Ecology Letters, (2006) **9**: 383–389 doi: 10.1111/j.1461-0248.2006.00890.x

LETTER

Predator disease out-break modulates top-down, bottom-up and climatic effects on herbivore population dynamics

Christopher C. Wilmers,[1]* Eric Post,[1] Rolf O. Peterson[2] and John A. Vucetich[2]

[1]Department of Biology, Penn State Institutes of the Environment, Penn State University, 208 Mueller Lab, University Park, PA 16802, USA

[2]School of Forest Resources and Environmental Science, Michigan Technological University, Houghton, MI 49931, USA

**Correspondence and present address: Department of Environmental Science and Policy, University of California, Davis, CA 95616, USA. E-mail: cwilmers@ucdavis.edu*

Abstract

Human-introduced disease and climatic change are increasingly perturbing natural ecosystems worldwide, but scientists know very little about how they interact to affect ecological dynamics. An outbreak of canine parvovirus (CPV) in the wolf population on Isle Royale allowed us to test the transient effects of an introduced pathogen and global climatic variation on the dynamics of a three-level food chain. Following the introduction of CPV, wolf numbers plummeted, precipitating a switch from top-down to bottom-up regulation of the moose population; consequently, the influence of climate on moose population growth rate doubled. This demonstrates that synergistic interactions between pathogens and climate can lead to shifts in trophic control, and suggests that predators in this system may play an important role in dampening the effects of climate change on the dynamics of their prey.

Keywords

Alces alces, alternate state, canine parvovirus, *Canis lupus*, climate change, food chain, Isle Royale, moose, trophic cascade, wolves.

Ecology Letters (2006) 9: 383–389

INTRODUCTION

Understanding the synergistic effects of pathogens and climate on population dynamics accrues added importance as anthropogenic epizootics and climate change exert increasing influence on natural ecosystems. Previous work linking climate and disease has focused primarily on the effects of climate on host susceptibility (Kiesecker *et al.* 2001) and disease transmission (Harvell *et al.* 2002; Rodo *et al.* 2002). These studies reveal that warming temperatures can increase pathogen development, survival rates and disease spread, with deleterious effects on host populations. Furthermore, when weather conditions are correlated over the spatial distribution of a disease and its host, climatic events can drive synchrony in host population dynamics by mediating the density-dependent transmission of parasites between individuals (Cattadori *et al.* 2005). While many studies have thus focused on the impacts of climate on disease abundance and impacts on host populations, knowledge of how disease may influence the effects of climate on community dynamics is limited. Here, we consider how the introduction of an epizootic may influence the effects of large-scale climate on a three-level food chain by modulating the relative influence of top-down and bottom-up effects.

Isle Royale, USA, is a US National Park and federally designated wilderness area that has been the focus of the longest-running study of a three-trophic level system including gray wolves (*Canis lupus*), moose (*Alces alces*) and their primary winter food resource, balsam fir (*Abies balsamea*). Our previous work has shown that increases in winter snow related to the North Atlantic Oscillation (NAO) influence wolf kill rates on moose, with cascading effects on balsam fir growth (Post *et al.* 1999). Canine parvovirus (CPV) was introduced accidentally by humans in 1980 or 1981, and resulted in a dramatic crash of the wolf population from 1980 to 1982 (Peterson *et al.* 1998) (Fig. 1). Even though CPV exposure was no longer detectable in wolf blood samples from 1990 onwards, average wolf abundance and the ratio of wolves to moose

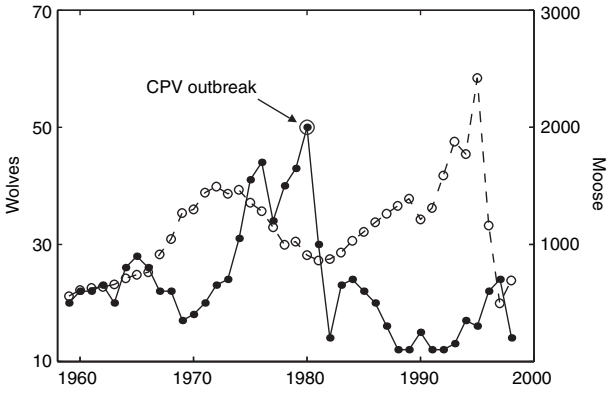

Figure 1 Wolf (solid line) and moose (dotted line) population dynamics, 1959–1998. Canine parvovirus (CPV), introduced inadvertently to the island in 1980–1981, causes a crash in the wolf population. From this point onwards the average number of moose per wolf was substantially higher (39.6 for 1959–1980; 79.8 for 1981–1998; $P < 0.01$).

