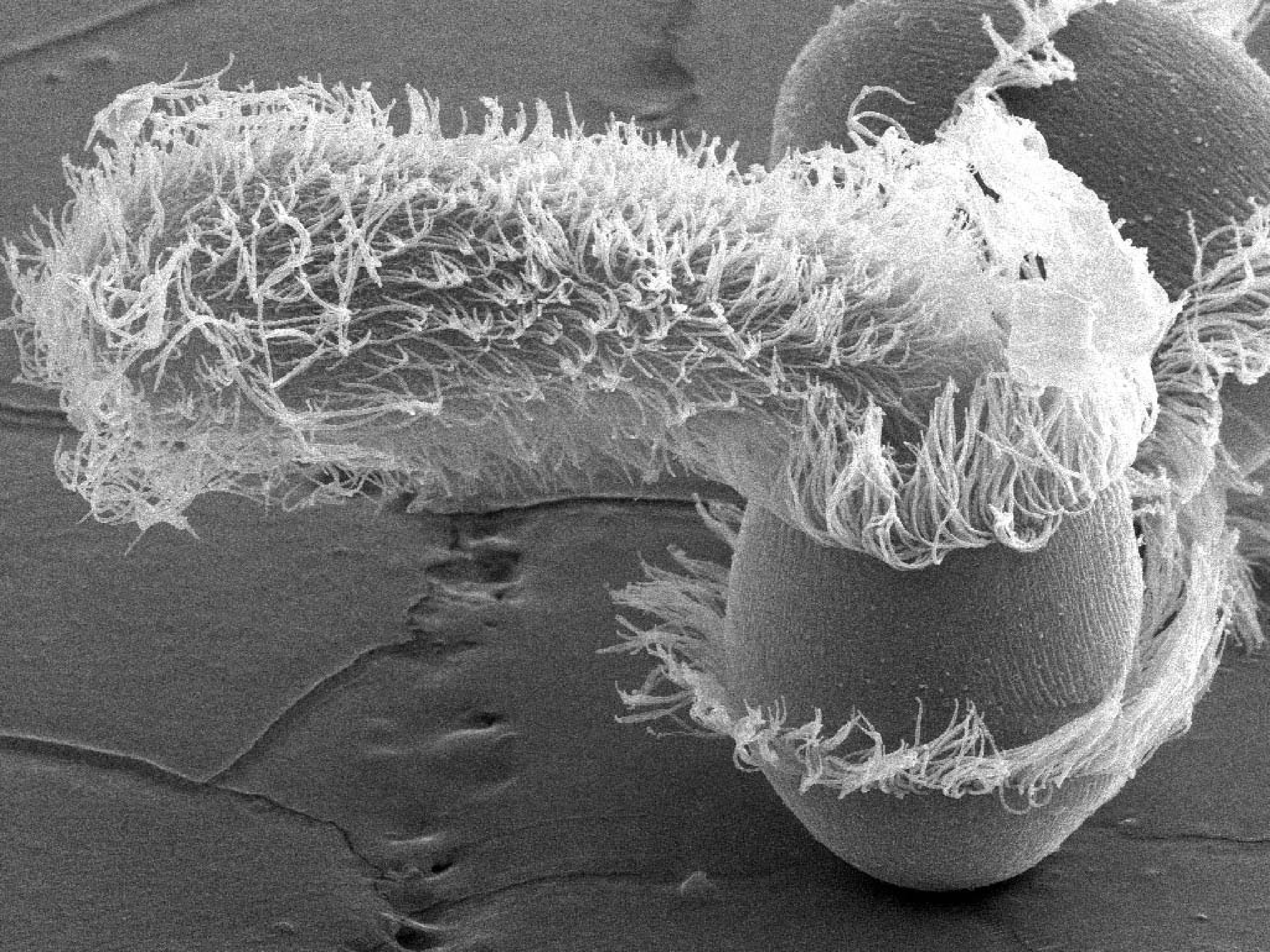

© QT Luong / terragalleria.com



Predation

- Definitions
- Examples
- Functional Response
- Numerical Response
- Simple predator-prey models
- Complex interactions
  - trophic cascades
  - hyperpredation and subsidies
  - indirect effects (the ecology of fear)

Definitions-eating of one species by another…

- **Herbivory** -animals feeding on green plants
- **Carnivory** -animals feeding on other animals
- **Parasitism** a) Animals (or plants) feeding on other organisms without killing them and b) **parasitoids**, usually insects, laying eggs on hosts which are completely consumed by developing larvae
- **Cannibalism-** intraspecific predation

Direct effects:Examples from Krebs Ecology text 2001

- Gause (laboratory)
- Huffaker (laboratory)
- host-parasitoid cycles in laboratory
- Duck hatching rates with and without skunks
- Red kangaroos and dingos in Australia (116x increase; emus 20x increase)
- Lake trout in the Great Lakes

Direct effects

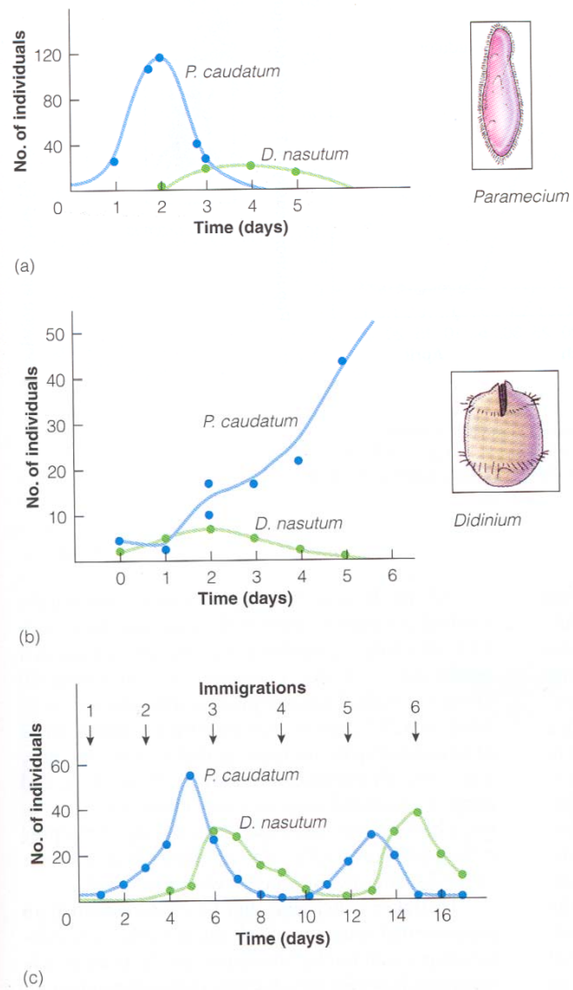


FIGURE 13.7
Predator-prey interactions between the protozoans Paramecium caudatum *and* Didinium nasutum *in three microcosms: (a) oat medium without sediment, (b) oat medium with sediment, and (c) oat medium without sediment and with immigration. (After Gause 1934.)*

Direct effects

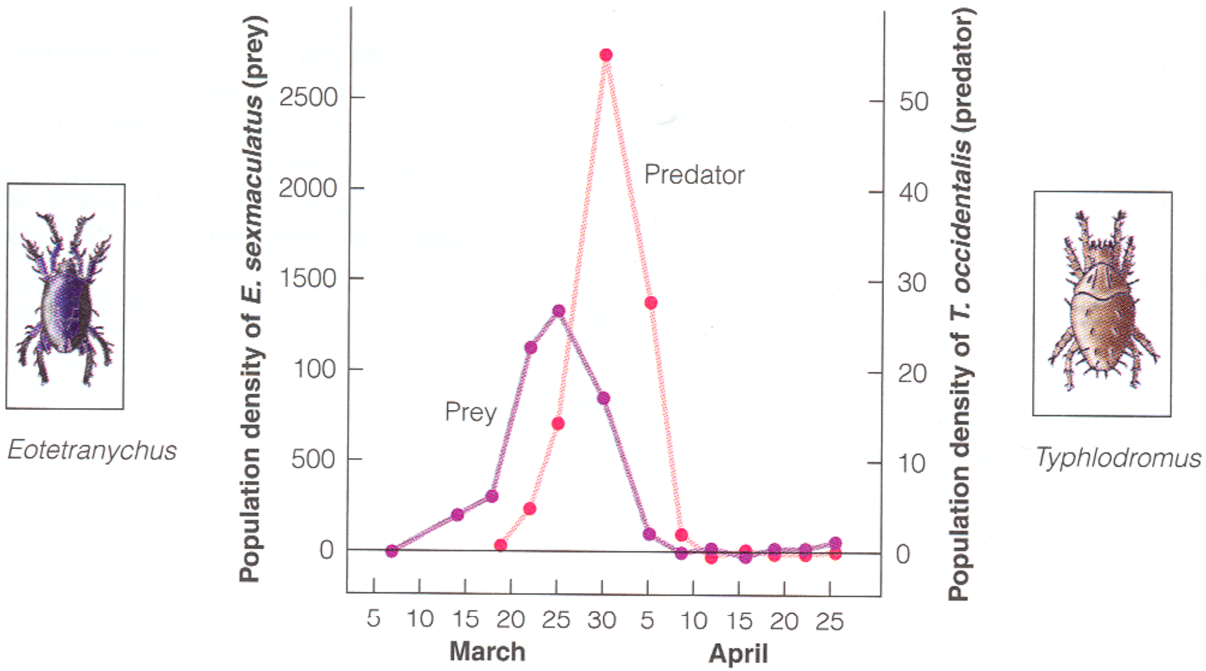


FIGURE 13.8
Densities (per unit area of orange) for the prey mite Eotetranychus sexmaculatus *and the predator mite* Typhlodromus occidentalis, *with 40 oranges, 20 of which provided food for the prey alternating with 20 foodless (covered) oranges. (After Huffaker 1958.)*

Direct effects

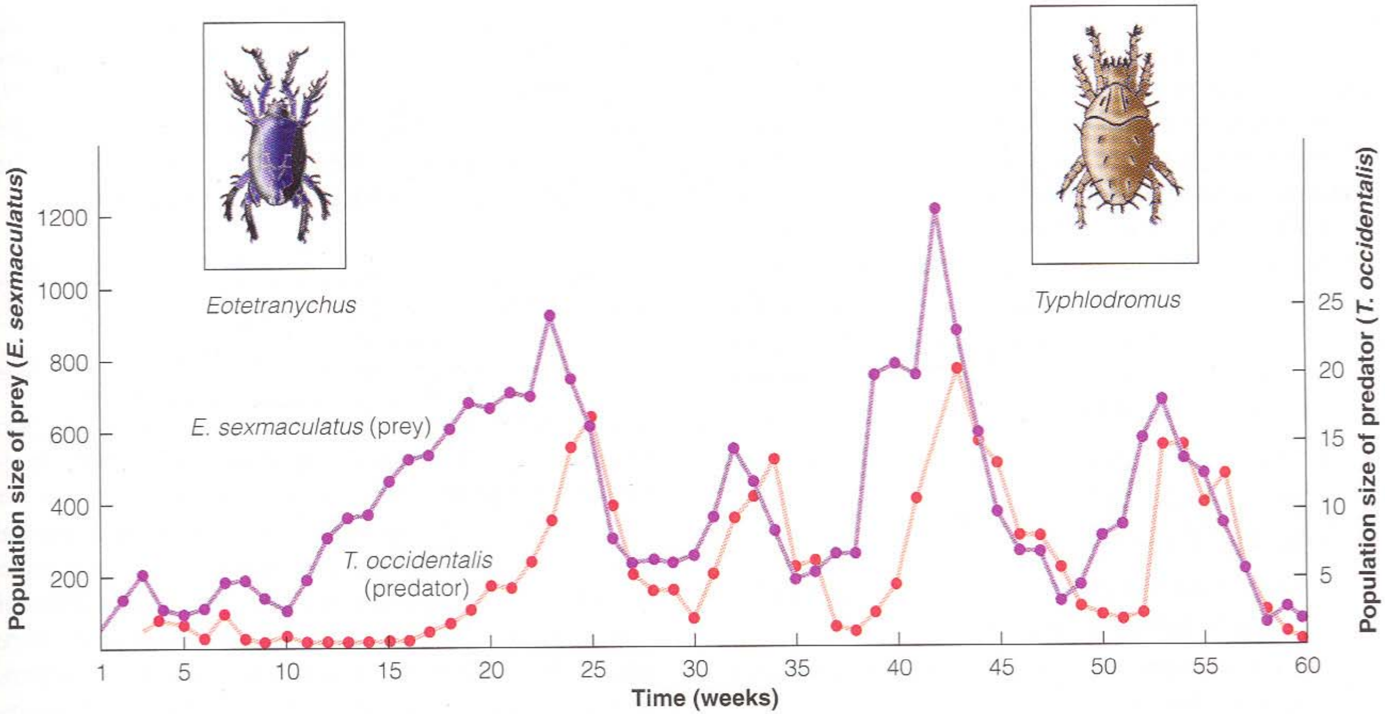
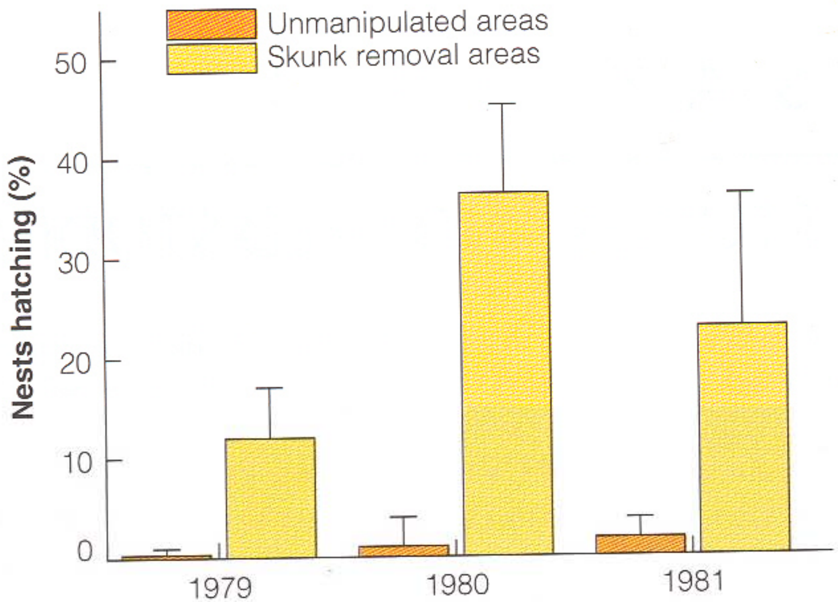


