

Lecture 10. Predation, Parasitism, and Herbivory.

Reading:

Gotelli, 2001, *A Primer of Ecology*, Chapter 6, pages 125-153.

Renshaw (1991) Chapter 6 Predator-prey processes, Pages 166-204.

Optional:

Gasaway, W. C., R. D. Boertje, D. V. Grangaard, D. G. Kelleyhouse, R. O. Stephenson, and D. G. Larsen. 1992. The role of predation in limiting moose at low densities in Alaska and Yukon and implications for conservation. *Wildlife Monograph* 120. 59 pp.

Boutin, S. 1992. Predation and moose population dynamics: a critique. *Journal of Wildlife Management* 56:116-127.

Krebs, C. J. et al. 1992. What drives the snowshoe hare cycle in Canada's Yukon? Pages 886-896 in D. R. McCullough and R. H. Barrett, eds. *Wildlife 2001: Populations*. Elsevier Applied Science, New York, New York, USA.

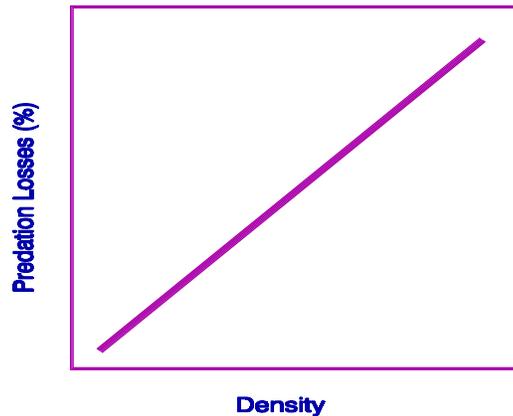
The notion that one trophic level controls the dynamics of another trophic level is central to ecology. Energy flows between trophic levels. The input of energy to a higher trophic level population from a lower trophic level population can be controlled by the amount of energy available in the lower level, or by the amount of energy the higher trophic level is able to consume. Hence, the ideas of this chapter are as pertinent to herbivores and plants as they are to predators and their prey. If the prey population limits the predator population, the system is referred to as bottom-up control. Vice versa, if the predator limits the prey population, then the system is referred to as top-down control.

The idea that predators control prey (Hairston et al. 1960), or top-down control, has been pervasive in the literature for a long time. In a review of the impact of small-rodent population dynamics, Hanski et al. (2001) conclude that predators are indeed causing the regular multi-annual population oscillations of boreal and arctic small rodents (voles and lemmings).

Alternatively, the notion that predators only take the weak and the sick (Errington 19??) suggests that predators are not controlling prey populations. Rather, predators are only taking the “doomed surplus”, and therefore, are not additive to the prey death rate, but are compensatory.

Cause and Effect. Demonstration of predators having an impact on prey population must be done via manipulative experiments. The following graph shows a correlation between predator density and the proportion of prey consumed (killed by predators). However, this correlation is not evidence that the predator population is in any way controlling the prey population density. For example, reduction in predator numbers might result in increased starvation of prey because of density dependence, and hence predation is compensatory to starvation. Unfortunately, actual manipulations of predator and prey

populations are limited, and the few that do appear in the literature usually lack replication and appropriate spatial and temporal controls. As a result, this area of population dynamics is still highly controversial.



Most of the predator-prey models stem either from the original models of Lotka (1925) and Volterra (1926) or from those of Nicholson (1933) and Nicholson and Bailey (1935). These models provide an historical perspective and a foundation on which to build a rigorous mathematical model to base our discussion. Both types of model are based on simple assumptions. Both are a closed system involving coupled interactions. Neither model involves age structure for either the predator or prey. Predation is a linear function of prey density, implying insatiable predators with no handling time. And, both models assume that the prey eaten are directly converted into new predators. The chief difference lies in the Lotka-Volterra models being differential equations, while the Nicholson-Bailey models are based on difference equations giving discrete, non-overlapping generations (Hassell and Anderson 1989). The following equations are the Lotka-Volterra model:

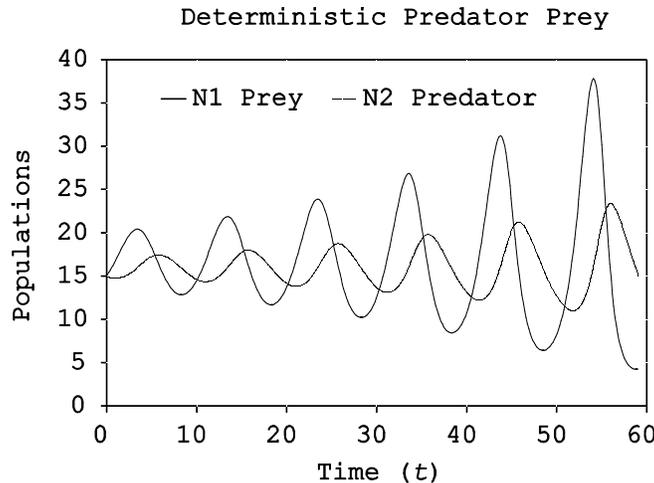
$$\begin{aligned}\frac{dN_1}{dt} &= b_1N_1 - d_1N_1 = b_1N_1 - \delta N_1N_2 \\ \frac{dN_2}{dt} &= b_2N_2 - d_2N_2 = \beta N_1N_2 - d_2N_2\end{aligned}$$

The death rate (d_1) of the prey (N_1) is set proportional to the size of the predator population (N_2) as δN_2 . Likewise, the birth rate (b_2) of the predator population is set proportional to the prey population as βN_1 . Units of b_1 , b_2 , d_1 , and d_2 are time^{-1} , and units for β are predators born/predator/prey/time and for δ prey eaten/prey/predator/time. Thus, the equations generate units of prey/time or predators/time for the respective rates. Some of the important characteristics of these equations are:

- lack of density-dependence,
- death rate of prey only depends on predator and prey,

death rate of predator only depends on predator, and
birth rate of predator depends on predator and prey.

Lotka-Volterra equations predict a stable cycle of predator and prey for a “pathological” set of parameter values (Hassell and Anderson 1989), whereas the difference equation formulation results in a expanding oscillations, as shown in the following graph.



Equilibrium can only be obtained via very carefully set parameter values. Yet, the literature is full of theoretical results derived from these simple equations. More appalling, these equations have been suggested as explaining some of the classic predator-prey cycles observed in nature, e.g., the lynx cycle based on fur returns for the Mackenzie River region of Canada, 1821-1934, reported by Elton and Nicholson (1942). These equations are a classic case of the mathematical tail wagging the biological dog. The equations have so little reality that its hard to see how they have much to do with real systems. Still, numerous hypotheses have been developed from these equations. I will now explore some of the extensions, and the consequences of these modifications to the predicted behavior of the predator and prey populations.

Density Dependence. As a first attempt to incorporate some biological realism, density dependence of the prey population might be included. As formulated above, the prey population grows exponentially without predators. Density dependence might also be incorporated into the predator population, giving the following equations:

$$\frac{dN_1}{dt} = b_1 N_1 - \delta N_1 N_2 - c_1 N_1^2$$

$$\frac{dN_2}{dt} = \beta N_1 N_2 - d_2 N_2 - c_2 N_2^2$$

Cycles no longer result with these equations.

Extensions commonly made to the Lotka-Volterra model involve the functional and numerical responses. The functional response defines the changes in the *per capita* predation rate as prey density increases. The numerical response defines the changes in predator density as prey density increases.