since 1980 have been substantially lower than prior to the introduction of CPV (Vucetich & Peterson 2004a) (Fig. 1). Disease and inbreeding may have both contributed to the altered dynamics (Peterson *et al.* 1998; Vucetich & Peterson 2004a). Building upon our previous work, we set out to understand how the introduction of CPV influenced the relative strength of top-down, bottom-up and climatic effects on moose population dynamics. Specifically we tested the hypotheses that: (i) the relative influence of top-down vs. bottom-up factors on moose population dynamics changed after the outbreak of CPV; and (ii) climatic influences on moose dynamics would be altered if moose were relieved from limitation by an adjacent trophic level.

MATERIALS AND METHODS

Moose dynamics

To assess the influence of the CPV outbreak and subsequent crash of the Isle Royale wolves on moose population dynamics, we constructed several multiple linear regression models. We then used a number of diagnostics to test the hypotheses laid out in the previous section. The response variable for each model was the log-transformed moose population growth rate $r_t = m_{t+1} - m_t$, where m_t is the natural logarithm of the moose population in year t. Explanatory variables for each model were the NAO and log-transformed moose m, wolf w and balsam fir f abundances. In 1996, the moose population experienced a dramatic and rapid die off. The 3-week census of moose in that year took place in the middle of this die off. As such, population counts for 2 years, 1996 and 1997, reflect this die off even though most of the mortality occurred in <6 months. In order to avoid spurious correlations in our model, therefore, we exclude estimates of r_t for 1995 (because it is a large underestimate) and 1996 (because it is a large overestimate) from our analysis.

The wolf population crashed in 1980 due to an outbreak of CPV and has not yet returned to its previous range in densities due to either the disease, inbreeding, an interaction between disease and inbreeding or some as of yet undetermined factor (Peterson *et al.* 1998; Vucetich & Peterson 2004a). Our aim in this study was not to determine why the wolf population has not recovered to its pre-disease carrying capacity, but rather to use the fact that it appears to have attained a new equilibrium to investigate how the relative effects of top-down, bottom-up and climatic effects on moose may have changed. To determine whether the trophic and climatic influences on moose population dynamics differed before and after the introduction of CPV, we developed the following regression model using an indicator variable I_t that equals 0 for $t \leq 1980$ and 1 for $t > 1980$ and a measure of predation pressure p_t,

$$r_t = b_0 + a_0 I_t + (b_1 + a_1 I_t)p_t + (b_2 + a_2 I_t)m_t + (b_3 + a_3 I_t)f_t + (b_4 + a_4 I_t)\mathrm{NAO}_t + (b_5 + a_5 I_t)\mathrm{NAO}_{t-1} + (b_6 + a_6 I_t)\mathrm{NAO}_{t-2}. \quad (1)$$

Terms with I_t represent hypotheses that population dynamics prior to 1980 were different from those after 1980. We conducted two independent analyses of the model, one with $p_t = w_t$ and the other with $p_t = w_t/m_t$ as our measures of predation pressure. We used backwards elimination and Akaike's Information Criterion (AIC) scores to determine which interaction terms to keep (Table 1).

To quantify the relative effects of trophic interactions and climate on moose population dynamics, we built tri-trophic models with and without climate terms for the period before and after the outbreak of CPV given by,

$$r_t = b_0 + b_1 w_t + b_2 m_t + b_3 f_t + b_4 \mathrm{NAO}_t + b_5 \mathrm{NAO}_{t-1} + b_6 \mathrm{NAO}_{t-2} \quad (2)$$

for $t = 1959$–1980 and $t = 1980$–1998. The model was first fitted without the NAO terms. We then selected the most parsimonious climate terms by selecting the model with the lowest AIC (Table 2). For each model, we calculated the coefficient of partial determination of each of the independent variables (R^2_x) which determines the relative contribution of each variable to that model's total R^2 (Neter *et al.* 1996). Note that in this analysis we use only w_t as our measure of predation pressure because w_t/m_t is highly correlated with m_t, which would obscure any differences in bottom-up and top-down effects on r_t.