FIGURE 13.9

Predator-prey interaction between the prey mite Eotetranychus sexmaculatus *and the predator mite* Typhlodromus occidentalis *in a complex laboratory environment consisting of a 252-orange system in which one-twentieth of each orange was exposed for possible feeding by the prey. (After Huffaker et al. 1963.)*

Direct effects



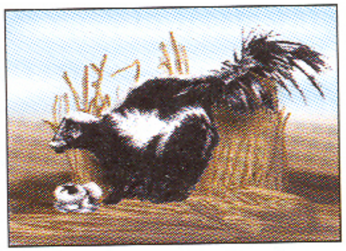


FIGURE 13.11
Mean hatching rates of upland duck nests in waterfowl areas of North Dakota from which striped skunks (Mephitis mephitis) *were removed during the nesting season, April–July 1979–1981. Skunk removal dramatically improved duck nesting success. (Data from Greenwood 1986, Table 3.)*

Direct effects

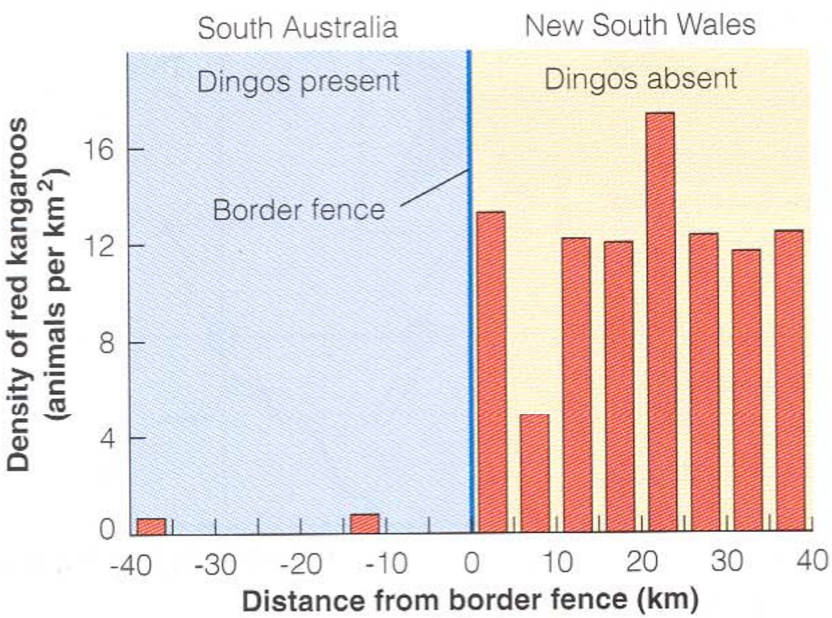


FIGURE 13.12
Density of red kangaroos on a transect across the New South Wales-South Australia border in 1976. The border is coincident with a dingo fence that prevents dingos from moving from South Australia into the sheep country of New South Wales. (After Caughley et al. 1980.)

Direct effects

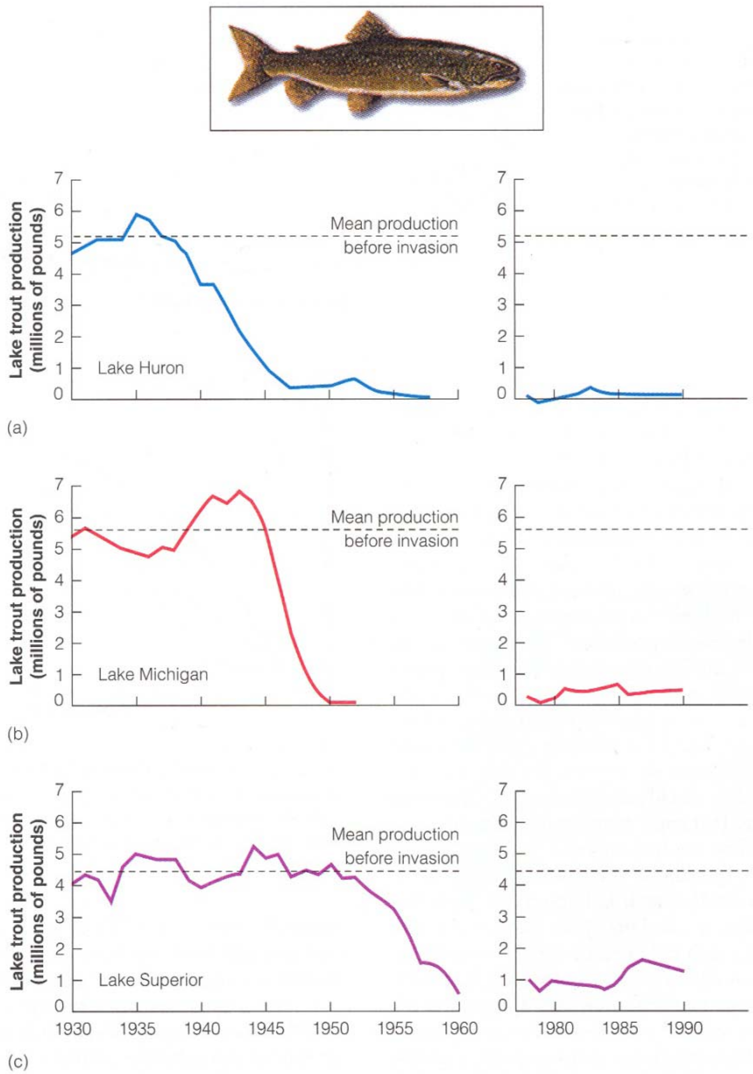


FIGURE 13.14
Effect of sea lamprey introduction on the lake trout fishery of the upper Great Lakes of North America. Lampreys were first seen in (a) Lake Huron in 1937, (b) Lake Michigan in 1936, and (c) Lake Superior in 1938. Commercial fish production from 1978 to 1990 is shown in the right panel. Lake trout have not recovered in the Great Lakes in spite of sea lamprey control. (Data from the Great Lakes Fishery Commission.)

Examples

- But, in many other cases, little evidence of population response of prey to predators
  – Populations of large mammals on Serengeti Plains appear to be weakly affected by a large suite of dramatic predators (lions, leopards, cheetahs, wild dogs, spotted hyenas).
  – Why?

Predator-prey evolutionary “arms race”

- Predation is a strong ecological and evolutionary force
- If predator is too efficient, it will consume all of it’s prey. If predator is too inefficient, it will starve.
- Dawkins and Krebs (1979): race between fox and rabbit
  - if fox losses, can still reproduce if it doesn’t eat the *particular* rabbit it’s chasing
  - if the rabbit loses...

Definitions

- Pimm (1979, 1980)
  - **Donor-controlled system**, prey supply is controlled by factors other than predators
    - Errington (1963) suggested that in some systems, predators were merely the "executioners" removing doomed individuals
    - Migration of large mammals on Serengeti Plains
  - **Predator-controlled system**-predators affect prey population growth rate
    - Dingos and kangaroos

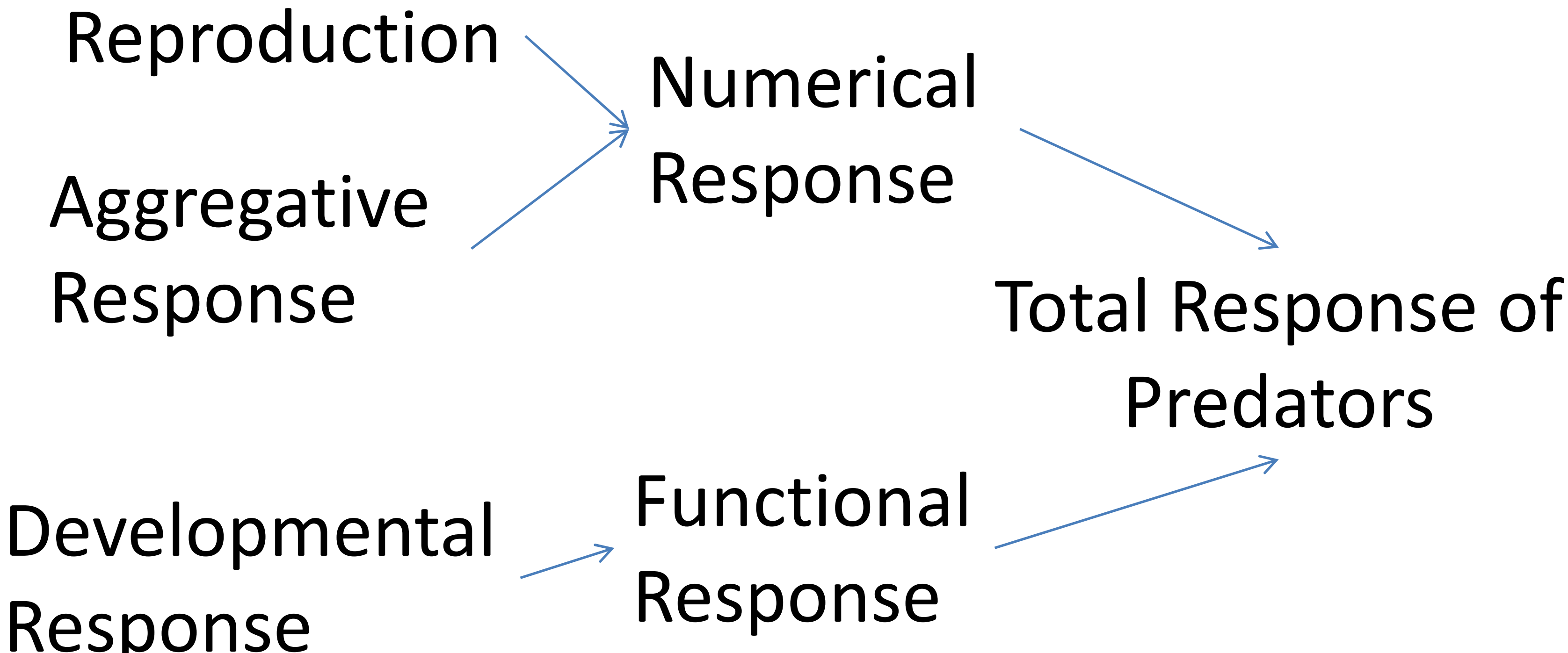
Predator-prey models

- What, specifically, is it about some predator-prey systems that makes predators effective in limiting prey populations?
- Can we predict how prey and predator populations will change with changing prey and predator density?

Buzz Holling (1959)

- How might predators respond to an increase in prey population density?
  - one predator, one prey system
- Potential responses:
- **Numerical response**, change in the density of predators in an area
- **Functional response,** change in the consumption (kill) rate by predators

Holling (1959)

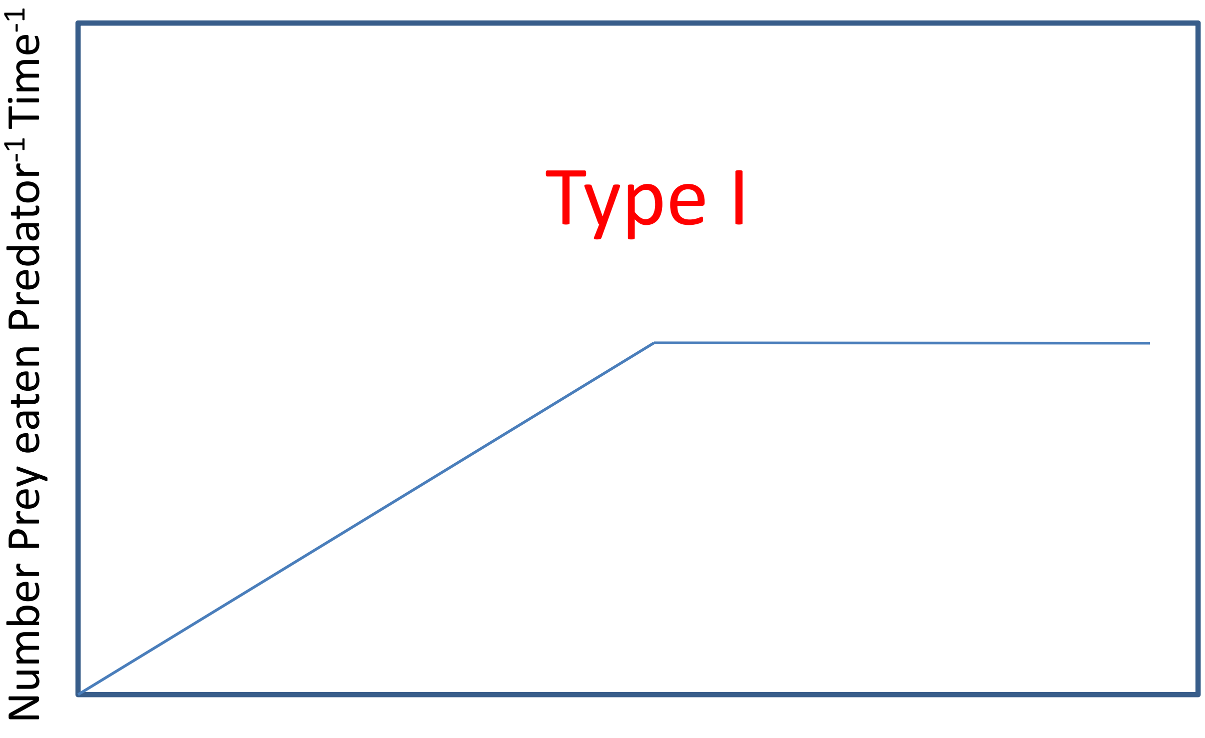


Functional Response Curves

- Kill rate / predator = f(search time, handling time, satiation)