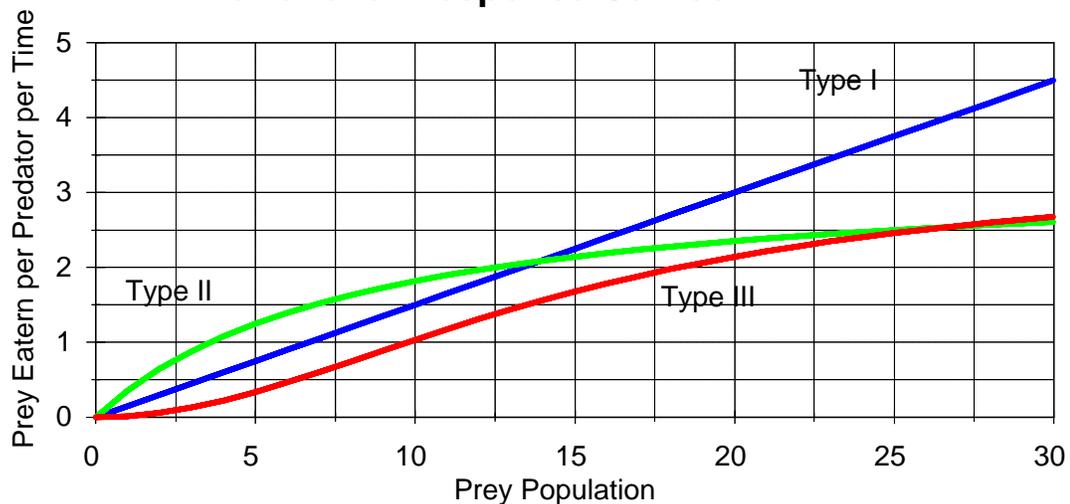
Functional Response Curves. A predator's functional response is its per capita feeding rate on prey. Holling (1965, 1966) suggested that the predator should not be able to consume an unlimited number of prey as the prey population increases. That is, in the Lotka-Volterra equations, the number of prey consumed per predator is unlimited as the prey population increases. The number of prey removed is $\delta N_1 N_2$, so that the number of prey eaten per predator is unlimited as N_1 increases to infinity. Holling proposed 3 models of the rate of prey capture per predator as a function of prey population density: Types I, II, and III. Type I is the default case already modeled in the Lotka-Volterra equations.

Type I: $m_1 = aTN_1$, where m_1 is number of prey eaten over a time period T , per predator, and a is a proportionality constant.

Type II: $m_1 = a T N_1 / (1 + a T_H N_1)$, where T_H is the time required to catch and devour a prey (handling time). This equation is obtained by substituting into the Type I and correcting for the handling time, $m_1 = a(T - m_1 T_H)N_1$, and solving for m_1 .

Type III: $m_1 = a T N_1^\eta / (1 + a T_H N_1^\eta)$, where η is generally set equal to 2. η generates a lag time in the learning curve of the predator, or a "training effects". The idea of adding a power term is a common mathematical trick, e.g., the Richard's equation described as a modification of the logistic population growth model. For the following examples, the parameter values are I: $a = 0.5$, $T = 1$; II: $a = 0.4$, $T = 1$, $T_H = 0.3$, and III: $a = 0.015$, $T = 1$, $T_H = 0.3$, and $\eta = 2$.

Functional Response Curves



Depensatory mortality is a term used to describe the decrease in the rate of prey mortality as the prey population increases. That is, as all the predators become satiated because of the large numbers of prey, the number of prey killed per number of prey available declines, so that the prey survival rate will increase. This phenomena will occur as the Type II and III curves reach asymptotic values of the prey population. The Lotka-Volterra model is extended to incorporate Type II and III curves, i.e., the rate at which predators eat prey is modified by the functional responses shown above. The cyclic behavior can persist with Type II and III functional responses incorporated into the Lotka-Volterra equations.

Holling (1965) justified the Type III functional response curve based on empirical evidence. He buried sawfly cocoons in sand (the prey), and let mice (*Peromyscus*) search for them. Until they learned how to find the cocoons, they were less effective than after they had become proficient. Swenson (1977) (in Emlen 1984:108) constructed functional response curves for walleye and sauger, and found that a Type II curve was adequate.