We tested for nonlinearity in our independent variables on response variables using generalized additive models (GAMs) applying the back-fitting algorithm of smoothing

Table 1 Performance of models* predicting moose population growth rate r_t (1959–1998)

Equations for r_t	ΔAIC†		R^2‡
	$p_t = w_t$	$p_t = w_t/m_t$	
$b_0 + a_0I_t + (b_1 + a_1I_t)p_t + (b_2 + a_2I_t)m_t + (b_3 + a_3I_t)f_t + (b_4 + a_4I_t)\mathrm{NAO}_t + (b_5 + a_5I_t)\mathrm{NAO}_{t-1} + (b_6 + a_6I_t)\mathrm{NAO}_{t-2}$	8.43	9.25	0.70
$b_0 + a_0I_t + (b_1 + a_1I_t)p_t + (b_2 + a_2I_t)m_t + (b_3 + a_3I_t)f_t + (b_6 + a_6I_t)\mathrm{NAO}_{t-2}$	2.94	3.76	0.68
$b_0 + a_0I_t + (b_1 + a_1I_t)p_t + (b_2 + a_2I_t)m_t + b_3f_t + (b_6 + a_6I_t)\mathrm{NAO}_{t-2}$	0.95	1.77	0.68
$b_0 + a_0I_t + (b_1 + a_1I_t)p_t + b_2m_t + (b_3 + a_3I_t)f_t + (b_6 + a_6I_t)\mathrm{NAO}_{t-2}$	1.93	1.99	0.67
$b_0 + a_0I_t + b_1p_t + (b_2 + a_2I_t)m_t + (b_3 + a_3I_t)f_t + (b_6 + a_6I_t)\mathrm{NAO}_{t-2}$	3.61	4.43	0.65
$b_0 + a_0I_t + (b_1 + a_1I_t)p_t + b_2m_t + b_3f_t + (b_6 + a_6I_t)\mathrm{NAO}_{t-2}$	0	0	0.67
$b_0 + a_0I_t + b_1p_t + b_2m_t + b_3f_t + (b_6 + a_6I_t)\mathrm{NAO}_{t-2}$	1.82	2.63	0.63
$b_0 + b_1p_t + b_2m_t + b_3f_t + b_6\mathrm{NAO}_{t-2}$	20.74	21.55	0.29

Terms with I_t represent hypotheses that determinants of moose population dynamics prior to the introduction of canine parvovirus in 1980 differ from those after 1980. We conducted separate analyses using both w_t and w_t/m_t as measures of predation pressure (see Materials and methods).

*We display the fully parameterized model, the six models with lowest AIC scores and the model with no indicator terms.

†ΔAIC values are differences in AIC between the given model and the best model.

‡R^2 (i.e. coefficient of determination; Neter *et al.* 1996) values did not differ between models using different measures of predation pressure.

splines (Venables & Ripley 1994). If the use of a GAM, compared with our linear model, failed to significantly reduce the model's sum of squares, we used the linear model. All models were additionally checked for multicollinearity and normality assumptions.

Data

We used yearly abundance data of moose, wolves and balsam fir from 1959 to 1998 on Isle Royale in our analysis. The entire wolf population is censused annually and the total abundance of moose is estimated by a random-stratified aerial survey technique (Peterson & Page 1993). Balsam fir abundance is represented by a unit-less index based on analysis of annual growth increments of tree rings. Details on the methodology of data collection have been reported elsewhere (McLaren & Peterson 1994; Peterson *et al.* 1998; Vucetich & Peterson 2004b). We used balsam fir data from the east side of the Island, which have been shown to have the largest effect on moose population dynamics (correlation between moose growth rate and fir growth is − 0.01 for the west end and 0.39 for the east end of the island respectively) (Vucetich & Peterson 2004b). This is presumably due in large part to the fact that most of the moose reside on the east end of the island, and that the east end of the island is a healthy balsam fir forest with abundant browse-age trees whereas the west side of the island has very few fir trees of browsing height.

The NAO is a large-scale fluctuation in mass balance between air pressure centres over the Azores and Iceland that affects winter weather patterns over large portions of the northern hemisphere (Hurrell 1995). We obtained values of the winter NAO index from http://www.cgd.ucar.edu/cas/catalog/climind/ which has previously been linked to ungulate population dynamics in Isle Royale and elsewhere (Post & Stenseth 1998; Post *et al.* 1999; Post & Forchhammer 2002; Vucetich & Peterson 2004b).