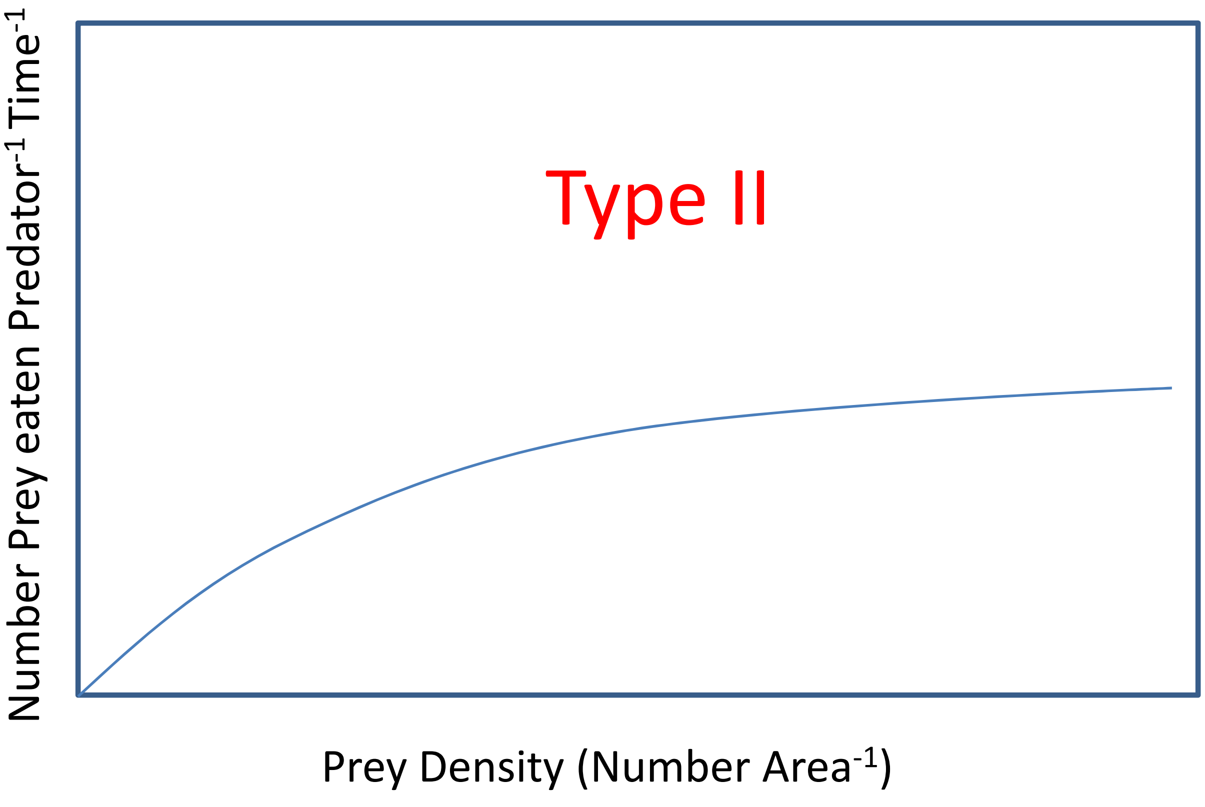
Functional Response Curves

- Kill rate / predator = f(search time, handling time, satiation)



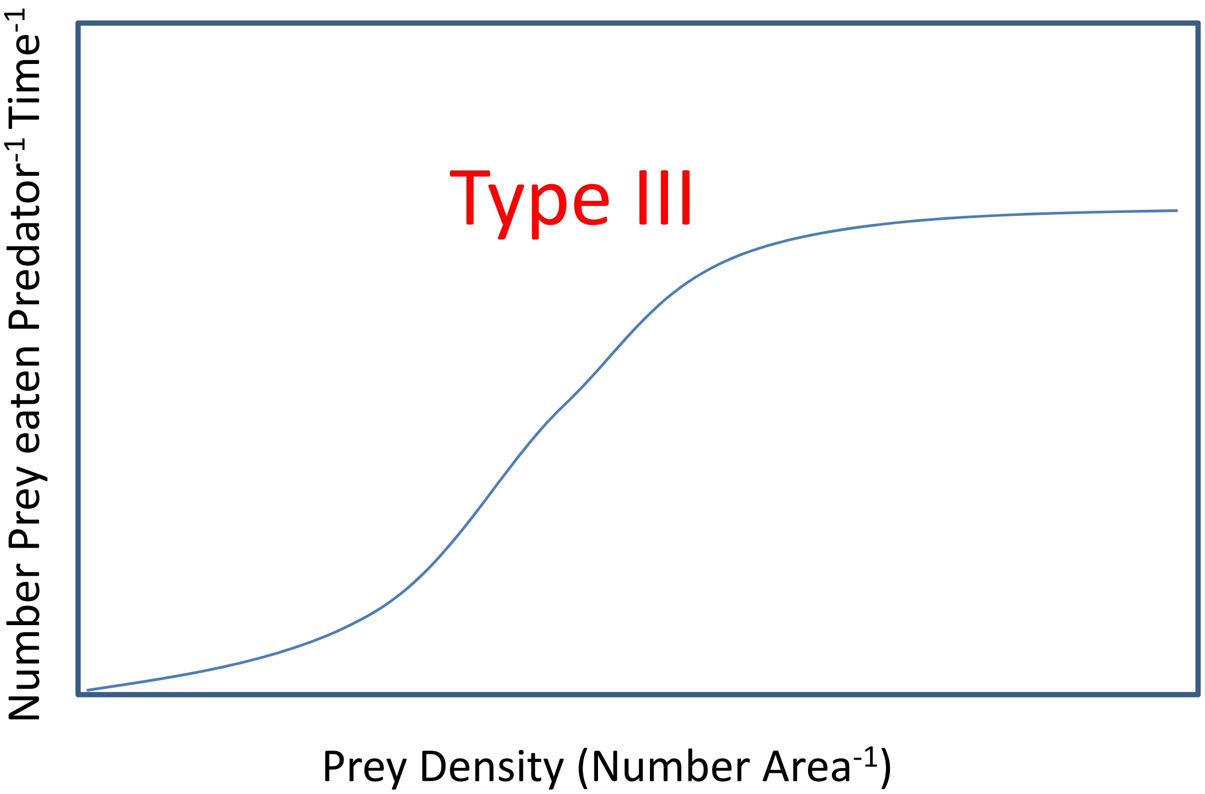
Functional Response Curves

- Kill rate / predator = f(search time, handling time, satiation)



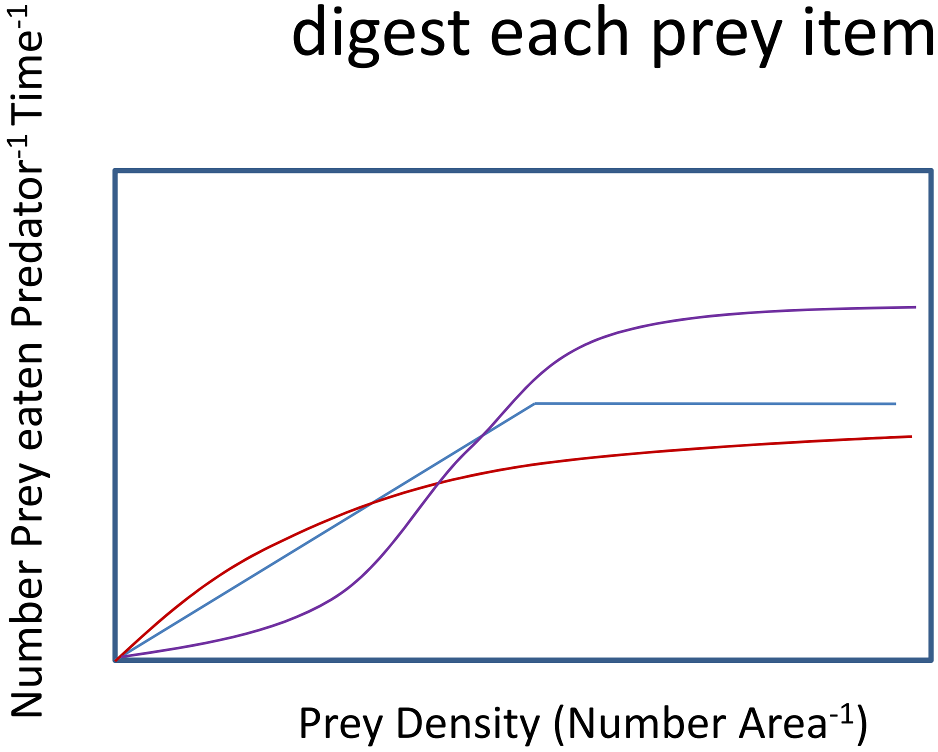
Functional Response Curves

- Kill rate / predator = f(search time, handling time, satiation)



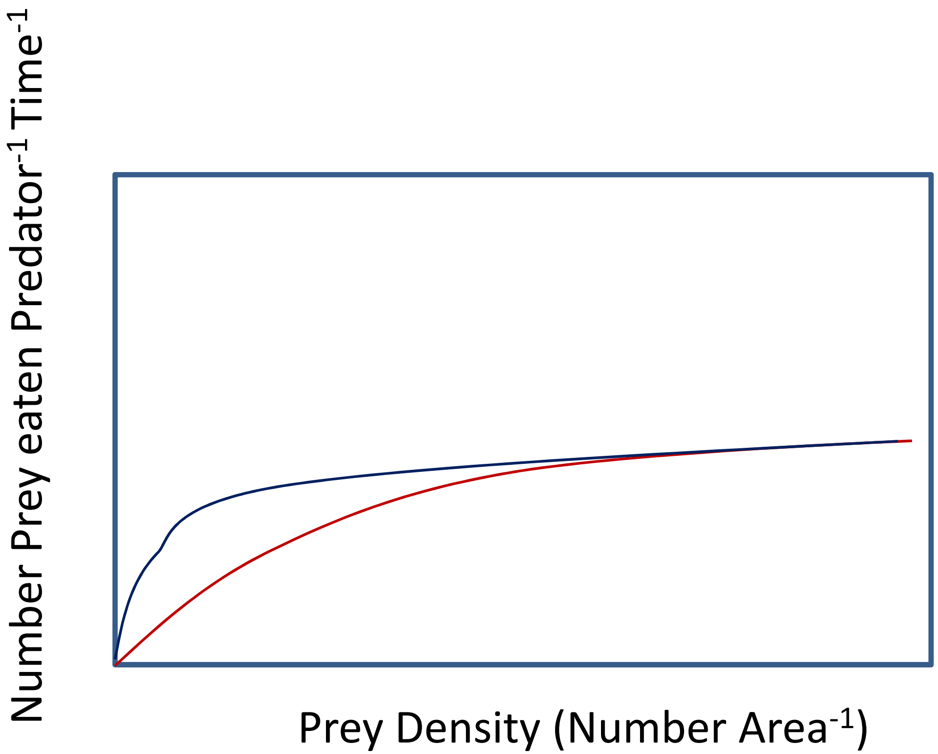
Characteristics

- **Shape** (Type I, II, or III)
- **Maximum consumption rate**
  - -How long it takes to capture, subdue, consume and digest each prey item



Characteristics

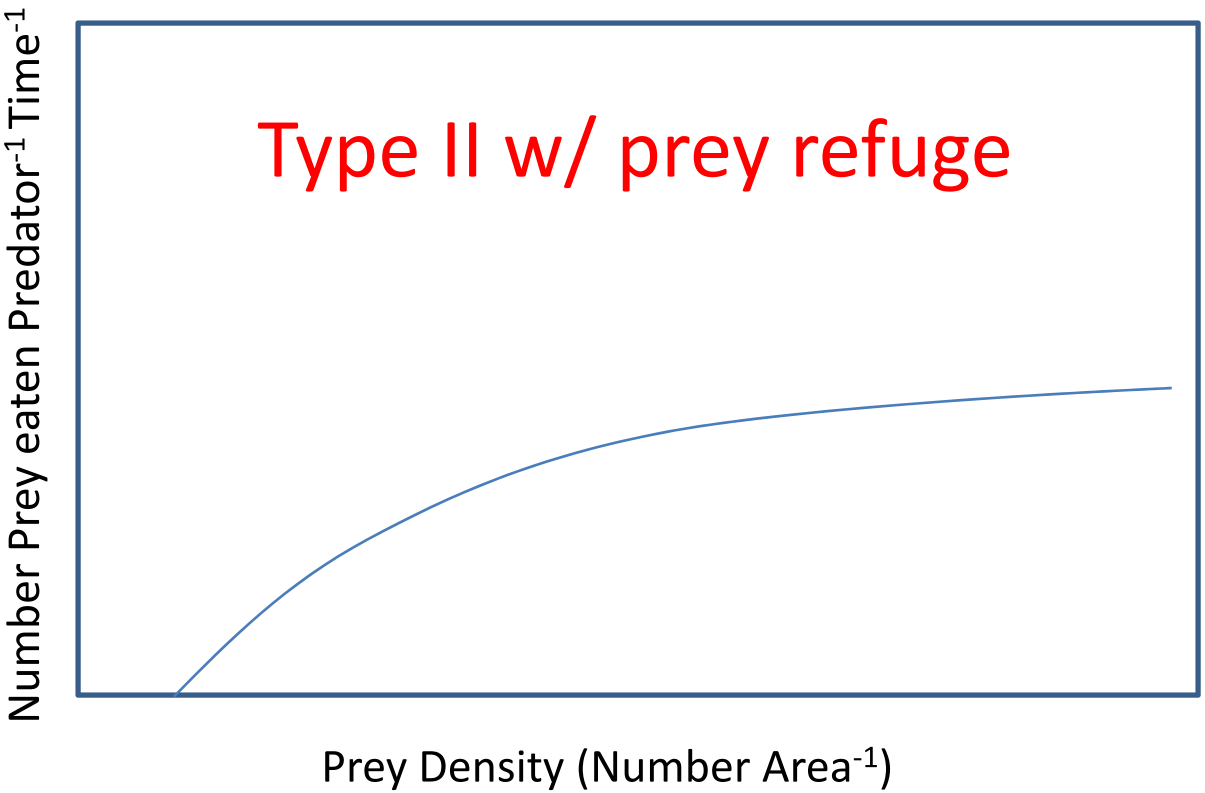
– **Rate of increase:**



– Predator skill in
  - searching
  - and catching prey
– Prey ability to remain hidden and escape when detected
– Depends on predator's decisions about where and how much time to spend searching for prey