Skalski and Gilliam (2001) review the literature on functional response curves and presented statistical evidence from 19 predator-prey systems that three predator-dependent functional responses (Beddington-DeAngelis, Crowley-Martin, and Hassell-Varley), i.e., models that are functions of both prey and predator abundance because of predator interference, can provide better descriptions of predator feeding over a range of predator-prey abundances. No single functional response best described all of the data sets. The Beddington-DeAngelis functional response curve (Beddington 1975, DeAngelis et al. 1975) is:

$$f(N_1, N_2) = \frac{aN_1}{1 + bN_1 + c(N_2 - 1)},$$

where when the value of c becomes zero, this functional response curve becomes identical to Hollings Type II curve. This model assumes that handling and interfering are exclusive activities. The Crowley-Martin (Crowley and Martin 1989) allows for interference among predators regardless of whether a particular individual is currently handling prey or searching for prey. The Crowley-Martin model thus adds an additional term in the denominator:

$$f(N_1, N_2) = \frac{aN_1}{1 + bN_1 + c(N_2 - 1) + bcN_1(N_2 - 1)},$$

which can be simplified to

$$f(N_1, N_2) = \frac{aN_1}{(1 + bN_1)(1 + c(N_2 - 1))}.$$

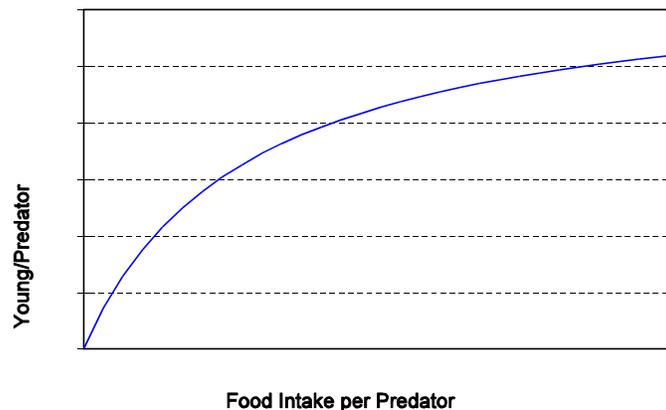
As with the Beddington-DeAngelis curve, making c equal to zero results in a Type II curve. The third functional response curve considered by Skalski and Gilliam (2001) was the Hassell-Varley (Hassell and Varley 1969) model:

$$f(N_1, N_2) = \frac{aN_1}{bN_1 + N_2^m},$$

where when m becomes zero reduces to a Type II curve.

Vucetich et al. (2002) also consider a number of different functional response curves to model the relationship between wolves and moose on Isle Royale. Predator density explained more variation in kill rate than did prey density ($R^2 = 0.36$ vs. $R^2 = 0.17$, respectively). The ratio-dependent model greatly outperformed the prey-dependent model. Nevertheless, the ratio-dependent model failed to explain most of the variation in kill rate ($R^2 = 0.34$). The ratio-dependent – prey-dependent controversy should disappear as investigators recognize that both models are overly simplistic.

Numerical Response Curves. Numerical response is the response of predator populations to prey populations. Predator birth rate may be a function of the food intake rate, so that increased prey availability may result in an increased birth rate, up to some asymptotic value. Another possibility is that predators immigrate to an area, or aggregate in an area of high prey density. Type II and III curves are also useful for modeling this process. This type of response results in a numerical response of the predator to the prey, i.e., the number of predators increases in response to the number of prey. The following graph illustrates an hypothetical example.