RESULTS AND DISCUSSION

Our best-fit model (ΔAIC = 0, Table 1) using indicator variables reveals an interaction between wolves and period (before and after), and between climate and period, indicating that the effects of these two variables on r_t differed before and after the outbreak of CPV on the island. This model also explained 67% of the variation in r_t, which was substantially more parsimonious than previous models using the entire time series have been able to explain despite including more biological variables (R^2 of 0.67 vs. 0.57 and ΔAIC = 8.1) (Vucetich & Peterson 2004b). When conducting an information theoretic analysis, models with a ΔAIC < 2 cannot be discounted (Burnham & Anderson

Table 2 Tri-trophic models of moose population dynamics before and after introduction of canine parvovirus (CPV) to wolf population in 1980 with 1 and 2 year lagged North Atlantic Oscillation (NAO)

Individual variable (ln)	Lag (years)	Standardized coefficient	Partial R^2
(a) Before (1959–1980), without climate			
Wolves (w_t)	0	**−0.59**	0.38
Moose (m_t)	0	−0.28	0.11
Fir (f_t)	0	0.16	0.02
Total			0.51
(b) After (1980–1998), without climate			
Wolves	0	−0.11	0.01
Moose	0	−0.37	0.08
Fir	0	***0.48***	0.20
Total			0.29
(c) Before (1959–1980), with climate			
Wolves	0	**−0.51**	0.33
Moose	0	−0.23	0.08
Fir	0	0.22	0.02
NAO	1	−0.30	0.14
Total			0.57
(d) After (1980–1998) with climate			
Wolves	0	−0.13	0.01
Moose	0	−0.30	0.06
Fir	0	***0.53***	0.22
NAO	1	−0.17	0.01
Total			0.30
(e) Before (1959–1980), with climate			
Wolves	0	**−0.52**	0.34
Moose	0	−0.29	0.11
Fir	0	0.19	0.02
NAO	2	−0.24	0.09
Total			0.56
(f) After (1980–1998), with climate			
Wolves	0	−0.16	0.01
Moose	0	**−0.49**	0.11
Fir	0	0.24	0.10
NAO	2	**0.61**	0.37
Total			0.59

Significant coefficients at $P < 0.05$ level are in bold whereas those at $P < 0.07$ level are in italics and bold. Models (c) and (f) were the most parsimonious ($\Delta AIC = 0$) fit models before and after CPV introduction respectively.

2002). Therefore, it is plausible that the effects of moose or balsam fir abundance on moose population growth rate also changed after the introduction CPV (see Table 1). Of the seven models with a $\Delta AIC < 2$ (four for $p_t = w_t$ and three for $p_t = w_t/m_t$), however, six revealed interactions between both period and wolves, and period and NAO. This gives us additional confidence that changes in the influence of these two variables underlie the change in moose population dynamics before and after 1980.

Deconstructing the full time series into before and after periods reveals that, prior to the outbreak of CPV, wolves exerted a strong regulatory influence on moose population dynamics while climate was a substantially weaker factor (Fig. 2a,b). After the CPV outbreak, which triggered a shift in the wolf population to lower densities (Peterson *et al.* 1998), wolf regulation of moose dynamics ceased, and climate exerted a strong influence on moose population growth rate r_t (Fig. 2a,b).

After the CPV-induced crash in the wolf population, biotic mechanisms controlling the moose population shifted from top-down (wolf abundance) to bottom-up (moose abundance and fir production) factors. Our tri-trophic model both with and without NAO terms reveals a strong