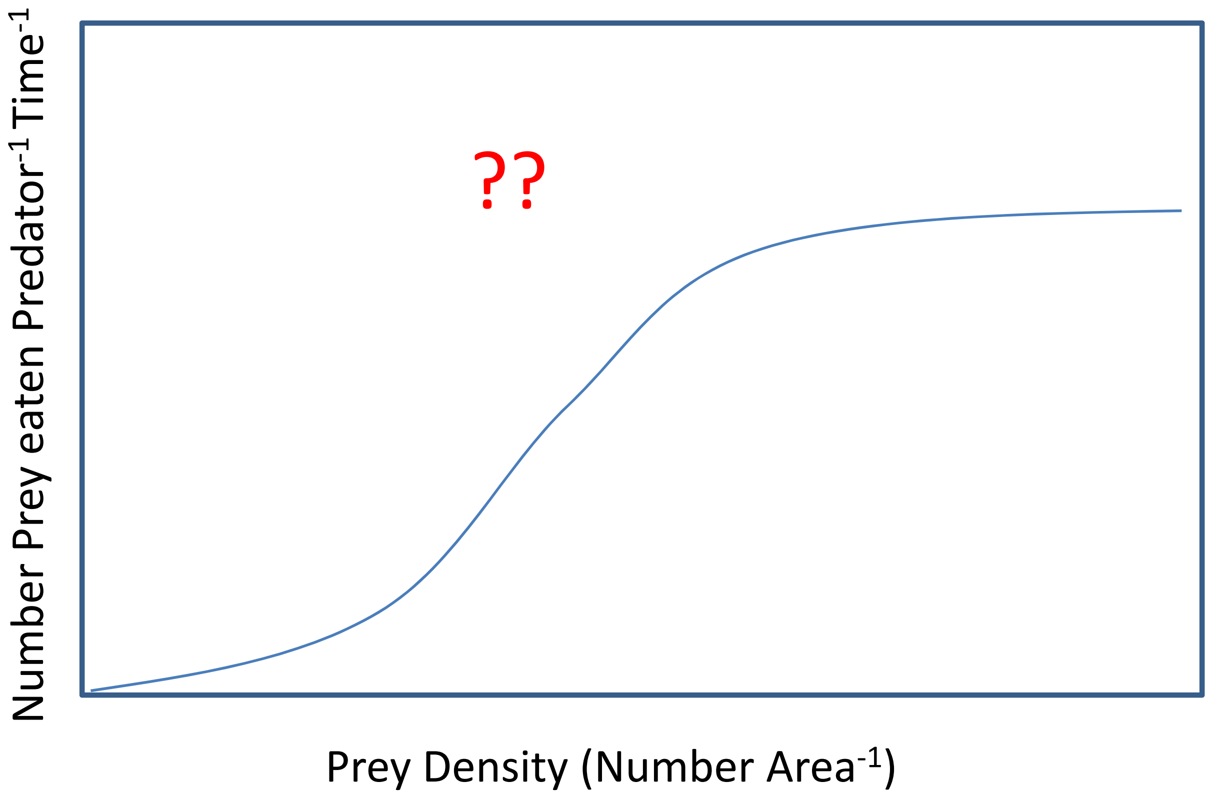
Functional Response Curves

- Kill / predator rate = f(search time, handling time, satiation)



Functional Response Curves

- Kill / predator rate = f(search time, handling time, satiation)



Combined response curves

- Murdoch and Sih (1978, in Krebs 2001), *Notonecta*
- Mills figure 8.1

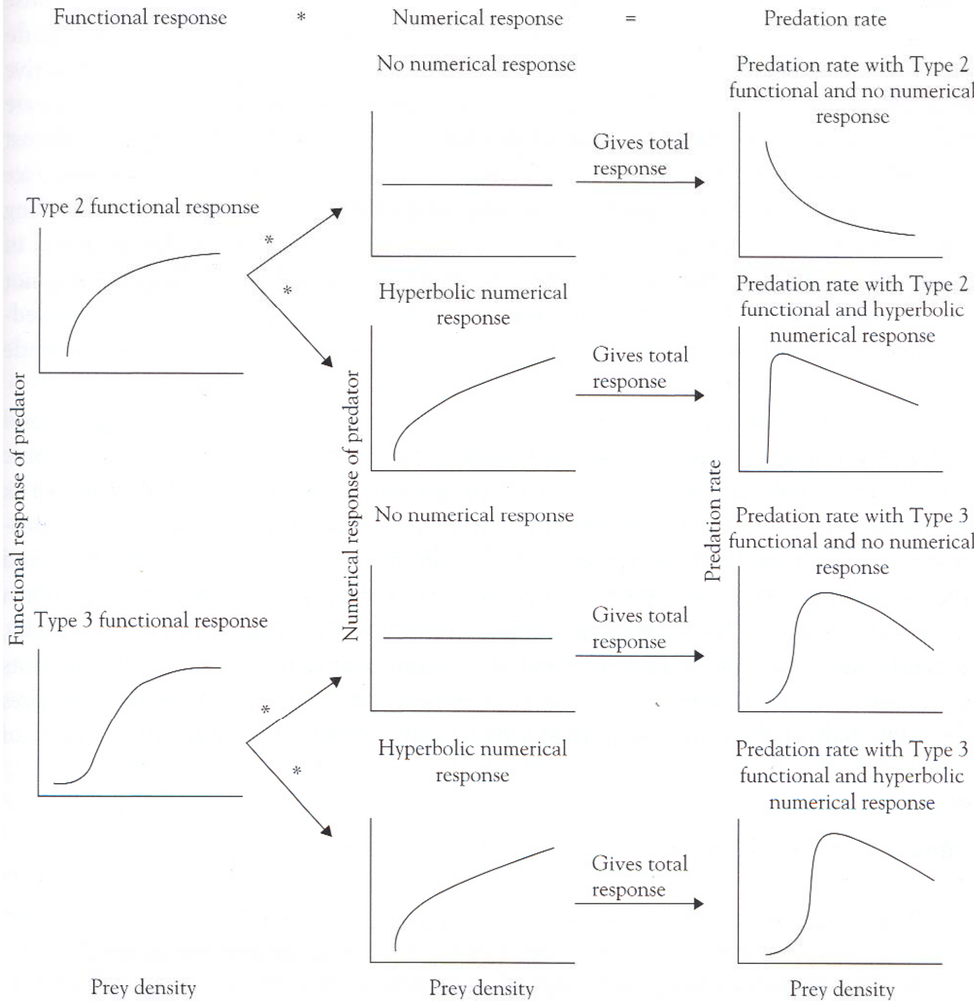


Fig. 8.1 How a predator's functional and numerical response can combine to affect predation rate on a prey population, using as examples curves derived from analyses on wolves and moose (Messier 1994, 1995). The left-hand panels are functional response curves (moose kills per wolf per 100 days) that are either Type 2 or Type 3. Each functional response is multiplied by a numerical response (middle panels; wolves per 1000 km^2) that is either constant (unresponsive to density) or a hyperbolic increase resulting from local births and/or an aggregative response by the predator. The right-hand panels show the predation rate (percentage of moose population killed by wolves per year).

How do predators and prey numbers change through time?

- Lotka-Volterra Model
- Assumptions
  - No time lags
  - Environment is homogeneous
  - Predator density does not effect functional response curve (probability of being eaten)
  - No density dependence in predator population

How do predators and prey numbers change through time?

- Lotka-Volterra Model
- Assumptions
  - In the absence of predators, prey grow exponentially (no density dependence)
  - In the absence of prey, the predator dies off exponentially
  - The functional response is Type 1 with no maximum kill rate
  - Each prey death contributes identically to the growth of the predator population

How do predators and prey numbers change through time?

- Lotka-Volterra Model
- Assumptions
  - In the absence of predators, prey (H) grow exponentially a rate *r:*

$$dH/dt = r H$$

How do predators and prey numbers change through time?

- Lotka-Volterra Model
- Assumptions
  - In the absence of prey, the predator (P) dies off exponentially at a rate *k*:

$$dP/dt = -k\,P$$

How do predators and prey numbers change through time?

- Lotka-Volterra Model
- Assumptions
  - The functional response (kill rate) is Type 1 (linear with slope *b*) with no maximum kill rate:

$$dH/dt = r\,H - bHP$$

How do predators and prey numbers change through time?

- Lotka-Volterra Model
- Assumptions
  - Each prey death contributes identically to the growth of the predator population

$$dP/dt = cHP - kP$$

Lotka-Volterra Model

$dH/dt = rH - bHP$

H = number of prey
r = prey population growth rate
b = attack rate

$dP/dt = cHP - kP$

P = number of predators
c = predator population growth rate due to predation
k = rate of predator decline in absence of prey

Lotka-Volterra Model

- Can the model explain predator-prey dynamics? (e.g., cycles?).
- Graphical analyses as in competition models

Prey Zero Isocline:

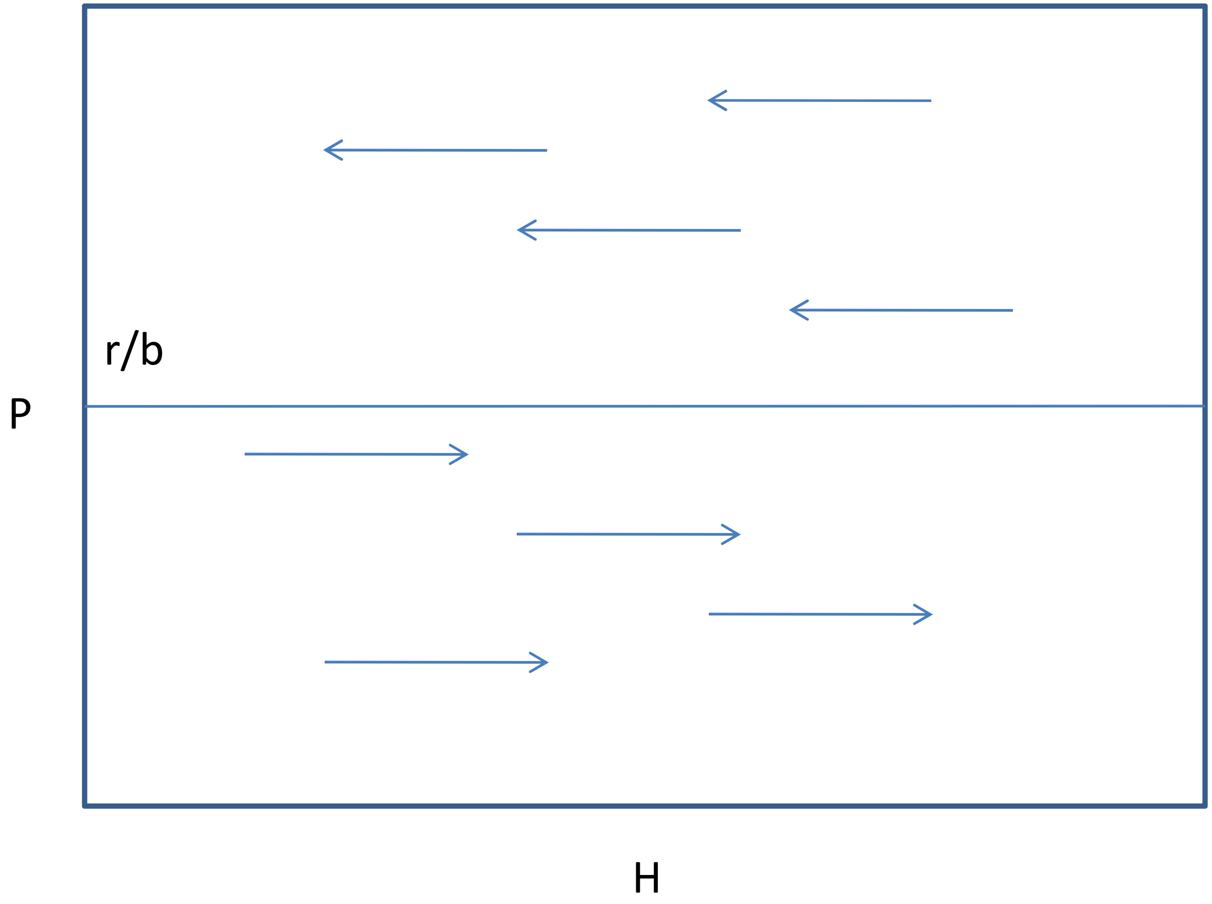
$dH/dt = rH - bHP = 0$

$rH = bHP$

$P = r / b$

Lotka-Volterra Model

Prey Zero Isocline:



Lotka-Volterra Model

- Can the model explain predator-prey dynamics? (e.g., cycles?).
- Graphical analyses as in competition models

Prey Isocline:

dH/dt = $r\,H - bHP = 0$

$rH = bHP$

$P = r / b$

Predator Zero Isocline:

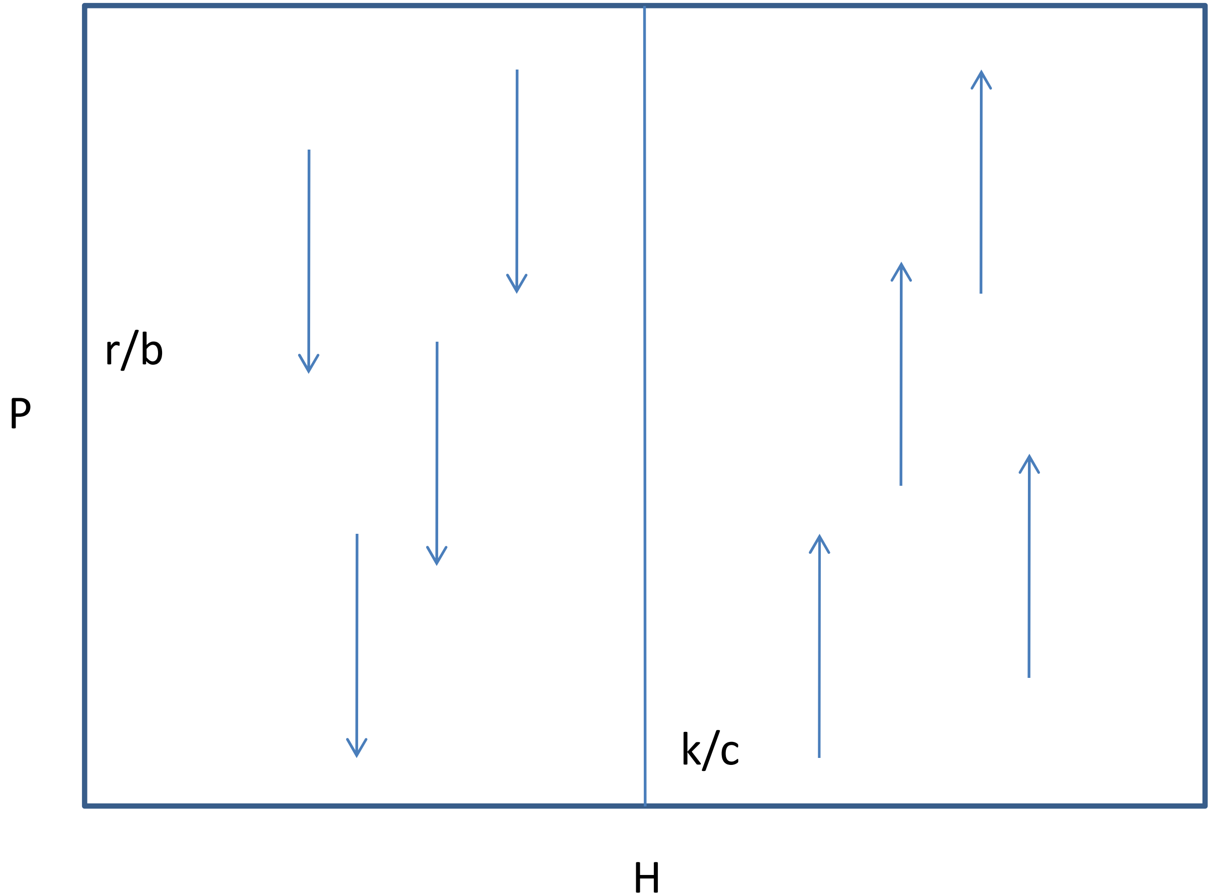
dP/dt = $cHP - k\,P = 0$

$cHP = kP$

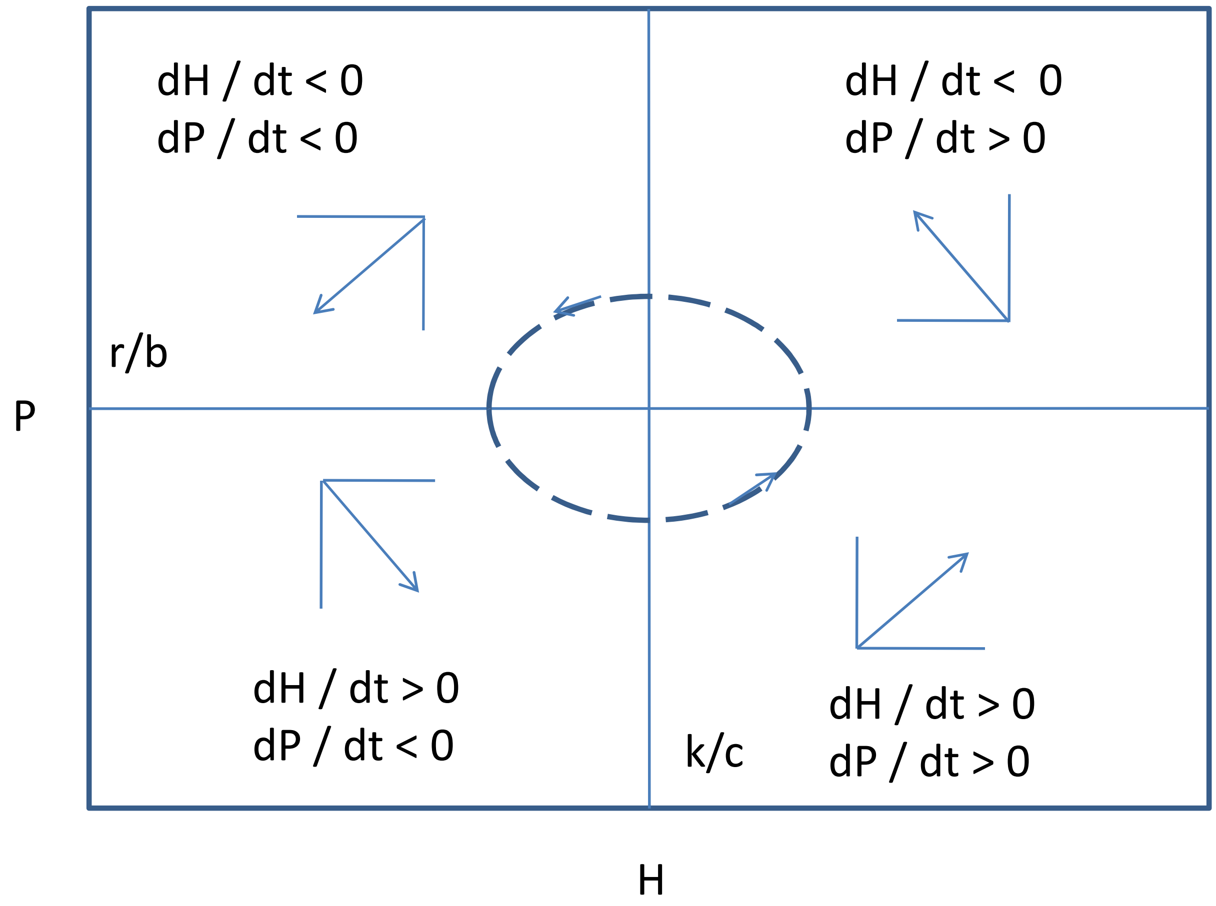
$H = k / c$

Lotka-Volterra Model

Predator Zero Isocline:



Lotka-Volterra Model



How do predators and prey numbers change through time?

- Lotka-Volterra Model—"Most Simple" Model
- Assumptions
  - In the absence of predators, prey grow exponentially (no density dependence)
  - In the absence of prey, the predator dies off exponentially
  - The functional response is Type 1 with no maximum kill rate
  - Each prey death contributes identically to the growth of the predator population

Prey Numbers:

$$dH/dt = rH - bHP$$

H = number of prey

r = prey population growth rate

b = attack rate

Prey Numbers:

$dH/dt = r\,H - bHP$

H = number of prey
r = prey population growth rate
b = attack rate

$dH/dt = r\,H$

Logistic growth in prey in absence of predators

$dH/dt = r\,H\,(1 - H/K)$

Prey Numbers:

$dH/dt = rH - bHP$

H = number of prey
r = prey population growth rate
b = attack rate

$dH/dt = rH(1-H/K) - bHP$

Functional response:

$dH/dt = -bHP \quad dH/dt\ 1/P = -bH$

Prey Numbers:

$dH/dt = r\,H - bHP$

H = number of prey
r = prey population growth rate
b = attack rate

$dH/dt = r\,H\,(1 - H/K) - bHP$

Functional response:

$dH / dt = -bHP \qquad dH / dt \;\; 1/P = -bH$

Holling's Type II: $1/P = -aH / (1 + aHh)$

Prey Numbers:

$dH/dt = r\,H - bHP$

H = number of prey

r = prey population growth rate

b = attack rate

$dH/dt = r\,H\,(1-H/K) - bHP$

Combined prey response w/

Prey logistic growth

Predator Type II functional resp.:

$dH\,/\,dt = r\,H\,(1-H/K) - aHP\,/\,(1+aHh)$

Predators rate of change?

$$dP/dt = cHP - k P$$

P = number of predators
c = predator population growth rate due to predation
k = rate of predator decline in absence of prey

Number of predators depends on prey numbers. Assume that predator growth is logistic with number of prey setting K

Let J = prey density required to support 1 predator per unit area

Predators rate of change?

$dP/dt = cHP - k P$

P = number of predators
c = predator population growth rate due to predation
k = rate of predator decline in absence of prey

Set predator carrying capacity to H / J

Logistic type growth for predators:
$dP/dt = cP (1-P/(H/J)) = cP (1-(PJ/H))$

Predators rate of change?

$dP/dt = cHP - kP$

P = number of predators
c = predator population growth rate due to predation
k = rate of predator decline in absence of prey

Combined Response:
$dP/dt = cP\,(1-(PJ/H)) - kP$

Lotka-Volterra Model

$dH/dt = rH - bHP$

H = number of prey
r = prey population growth rate
b = attack rate

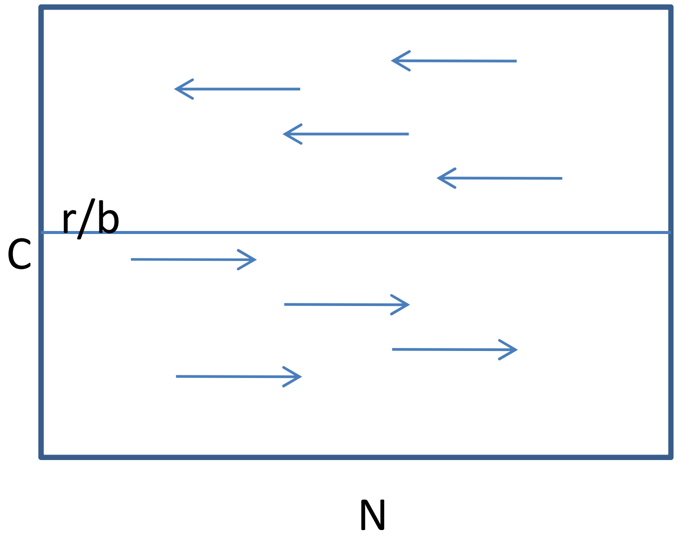
$dP/dt = cHP - kP$

P = number of predators
c = predator population growth rate due to predation
k = rate of predator decline in absence of prey

Graphical analyses:

Refined Prey Zero Isocline w/ density dependent prey population growth

Different predator densities



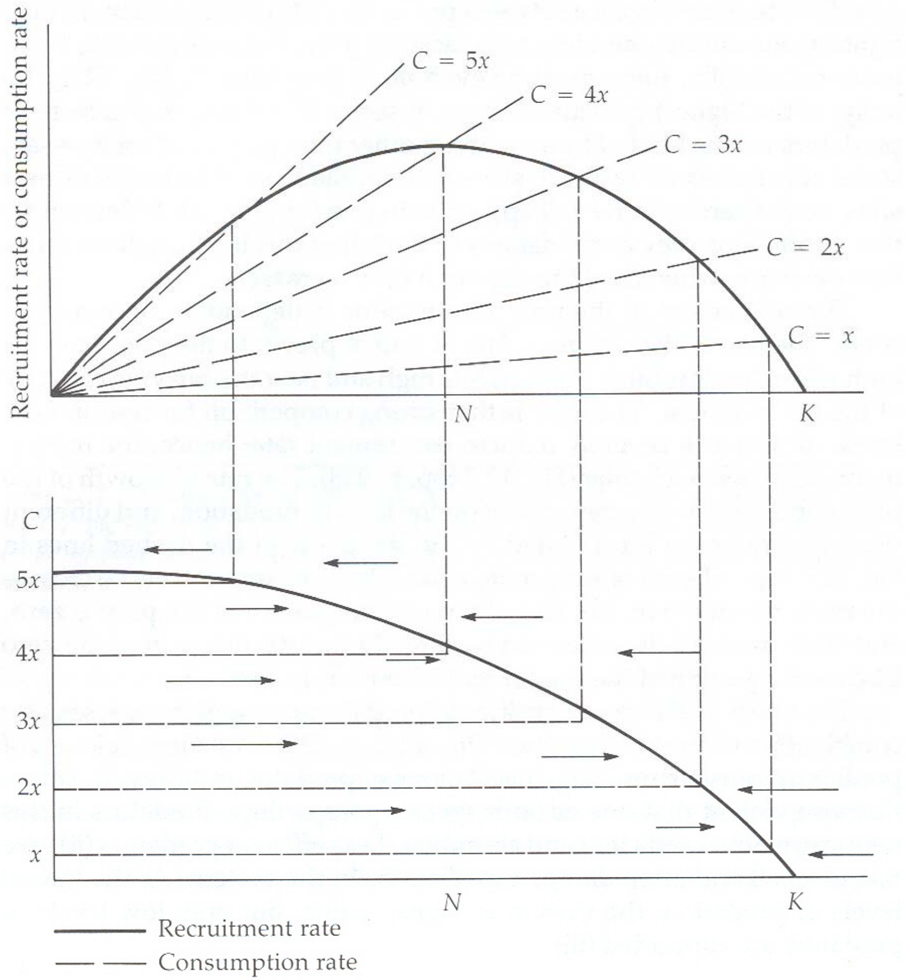


Figure 12.3 Refinement of prey zero isocline. The solid line in the top figure describes variation in prey recruitment rate with density. The dashed lines in the upper figure describe the removal or consumption of prey by predators. There is a family of dashed curves because the total rate of consumption depends on predator density: increasingly steep dashed curves reflect these increasing densities. At the points where a consumption curve crosses the recruitment curve, the net rate of prey increase is zero (consumption equals recruitment). Each of these points is characterized by a prey density and a predator density, and these pairs of densities therefore represent joint populations lying on the prey zero isocline in the bottom figure. The arrows in the lower figure show the direction of change in prey abundance. (Redrawn from Begon, Harper, and Townsend 1986.)

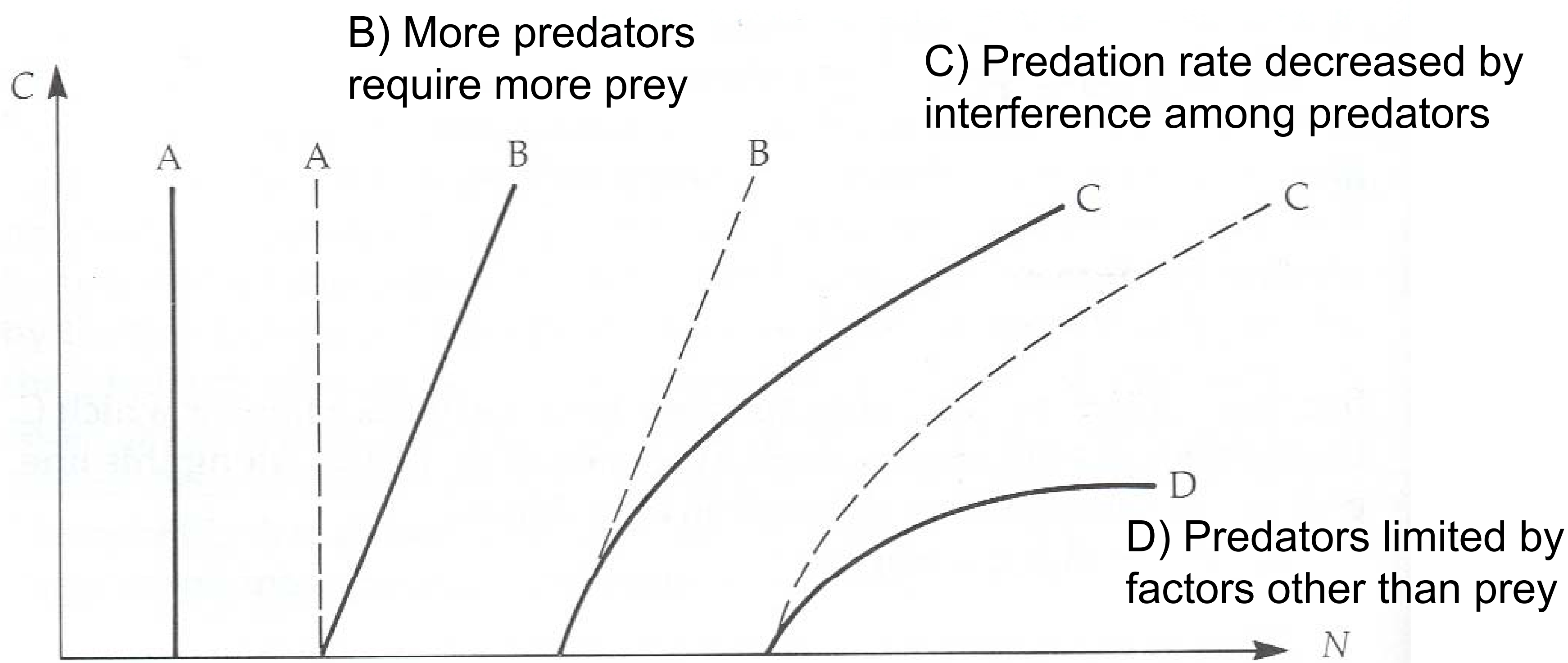


Figure 12.2 Predator zero isoclines of increasing complexity, A to D. A is the Lotka-Volterra isocline. B shows that more predators require more prey. C shows that the consumption rate is progressively reduced by mutual interference among predators. D shows that predators are limited by something other than their food.