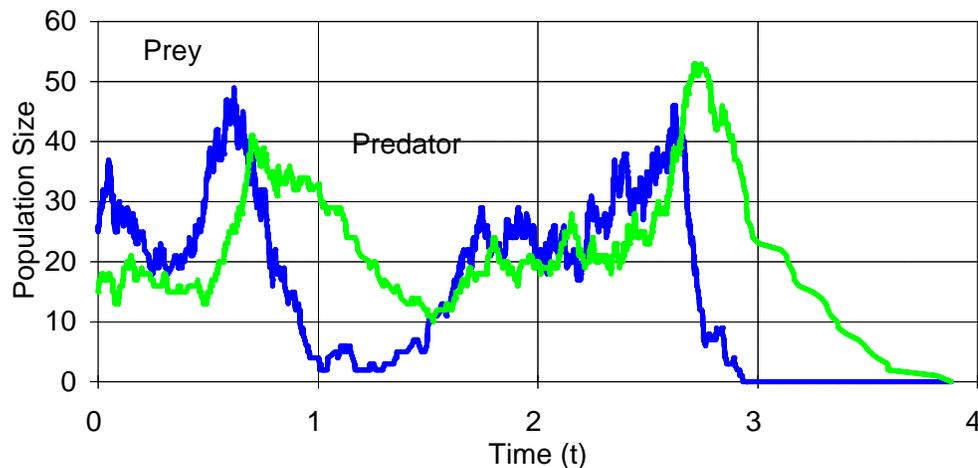


Maker (1970) (in Emlen 1984) demonstrated a Type III response of jaeger nest density to lemming density. Johnson and Carpenter (1994) used functional and numerical responses to develop a fish-angler interaction model.

Stochasticity. The cyclic behavior of these equations will not be retained in a model that includes demographic stochasticity. Typically, both populations will go extinct.

What predator-prey system can you think of that does not have demographic stochasticity? Environmental stochasticity might also reasonably be added to the Lotka-Volterra model, i.e., the basic rate constants become functions of the environment. For example, wolves pursuing moose benefit from snow conditions that support the weight of a wolf, but not the weight of a moose. An example of a Lotka-Volterra model which incorporates demographic stochasticity is shown in the following graph.

Stochastic Predator-Prey Model



Spatial stochasticity should also be added to provide a realistic model.

Examples of some of the hypotheses (some authors consider the following as “conclusions”) (taken from Emlen 1984):

1. Predator-prey system is more likely stable if the predator is not highly efficient at finding and capturing prey.
2. Predator-prey system is more likely stable if predator is not highly efficient in handling prey.
3. Predator-prey system is more likely stable if predator is not highly efficient at converting food to growth and reproduction.
4. Enrichment of a predator-prey system by addition of food for the prey or alternative sustenance for the predator destabilizes the system.
5. Many (most) prey species have available to them some form of “refuge” that prevents extinction by the predator. As a result, the predator-prey system may show greater amplitude in the cycling.
6. Time lags destabilize a predator-prey system.
7. Density-dependence takes out cyclic behavior of a predator-prey system.
8. Stochasticity (both demographic and environmental) takes out cyclic behavior of a predator-prey system.

9. Spatial stochasticity can be viewed as a refuge system, or as a metapopulation system. Too low of prey dispersal makes the system unstable, just as too high of prey dispersal does.

Coevolution. The predator-prey system is sensitive to the efficiency of the predator taking prey, and ultimately determines the stability of the system. We can assume that natural selection is constantly operating on the system, so that the predator is improving its abilities to capture prey, whereas the prey is improving its abilities to avoid capture. The predator and prey are co-evolving, like an evolutionary arms race.

Experimental Studies

Huffaker experiments (Huffaker 1958). Prey was six-spotted mite (*Eotetranychus sexmaculatus*), predator was a predatory mite, *Typhlodromus occidentalis*. Oranges, rubber balls, and wax paper were used to construct experiments where the prey had different habitat patches available to it, providing different configurations of prey to the predator. “To ensure that his experiment was sufficiently complex, Huffaker placed 40 oranges or balls in a 4 X 10 rectangular array on each of a number of adjacent trays. Migration of predators across trays was restricted by inserting vaseline barriers; whilst migration of prey over these barriers was achieved by providing each tray with wooden posts from which the prey could launch themselves on a silken thread, aided by currents from an electric fan.” (Renshaw 1991:206). This is an example of biological modeling not using equations. Huffaker did observe oscillations in the predator and prey system.