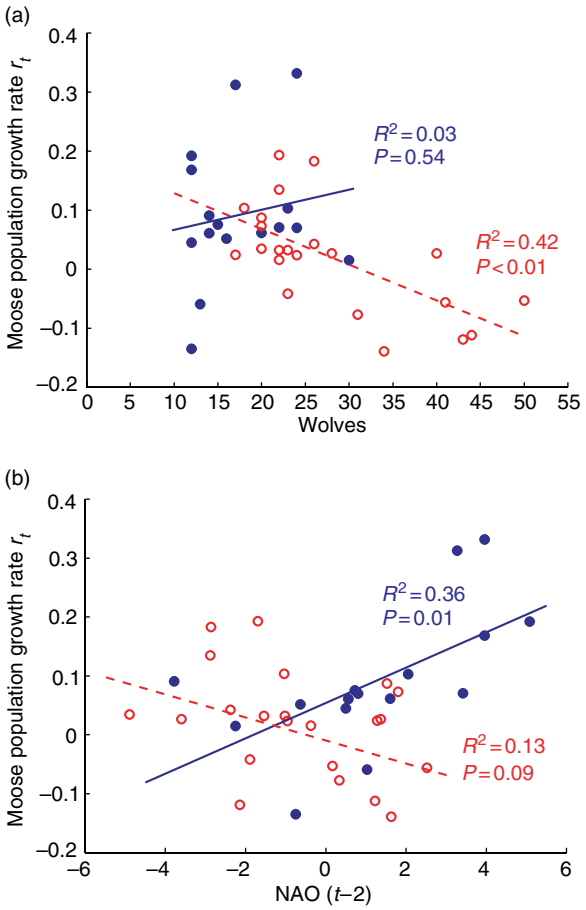

Figure 2 Progression of canine parvovirus (CPV) effects on moose population dynamics on Isle Royale. (a) Wolves have significant negative effect on moose population dynamics before (open circles) CPV outbreak and no effect after (closed circles) outbreak. (b) Climate, as measured by the North Atlantic Oscillation (NAO), has an insignificant effect on moose population dynamics before the crash in the wolf population and a highly significant influence after the crash.

significant ($P < 0.05$) negative influence of wolves on r_t before the CPV outbreak, and a weak insignificant influence afterwards (Table 2a–f). Top-down effects explained 38% of the variation in r_t compared with only 13% by bottom-up factors prior to 1980 (Table 2a, Fig. 3a). After 1980, top-down effects accounted for only 1% of the variation in r_t while bottom-up factors explained 28% (Table 2b, Fig. 3a). Additionally, variance in moose population abundance was greater after 1980 than before [SD(m) = 456 vs. 323]. Fir growth was an insignificant factor prior to 1980 but became a significant positive factor after the CPV outbreak (Table 2a–b).

Adding NAO terms to the model reveals the interaction between climate and predation on moose population dynamics. Before the switch from top-down to bottom-up dynamics in 1980, the NAO exerted a weak, 1 year lagged effect on moose population growth rate (Table 2c–f, Fig. 3b). However, after 1980 the NAO had a much more pronounced 2 year lagged effect on r_t (Table 2c–f, Fig. 3b). A GAM including the NAO did not reveal any nonlinearities. In addition, adding nonlinear NAO terms to the model did not improve the model's fit.

The increase in the NAO lag from 1 to 2 years with the switch from top-down to bottom-up control is consistent with a recent analysis of a theoretical model suggesting that indirect effects of climate on herbivore population dynamics should elicit a 1 year lagged effect when cascading through predators and a 2 year lagged effect when cascading through plants (Post & Forchhamer 2001). When winter climate mediates predation success, this is reflected in the number of moose counted the next year. However, when winter climate mediates plant growth this is reflected in the amount of browse availability the subsequent winter and thus affects the number of moose counted in the year after. However, this line of reasoning is speculative. Large-scale climate