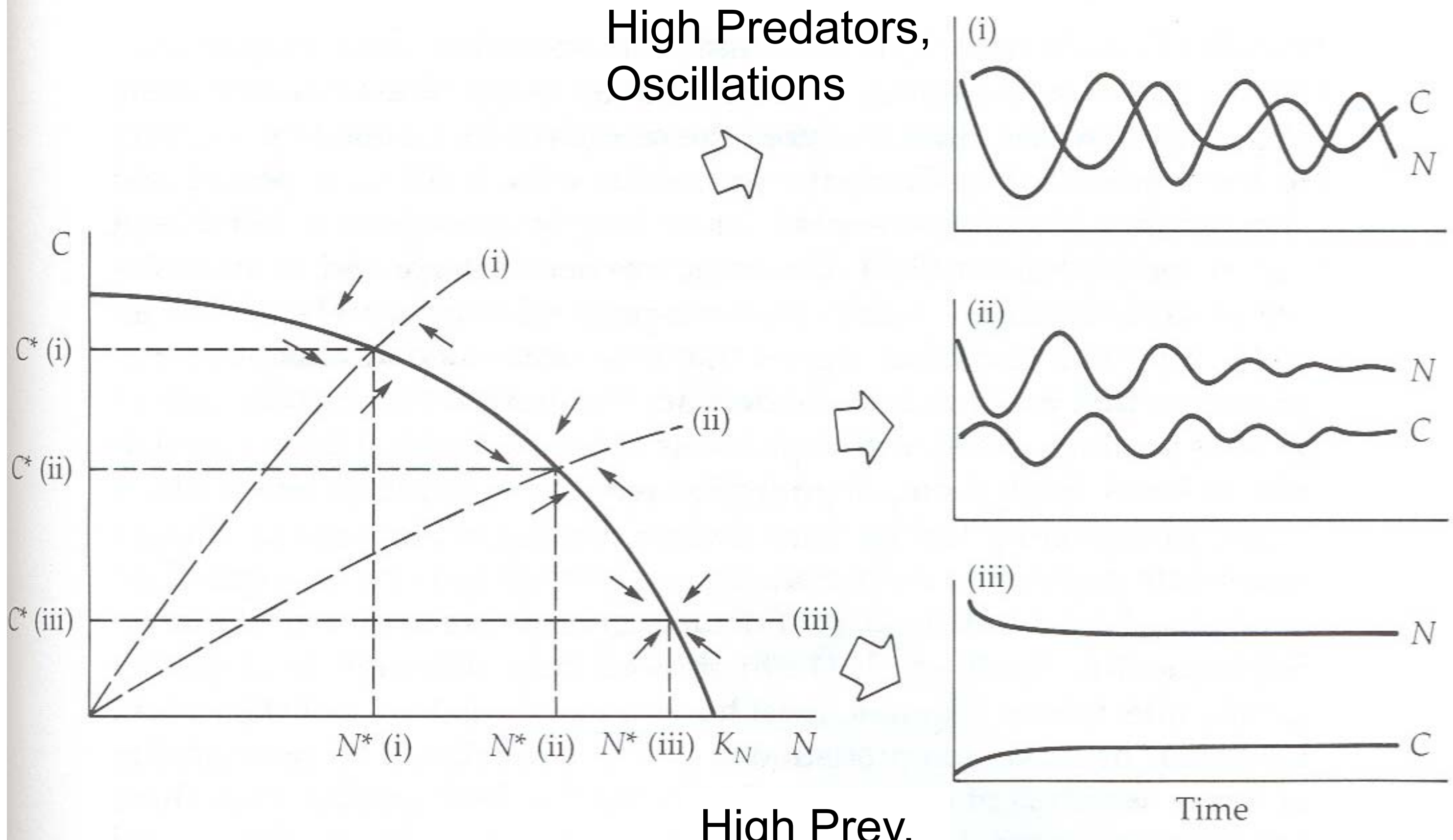


Figure 12.4 A prey zero isocline with se … *ned with predator zero isoclines with increasing levels of self-limitation: (i), (ii), and (iii).* C* is the equilibrium abundance of predators, and N* is the equilibrium abundance of prey. Combination (i) is least stable (most persistent oscillations) and generally has most predators and least prey: the predators are relatively efficient. Less efficient predators (ii) give rise to a lowered predator abundance, an increased prey abundance, and less persistent oscillations. Strong predator self-limitation (iii) can eliminate oscillations altogether, but C* is low and N* is close to K_N. (Redrawn from Begon, Harper, and Townsend 1986.)

Add prey refuges, shift predator zero isoclines to the right

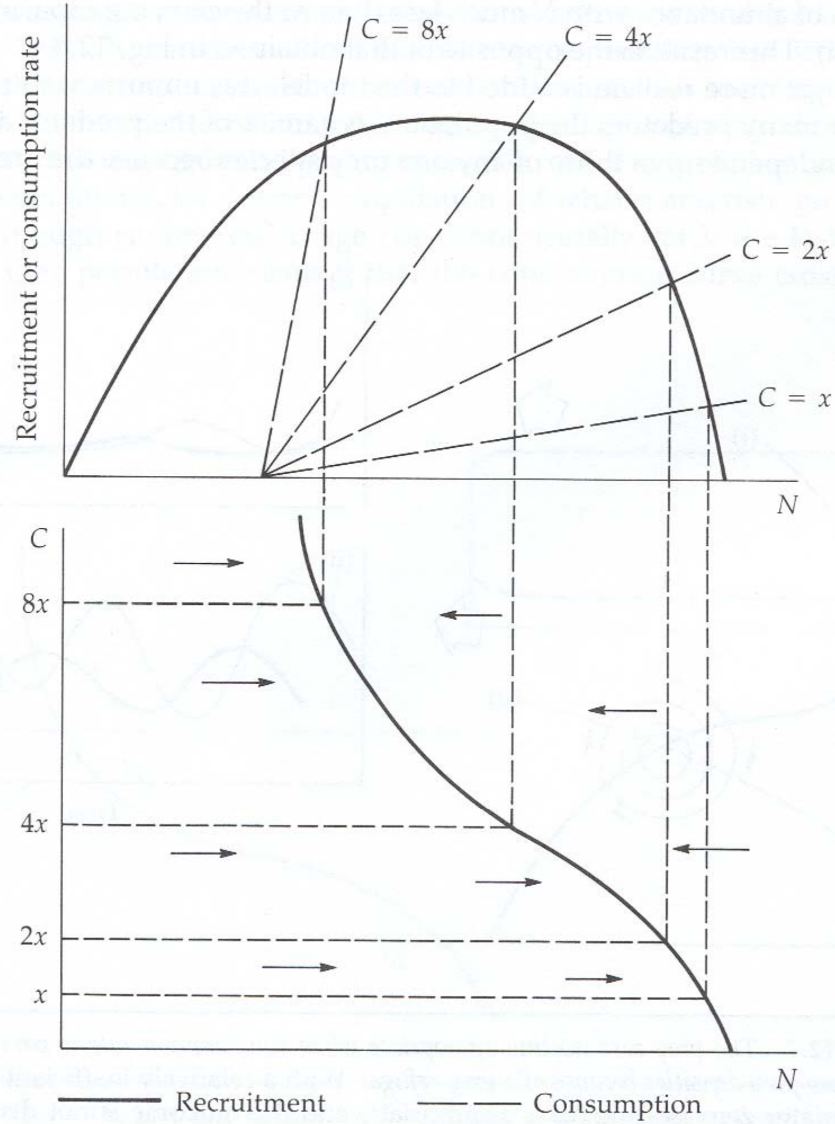
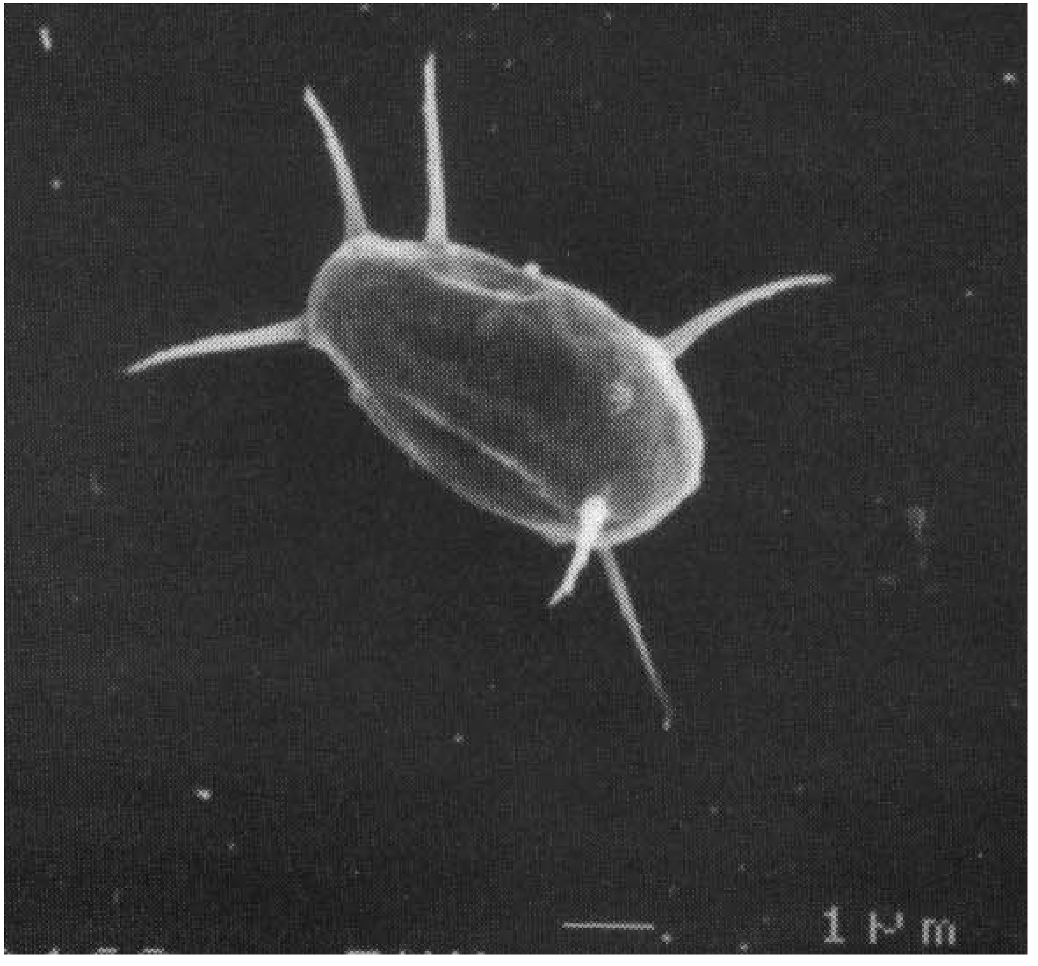
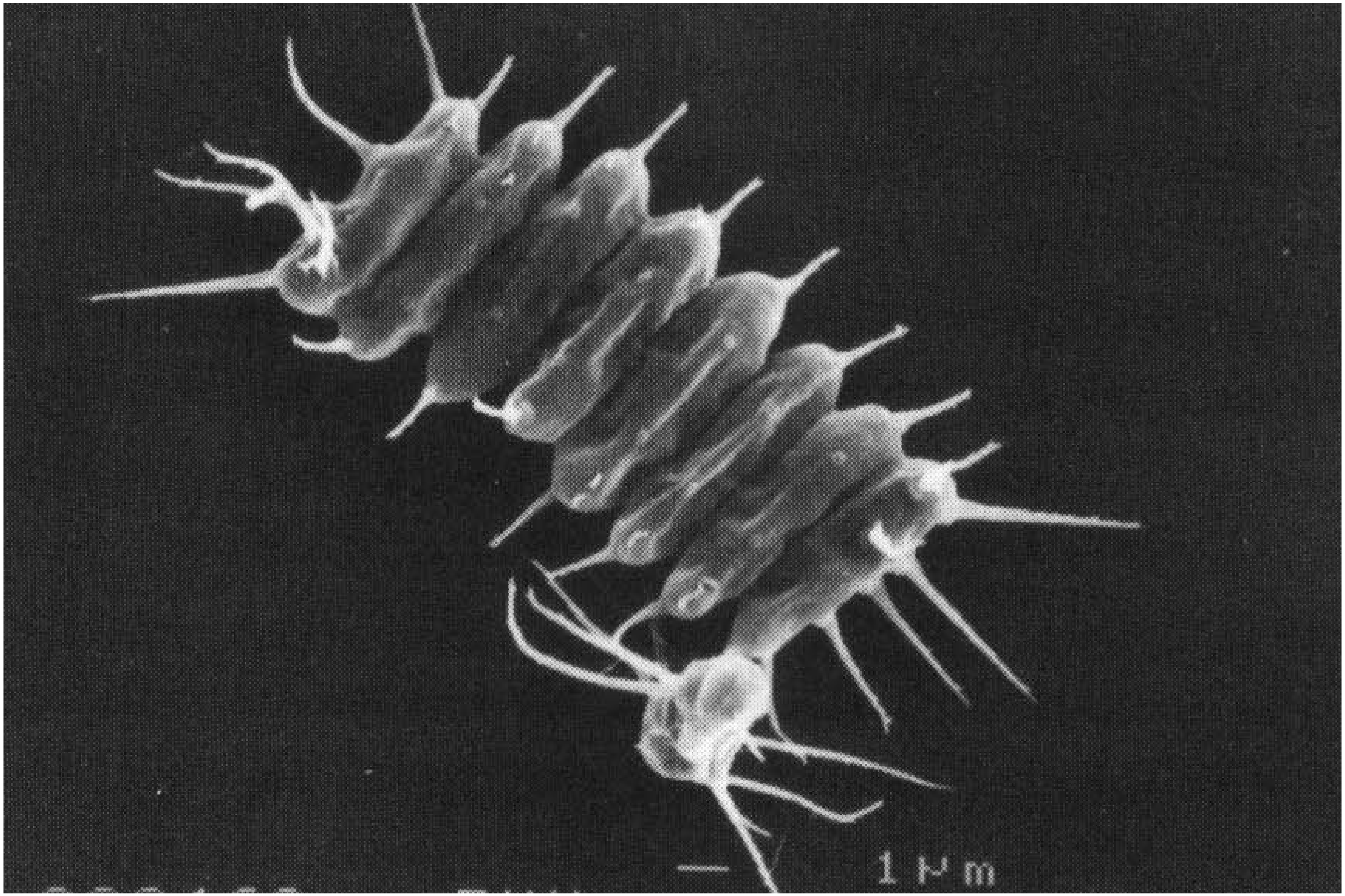


Figure 12.6 Revised prey zero isocline allowing prey refuge from predators. Here the total rate of consumption at low prey densities is zero, irrespective of predator abundance. The reason is a refuge that ensures complete safety of a small number of prey. This phenomenon leads to a vertical prey zero isocline at low densities. (Redrawn from Begon, Harper, and Townsend 1986.)

