., Peterson, B.V., Shewell, G.E., Teskey, H.J., Vockeroth, J.R., and Wood, D.M. 1987. Oestridae. *In* Manual of Nearctic Diptera. *Edited by* J.F. McAlpine. Biosystematics Research Centre, Ottawa, Ont. pp. 1147–1157.

Mihok, S., Lawton, T., and Schwartz, B. 1988. Fates and movements of meadow voles (*Microtus pennsylvanicus*) following a population decline. Can. J. Zool. **66**: 323–328.

Murray, D.L., Keith, L.B., and Cary, J.R. 1996. The efficacy of antihelmintic treatment on the parasite abundance of free-ranging snowshoe hares. Can. J. Zool. **74**: 1604–1611.

Murray, D.L., Cary, J.R., and Keith, L.B. 1997. Interactive effects of sublethal nematodes on nutritional status on snowshoe hare vulnerability to predation. J. Anim. Ecol. **66**: 250–264.

Norrdahl, K., and Korpimäki, E. 1995. Mortality factors in a cyclic vole population. Proc. R. Soc. Lond. B Biol. Sci. **261**: 49–53.

Ostlind, D.A., Cifelli, S., and Lang, R. 1979. Insecticidal activity of the anti-parasitic avermectins. Vet. Rec. **105**: 168.

Pollock, K.H., Nichols, J.D., Brownie, C., and Hines, J.E. 1990. Statistical inference for capture–recapture experiments. Wildl. Monogr. No. 107.

Reid, D.G., Krebs, C.J., and Kenney, A. 1995. Limitation of collared lemming population growth at low densities by predation mortality. Oikos, **73**: 387–398.

Smith, A., and Willebrand, T. 1999. Mortality causes and survival

rates of hunted and unhunted willow grouse. J. Wildl. Manag. **63**: 722–730.

Steen, H. 1994. Low survival of long distance dispersers of the root vole (*Microtus oeconomus*). Ann. Zool. Fenn. **31**: 271–274.

Steen, H. 1995. Untangling the causes of disappearance from a local population of root voles, *Microtus oeconomus*: a test of the regional synchrony hypothesis. Oikos, **73**: 65–72.

Steen, H., Holst, J.C., Solhøy, T., Klaussen, E., Prestegard, I., Sundt, R.C., and Johannesen, Ø. 1997. Mortality of lemmings, *Lemmus lemmus*, at a peak density in a mountainous area of Norway. J. Zool. (Lond.), **243**: 831–835.

Thirgood, S.J., Redpath, S.M., Haydon, D.T., Rothery, P., Newton, I., and Hudson, P.J. 2000. Habitat loss and raptor predation: disentangling long- and short-term causes of red grouse declines. Proc. R. Soc. Lond. B Biol. Sci. **267**: 651–656.

White, G.C., and Burnham, K.P. 1999. Program MARK: survival estimation from populations of marked animals. Bird Study, **46**(Suppl.): 120–138.