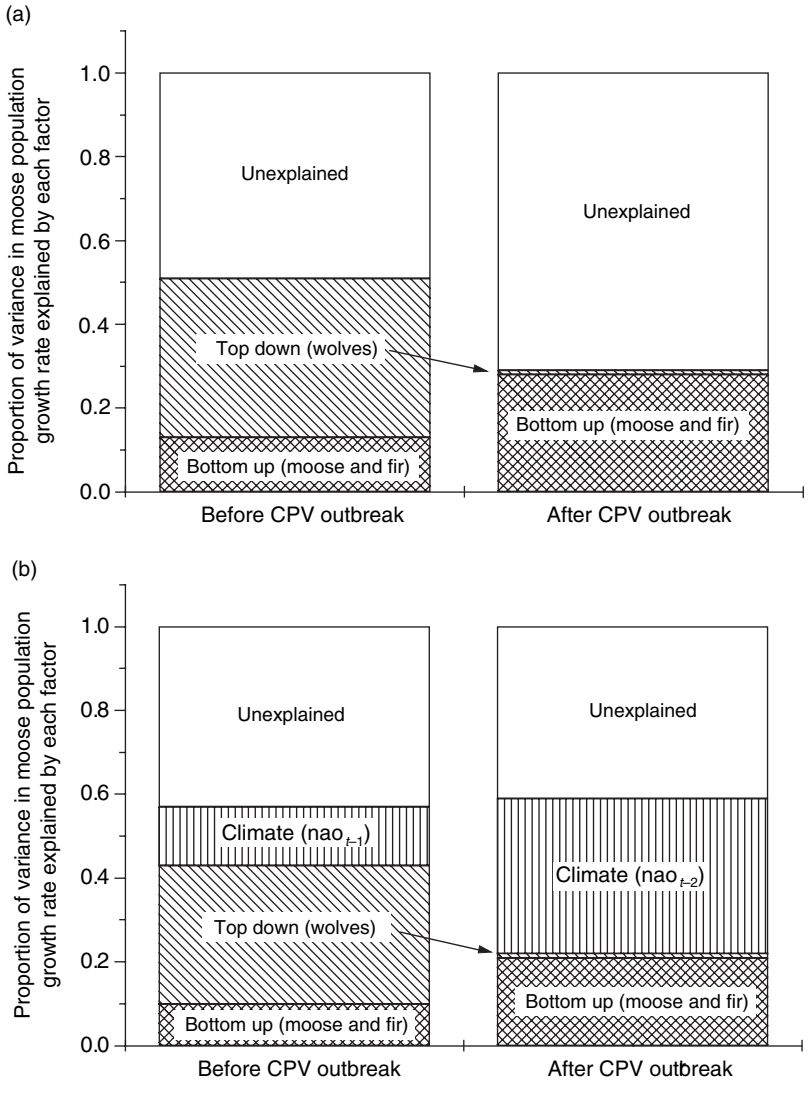

Figure 3 Role of canine parvovirus (CPV) outbreak on trophic factors affecting moose population dynamics. Hatched areas represent the variance R^2_x in moose population growth rate explained by each variable. (a) Before the outbreak of CPV, known biotic factors regulating moose population dynamics are primarily top-down (3 : 1 ratio) while after outbreak they are primarily bottom-up (28 : 1). (b) Adding climate to the model illustrates the accrued importance of global climatic variation on moose dynamics after the CPV-induced crash in the wolf population. The arrow points to the proportion of variation explained by top-down control in the post-CPV period.

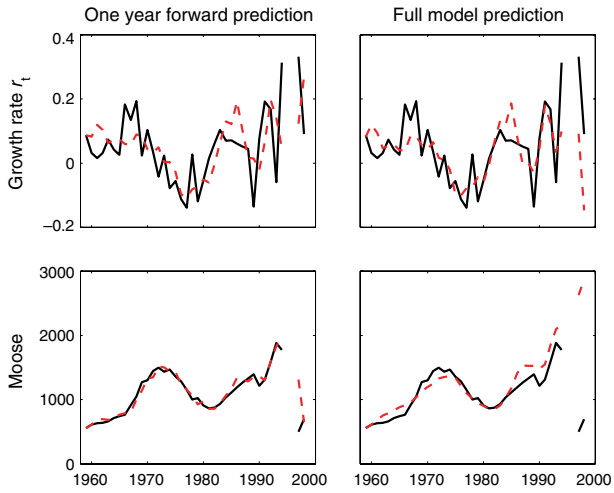

Figure 4 Observed (solid line) and predicted (dashed line) population and population growth rates for moose, 1959–1998, using the most parsimonious (ΔAIC = 0) model from Table 1 with wolves, moose, fir and NAO ($t - 2$) as predictor variables. The 1 year forward prediction is a run of the model where the value of moose at each time step is updated from the data in order to predict moose abundance and growth rate the next year. The full model prediction is a run of the model using data for moose abundance from 1959 only and predicting each subsequent value from the model. The years 1995 and 1996 were excluded from the model because of census issues in 1996 (see Materials and methods).

phenomena such as the NAO often tend to predict local dynamics better than local weather variables because they are integrative measures that encompass many different aspects of climatic variability (Post & Stenseth 1999; Hallett *et al.* 2004). Thus, while the use of NAO increases our predictive ability it may obscure a mechanistic understanding of how climate is influencing the system.

While our best model explained a substantial amount of the variation in moose population growth rate, 33% of the variation in r_t remains unexplained. Additional biotic factors such as outbreaks of winter ticks may also play an important role in regulating moose dynamics (Delgiudice *et al.* 1997), but long-term data are not yet available to assess these effects. Various forms of competition between moose may also contribute to the unexplained variation in the model. While our technique does incorporate some competition by including a moose density term as an independent variable, there may be competition between moose for such things as enemy-free space that increase nonlinearly with density and are hence not captured by the model. Despite the unexplained variation in r_t, our model expands our previous efforts to understand the dynamics of this system, and performs well in explaining the past dynamics of this moose population (Fig. 4).