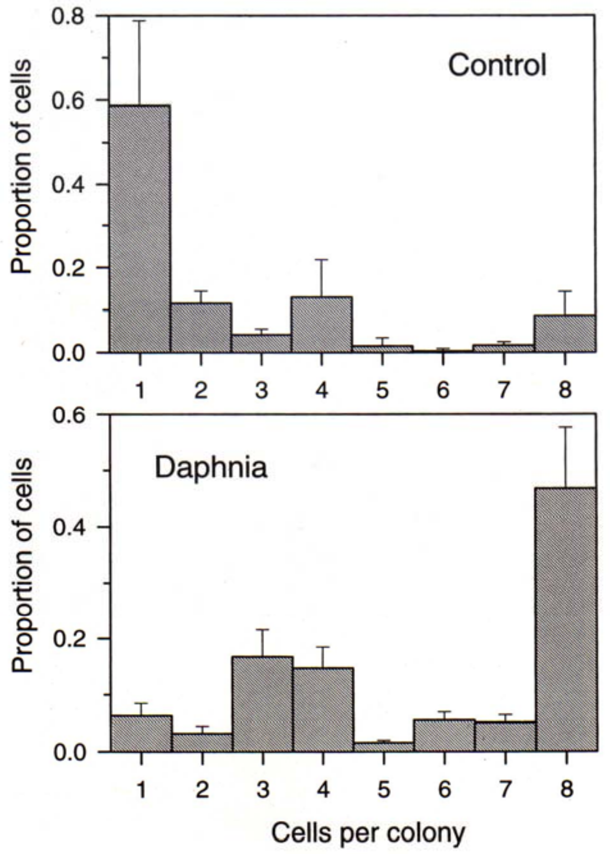
Refuges

- Spatial refuges
  - Burrows
- Morphological defenses
  - phenotypic plasticity: altered phenotype in response to environmental cues
  - Predation as strong selective force


1 μm

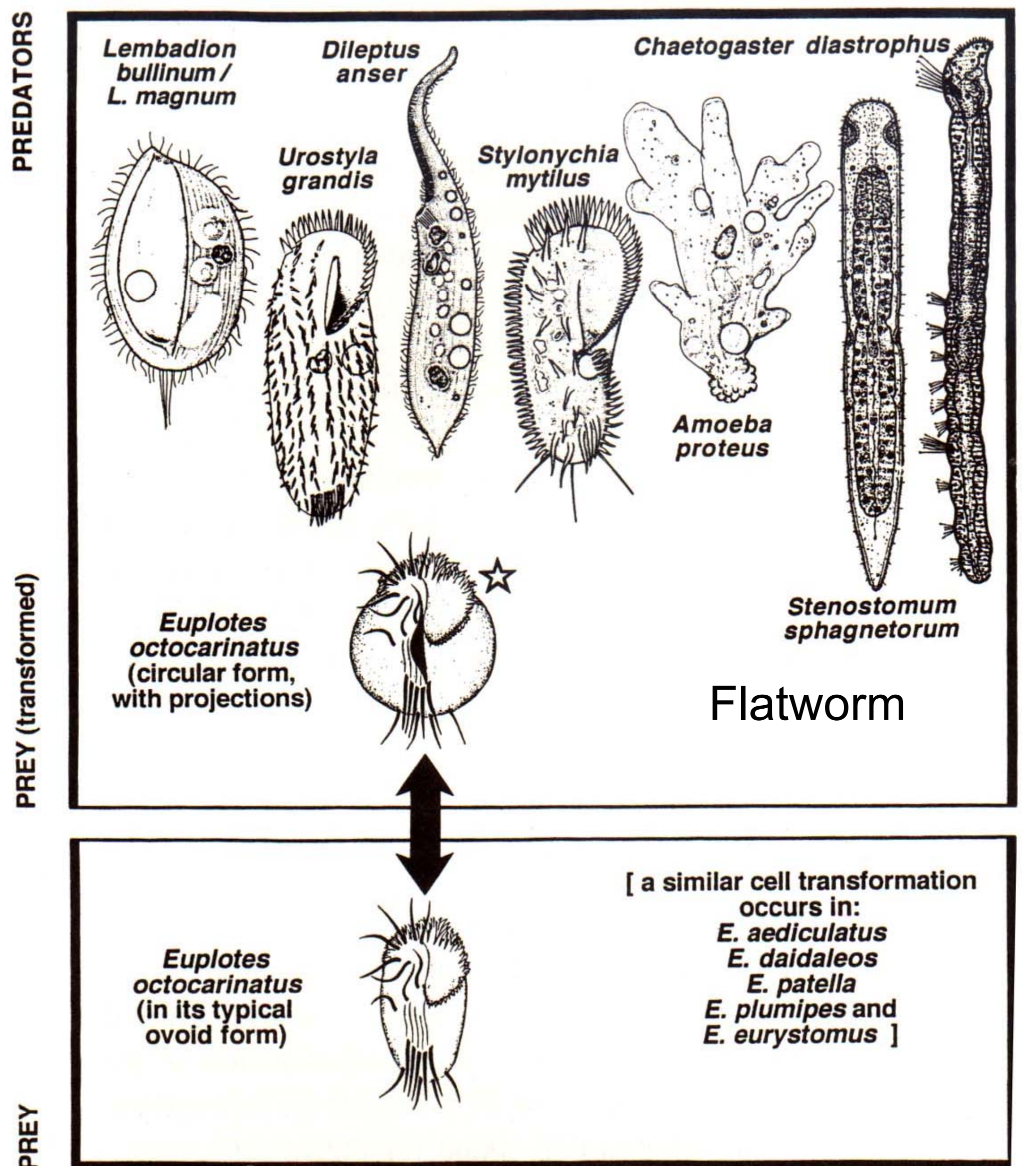

1 μm

Daphnia kairmones induce colony formation in *Scenedesmus*

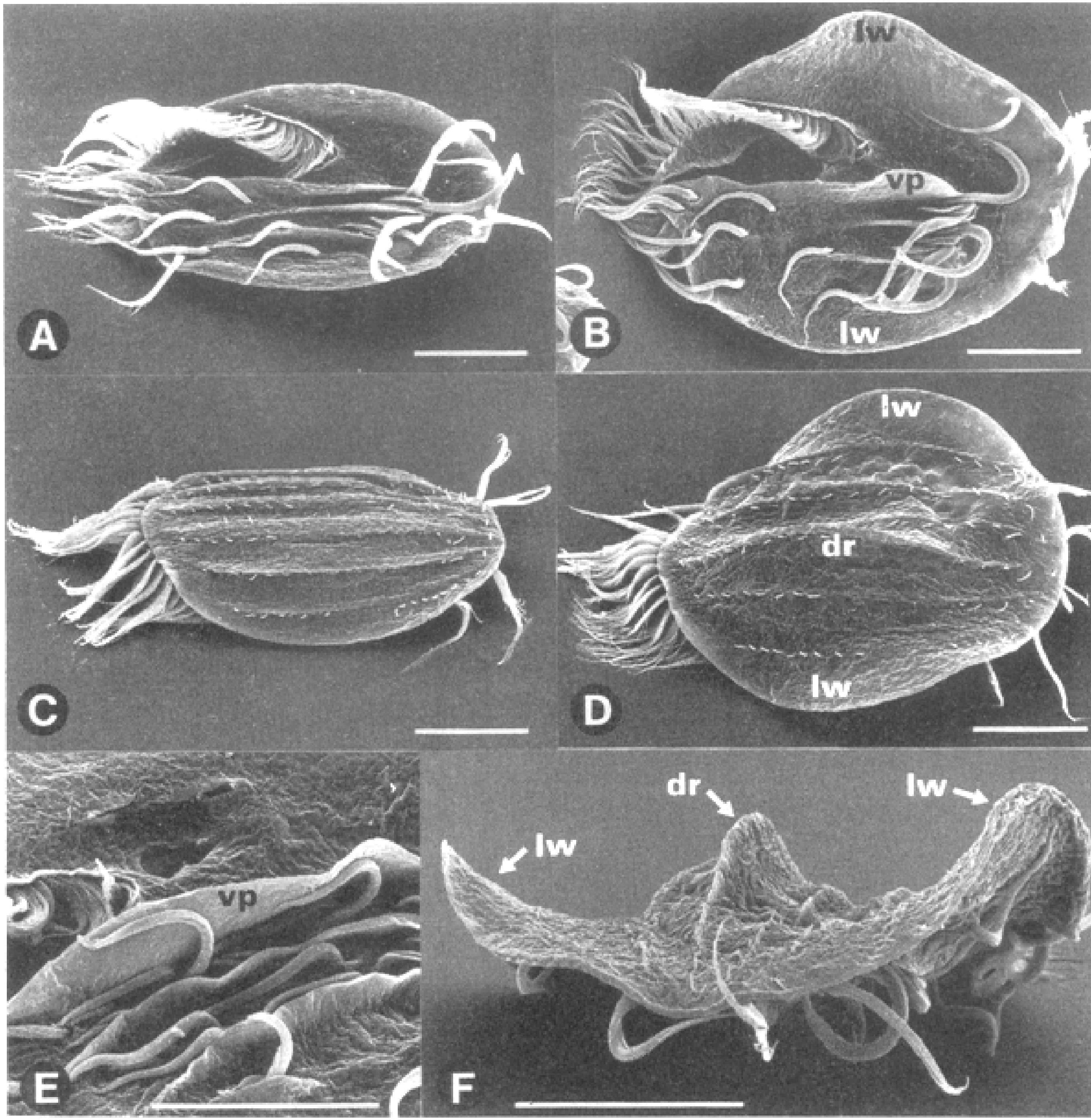


Possibly cued by urea (Wiltshire and Lampert 1999)