Wolves and Moose in Alaska and Yukon (Gasaway et al. 1983, 1992; Boutin 1992). High predation on moose calves by wolves was thought to be limiting the moose population. After wolves were removed from the system, the moose populations responded upward. Hence, moose at low densities were being controlled by wolves. Gasaway et al. (1992) termed the original state of the system as a Low Density Dynamic Equilibrium (LDDE). When wolves were removed temporarily from the system, the moose population climbed towards the carrying capacity set by browse, and escaped the wolf predation limitation because relatively more moose calves escaped wolf predation.

Mathematics (if any) of LDDE (Messier 1994)

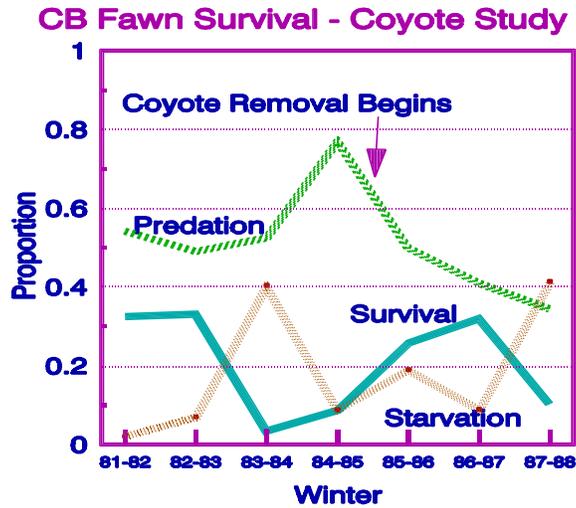
Is the reason that cycles are most often observed in Arctic systems because of the simplicity of these systems, and the strong seasonality?

Wolves and Moose on Isle Royale (McLaren and Peterson 1994).

This article infers top down control because the only manipulation(?) was a change in the wolf population. Are their inferences really valid, given the lack of a true experiment?

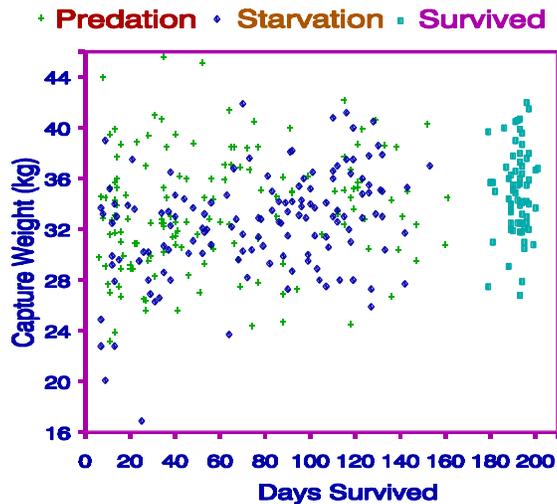
Coyotes and Mule Deer in Northwestern Colorado (Bartmann et al. 1992). These authors

observed high overwinter fawn mortality from coyote predation.



Removal of coyotes (93, 78, and 47 in 1985-86, 86-87, and 87-88, respectively) did not increase fawn survival compared to the previous 4 winters prior to coyote removal, and suggested an increase in starvation of fawns. Hence coyotes are providing compensatory mortality in the fawn population, not additive mortality as would be suggested by the large numbers of fawns killed by coyotes each winter.

Predators only take weak and sick -- NOT!, but prudent predators do take the easiest prey.



Mule deer at KCC.