Because they exert biotic control over ecosystem processes, consumers may play an important role in dampening climate-induced fluctuations in natural systems. Recent analyses of multispecies data from Yellowstone National Park, for instance, have revealed that wolves buffer the effects of climate change on carrion availability to scavengers in that system (Wilmers & Getz 2005; Wilmers & Post 2006). In the Caribbean, sea urchins (*Diadema antillarum*) and herbivorous fish graze back invading macro algae after severe storms clear large areas of coral reef substrate. After an unidentified disease decimated urchin populations in 1983, coral reefs lost much of their resilience to large storms such that large areas of reef are now dominated by algae (McManus & Polsenberg 2004). Our study reveals that the release of moose from top-down control by wolves strengthens the contribution of climate to moose population dynamics on Isle Royale. The reduction in control of moose by biotic factors and the corresponding increase in abiotic climatic factors may erode the stability of this community (Post & Forchhamer 2001). Moose population dynamics prior to the outbreak of CPV in 1980, for instance, were characterized by a relatively slow and steady increase and decrease in the population (Fig. 1). Since 1980, however, the moose population has displayed irruptive dynamics characterized by a steep increase in a population relatively free from significant predatory pressure, and dramatic declines in numbers when winters are severe.

The effects of introduced CPV are not unlike the introduction of top predators to other ecosystems. In western Alaska, prey switching by killer whales from seals to sea otters caused a dramatic decline in otter populations, which led to an increase in sea urchins and a decline in kelp (Estes *et al.* 1998). In this prototypical example of a trophic cascade, each level in the food chain was reset to a new equilibrium density. While the introduction of CPV to Isle Royale has led to a reduction in the wolf population, the cascading effect of wolves on moose is most strongly felt in the population dynamics of moose, rather than in their equilibrium density. Prior to the appearance of CPV moose growth rate was regulated, in part, by wolves leading the moose population to fluctuate with low variance. Since CPV decimated the wolf population in 1980, moose growth rate has been regulated primarily by bottom-up factors and climate, which have led to relatively high variance fluctuations in the population.

While CPV disappeared from the wolf population of Isle Royale in the late 1980s (Peterson *et al.* 1998), wolf numbers have still not recovered to pre-1980 levels. Genetic studies of wolves on Isle Royale have revealed that they are highly inbred, having all descended from a single maternal ancestor (Wayne *et al.* 1991). The CPV-induced population crash in 1980 may have further eroded the genetic variability of the

wolf population, potentially causing recruitment difficulties. Alternative explanations, such as demographic stochasticity associated with so few breeding adults, however, cannot be discounted. Continued monitoring of the population into the future will reveal whether we are witnessing a permanent shift in the dynamics of the system, or simply a long-term perturbation from historical dynamics.

The synergistic effects of disease and climate may have long-term effects on community dynamics. Our analysis shows how a disease outbreak, acting as a pulse perturbation, has led to a shift from top-down to bottom-up control of moose population dynamics. As a result, the effects of large-scale climatic forcing are much stronger on this population than they had been previously. Whether the system returns to top-down control when and if wolf population densities return to the higher values they once exhibited would be conjecture, but results from experimental studies suggest that perturbations from top-down to bottom-up control can be persistent even with the resumption of predation (Schmitz 2004). CPV no longer persists in the wolf population on Isle Royale, yet its impact on community dynamics is still felt over 2 decades after its appearance in 1980–1981.

ACKNOWLEDGEMENTS

E.P. acknowledges the financial support of the US National Science Foundation (NSF). R.O.P. and J.A.V thank the NSF (DEB-9903671 and DEB-0424562) and Isle Royale National Park (ISRO). The content of study does not necessarily reflect the views of the NSF or ISRO.

REFERENCES

Burnham, K.P. & Anderson, D.R. (2002). *Model Selection and Multimodel Inference: A Practical Information-theoretic Approach*, 2nd edn. Springer-Verlag, New York.

Cattadori, I.M., Haydon, D.T. & Hudson, P.J. (2005). Parasites and climate synchronize red grouse populations. *Nature*, 433, 737–741.

Delgiudice, G.D., Peterson, R.O. & Samuel, W.M. (1997). Trends of winter nutritional restriction, ticks and numbers of moose on Isle Royale. *J. Wildl. Manage.*, 61, 895–903.

Estes, J.A., Tinker, M.T., Williams, T.M. & Doak, D.F. (1998). Killer whale predation on Sea otters linking oceanic and nearshore ecosystems. *Science*, 282, 473–476.

Hallett, T.B., Coulson, T., Pilkington, J.G., Clutton-Brock, T.H., Pemberton, J.M. & Grenfell, B.T. (2004). Why large-scale climate indices seem to predict ecological processes better than local weather. *Nature*, 430, 71–75.

Harvell, C.D., Mitchell, C.E., Ward, J.F., Altizer, S., Dobson, A.P., Ostfeld, R.S. *et al.* (2002). Climate warming and disease risks for terrestrial and marine biota. *Science*, 296, 2158–2162.

Hurrell, J.W. (1995). Decadal trends in the NAO: regional temperature and precipitation. *Science*, 269, 676–679.

Kiesecker, J.M., Blaustein, A.R. & Belden, L.K. (2001). Complex causes of amphibian population declines. *Nature*, 410, 681–684.

McLaren, B.E. & Peterson, R.O. (1994). Wolves, moose, and tree rings on Isle Royale. *Science*, 266, 1555–1558.

McManus, J.W. & Polsenberg, J.F. (2004). Coral-algal phase shifts on coral reefs: ecological and environmental aspects. *Prog. Oceanogr.*, 60, 263–279.

Neter, J., Kutner, M.H., Nachtsheim, C.J. & Wasserman, W. (1996) *Applied Linear Statistical Models*, 4th edn. McGraw-Hill, Boston, MA.

Peterson, R.O. & Page, R.E. (1993). Detection of moose in midwinter from fixed-wing aircraft over dense forest cover. *Wildl. Soc. Bull.*, 21, 80–86.

Peterson, R.O., Thomas, N.J., Thurber, J.M., Vucetich, J.A. & Waite, T.A. (1998). Population limitation and the wolves of Isle Royale. *J. Mammal.*, 79, 828–841.

Post, E. & Forchhamer, M.C. (2001). Pervasive influence of large-scale climate in the dynamics of a terrestrial vertebrate community. *BMC Ecol.*, 1, 5–12.

Post, E. & Forchhammer, M.C. (2002). Synchronization of animal population dynamics by large-scale climate. *Nature*, 420, 168–171.

Post, E. & Stenseth, N.C. (1998). Large-scale climatic fluctuation and population dynamics of moose and white-tailed deer. *J. Anim. Ecol.*, 67, 537–543.

Post, E. & Stenseth, N.C. (1999). Climatic variability, plant phenology, and northern ungulates. *Ecology*, 80, 1322–1339.

Post, E., Peterson, R.O., Stenseth, N.C. & McLaren, B.E. (1999). Ecosystem consequences of wolf behavioural response to climate. *Nature*, 401, 905–907.

Rodo, X., Pascual, M., Fuchs, G. & Faruque, A.S.G. (2002). ENSO and cholera: a nonstationary link related to climate change? *Proc. Natl Acad. Sci. USA*, 99, 12901–12906.

Schmitz, O.J. (2004). Perturbation and abrupt shift in trophic control of biodiversity and productivity. *Ecol. Lett.*, 7, 403–409.

Venables, W.N. & Ripley, B.D. (1994) *Modern Applied Statistics in S-Plus*. Springer, New York.

Vucetich, J.A. & Peterson, R.O. (2004a). The influence of prey consumption and demographic stochasticty on population growth rate of Isle Royale wolves *Canis lupus*. *Oikos*, 107, 309–320.

Vucetich, J.A. & Peterson, R.O. (2004b). The influence of top-down, bottom-up and abiotic factors on the (*Alces alces*) population of Isle Royale. *Proc. R. Soc. Lond. B, Biol. Sci.*, 271, 183–189.

Wayne, R.K., Lehman, N., Girman, D., Gogan, P.J.P., Gilbert, D.A., Hansen, K. *et al.* (1991). Conservation genetics of the endangered Isle-Royale gray wolf. *Conserv. Biol.*, 5, 41–51.

Wilmers, C.C. & Getz, W.M. (2005). Gray wolves as climate change buffers in Yellowstone. *PLoS Biol.*, 3, 571–576.

Wilmers, C.C. & Post, E. (2006). Predicting the influence of wolf-provided carrion on community dynamics under climate change scenarios. *Global Change Biol.*, 12, 403–409.

Editor, Boris Worm
Manuscript received 4 October 2005
First decision made 14 November 2005
Second decision made 12 December 2005
Manuscript accepted 20 December 2005