Snowshoe hare cycles (Krebs et al. 1992). Krebs et al. (1992) present good evidence of snowshoe hare (*Lepus americanus*) cycles in southwestern Yukon. Four hypotheses have been proposed to explain the hare cycles:

1. Keith hypothesis: winter food shortage reduces reproduction at the population peak and causes starvation losses which start the cyclic downturn, and predation, which continues the downturn and reduces hares to low numbers.
2. Plant chemistry hypothesis: qualitative (nutritional) changes in the hare's food plants.
3. Predation hypothesis: differs from Keith's hypothesis by being a single-factor model for the hare cycle.
4. Chitty hypothesis, or polymorphic behavior hypothesis: driving mechanism is the spacing behavior of the hares themselves.

In their study at Kluane Lake during 1977-84, they have performed experiments with winter food supplementation of hares. "The overall results of these studies were rather inconsistent with the food hypothesis. Winter food shortage was not necessary for the cyclic decline, and extra food would neither slow the rate of decline of the hares nor delay in all populations the start of the decline. In these experiments, we did not control for increased predation on the food-supplemented areas..." (Krebs et al. 1992:888). "Keith et al. (1984), studying the same cyclic decline in central Alberta hares, also concluded that food shortage was not present on all areas where hares were declining. If these results are accepted, neither the original Keith hypothesis nor the plant chemistry hypothesis can be supported as an explanation of cyclic events on a local scale." (Krebs et al. 1992:888). Krebs et al. (1992) eliminate the Chitty hypothesis because no evidence of social mortality, either directly through infanticide or fighting, or indirectly through territoriality and dispersal. Through the process of elimination, the only hypothesis left is predation. From 1986-present, they have set up experiments that provide controls, fertilization plots, food supplementation, predator-proof fences (which do not eliminate avian predators), and food+predator fence. Results as of 1992 suggested food supplementation had increased April 1 populations, but they had not yet observed the down-swing in the hare cycle. Presumably, eliminating predators would keep the hare population high if predation was the mechanism that causes the down turn.

Largemouth bass in a Michigan lake (Mittelbach et al. 1995).

This paper presents the results of a long-term study of changing predator densities and cascading effects in a Michigan lake in which the top carnivore, the largemouth bass (*Micropterus salmoides*), was eliminated in 1978 and then reintroduced in 1986. The elimination of the bass was followed by a dramatic increase in the density of planktivorous fish, the disappearance of large zooplankton (e.g., two species of *Daphnia* that had historically dominated the zooplankton community), and the appearance of a suite of small-bodied

cladoceran (zooplankton) species. The system remained in this state until bass were reintroduced. As the bass population increased, the system showed a steady and predictable return to its previous state; planktivore numbers declined by two orders of magnitude, large-bodied *Daphnia* reappeared and again dominated the zooplankton, and the suite of small-bodied cladocerans disappeared. Within each cladoceran species there was a steady increase in mean adult body size as planktivore numbers declined. Total zooplankton biomass increased 10-fold following the return of large-bodied *Daphnia*, and water clarity increased significantly with increases in *Daphnia* biomass and total cladoceran biomass. These changes in community structure and trophic-level biomasses demonstrate the strong impact of removing a single, keystone species, and the capacity of the community to return to its previous state after the species is reintroduced.

Competition for shelter space causes density-dependent predation in damselfishes.

Holbrook and Schmitt (2002) demonstrated that competition for shelter spaces caused more predation in damselfishes. These species shelter in branching corals or anemones, and when refuge spaces to protect them were filled with their intra-specific competitors, more mortality from predation was found.

Cycles. Post et al. (2002) suggest that wolves cause moose population on Isle Royale (island in Lake Superior) to cycle. Although the data do suggest that moose populations have peaked twice in the interval 1958-1998, claiming that wolf populations are responsible is not defensible. Rather, there does appear to be a correlation between these populations. However, many different variables, particularly weather variables, could be found to correlate with these populations, and most investigators would not claim a cause-and-effect relationship had been found.

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