FRESHWATER *EUPLOTES* SYSTEM

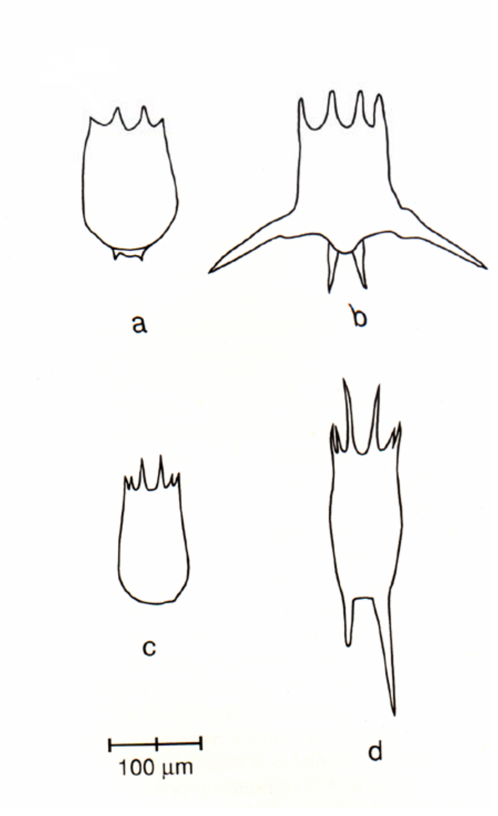


Kuhlman et al. 1999

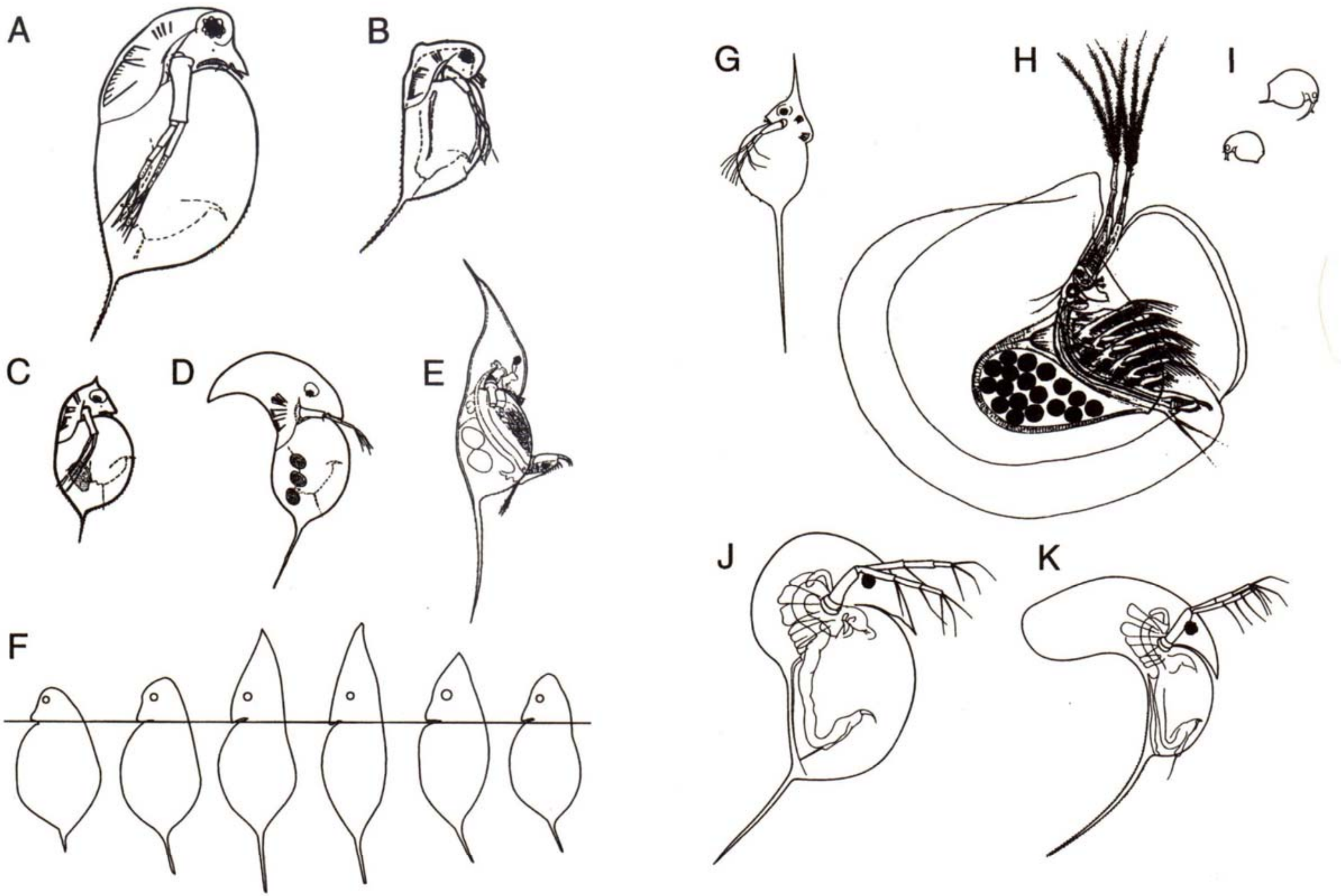

lw
vp
lw
lw
dr
lw
vp
dr
lw
lw
A
B
C
D
E
F

Rotifers

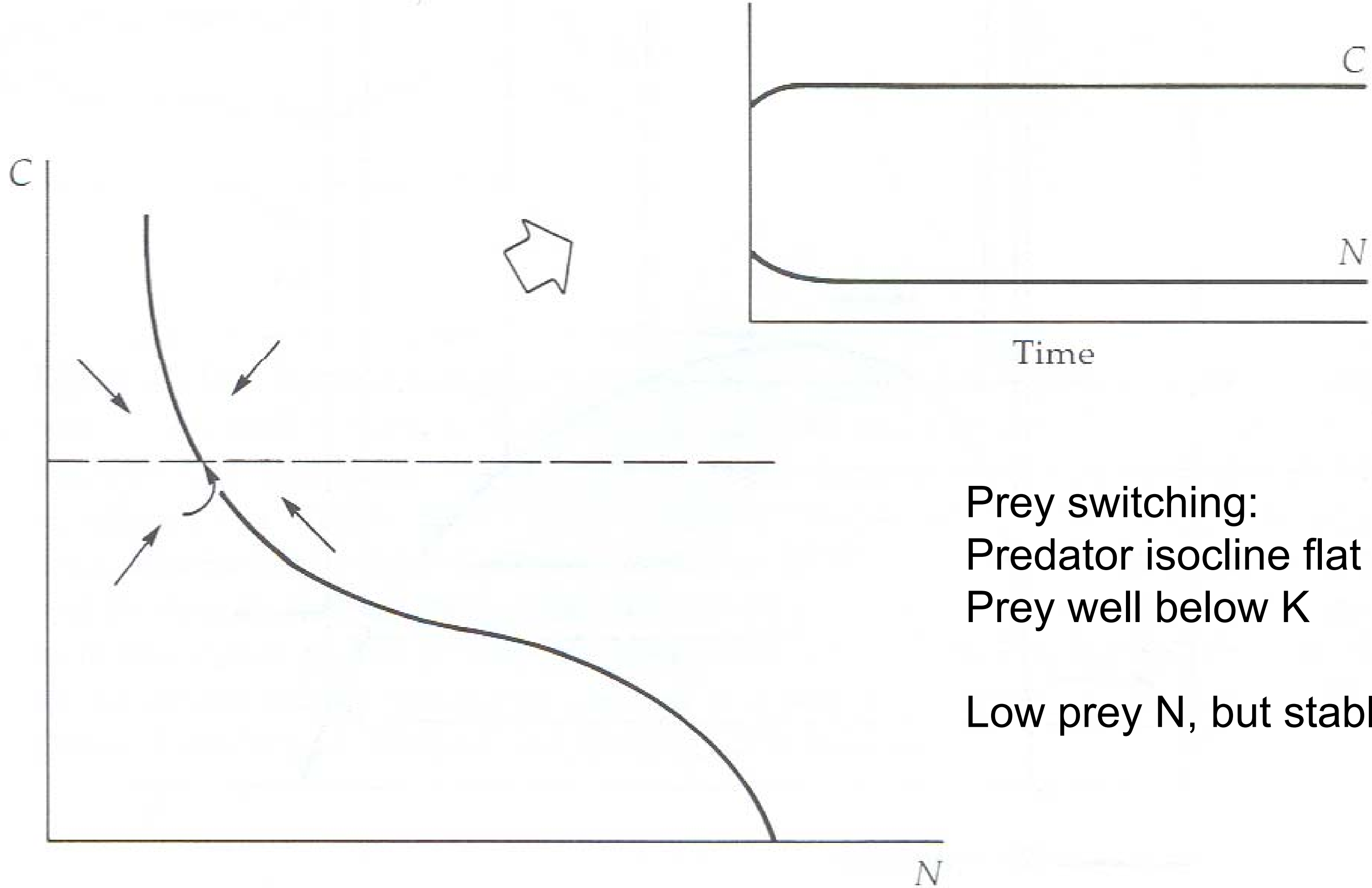
Brachionus spines induced by *Asplanchna* Kairomone(s):



Defenses induced in *Daphnia* by predator kairomones:



Deep-bodied carp induced by pike predator kairomones



Prey switching:
Predator isocline flat
Prey well below K

Low prey N, but stable

Figure 12.8 *The prey zero isocline when a predator exhibits switching behavior, that is, switches from one prey to another.* The predator's abundance may be independent of the density of any particular prey type, and the predator zero isocline may, therefore, be horizontal, that is, unchanging with prey density. This situation can lead to a stable pattern of abundance (inset) with prey density well below the carrying capacity.

Combine a prey refuge with prey Allee effect

-multiple equilibria
-Outbreaks

-Difficult to distinguish predator-prey interactions from other factors such as weather, etc.

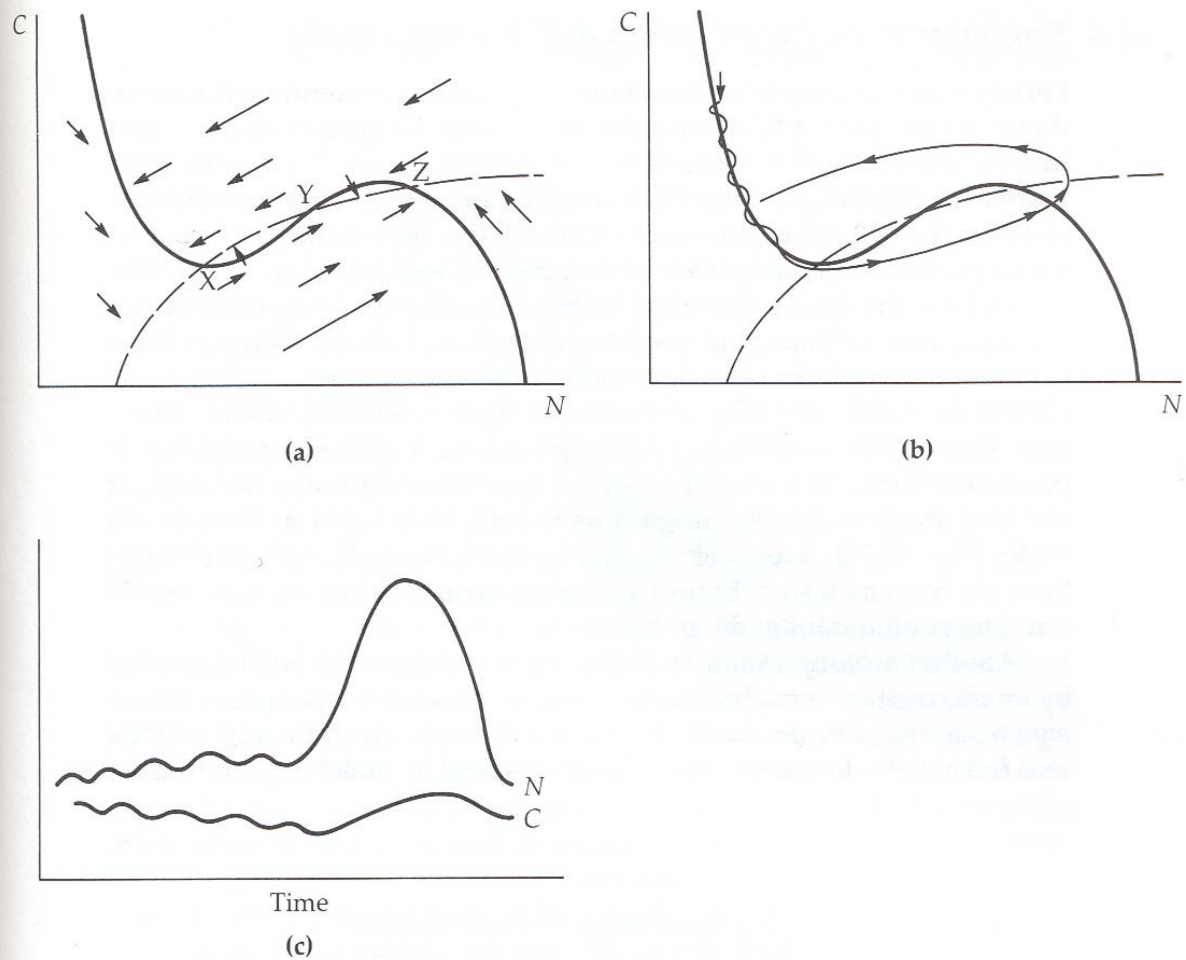


***Figure 12.10** A predator-prey zero isocline model with multiple equilibria.* **(a)** *The prey zero isocline with a vertical section at low densities and a hump.* The predator zero isocline can, therefore, cross it three times. Intersections X and Z are stable equilibria, but intersection Y is an unstable "breakpoint" from which the joint abundances move toward either intersection X or intersection Z. **(b)** *A feasible path that the joint abundances might take when subject to the forces shown in* (a). **(c)** *The same joint abundances plotted as numbers against time, showing that an intersection with the characteristics that do not change can lead to apparent "outbreak" in abundance.* (Redrawn from Begon, Harper, and Townsend 1986.)

Models

- Predator-prey models predict a wide variety of population dynamics using relatively simple and reasonable biological assumptions
- What do we observe in natural systems?

Population cycles as predicted by Lotka Volterra
Synchronous over large areas of boreal forest

Are cycles driven by predation?

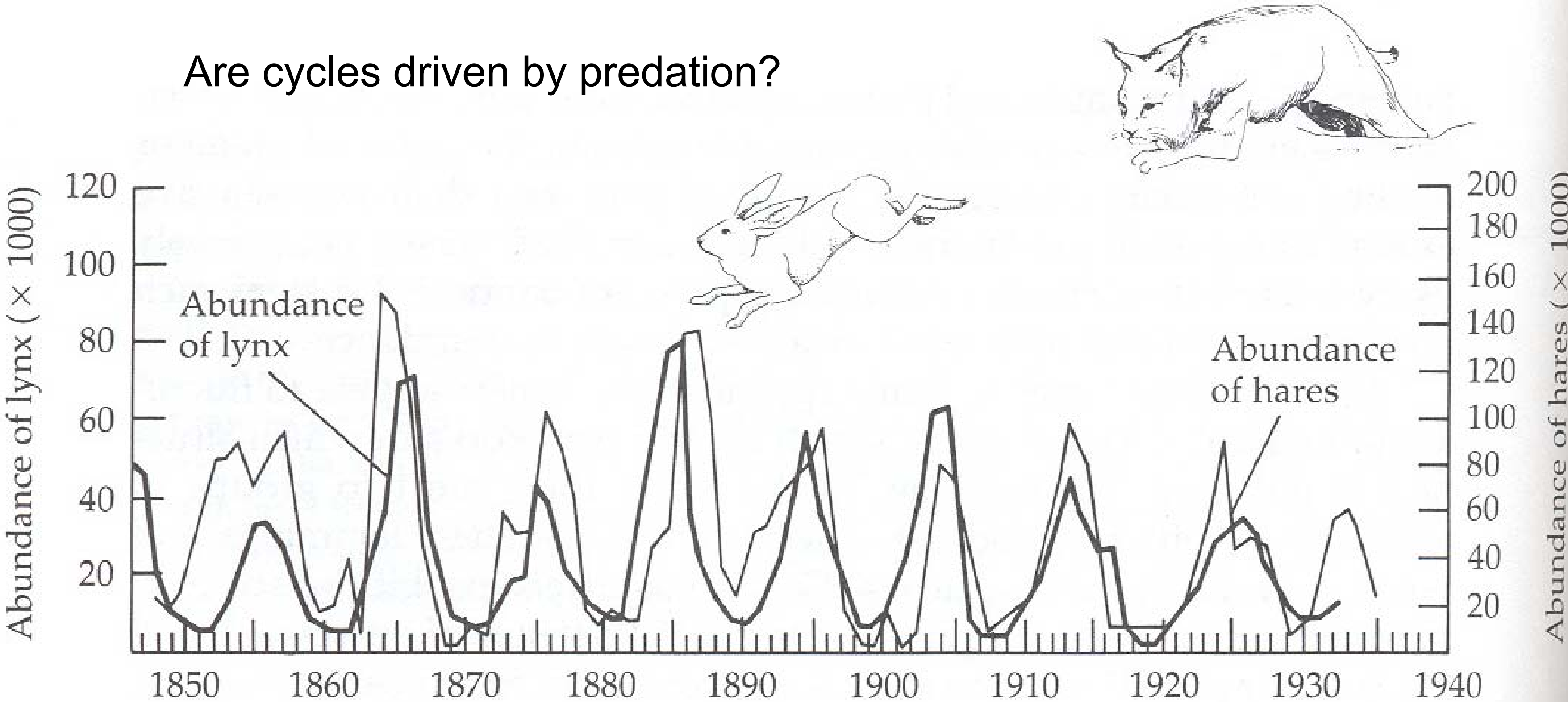


Figure 12.5 *The apparently coupled oscillations in abundance of the snowshoe hare* (Lepus americanus) *and Canada lynx* (Lynx canadensis) *as determined from numbers of pelts lodged with the Hudson's Bay Company.*

Snowshoe hares and food

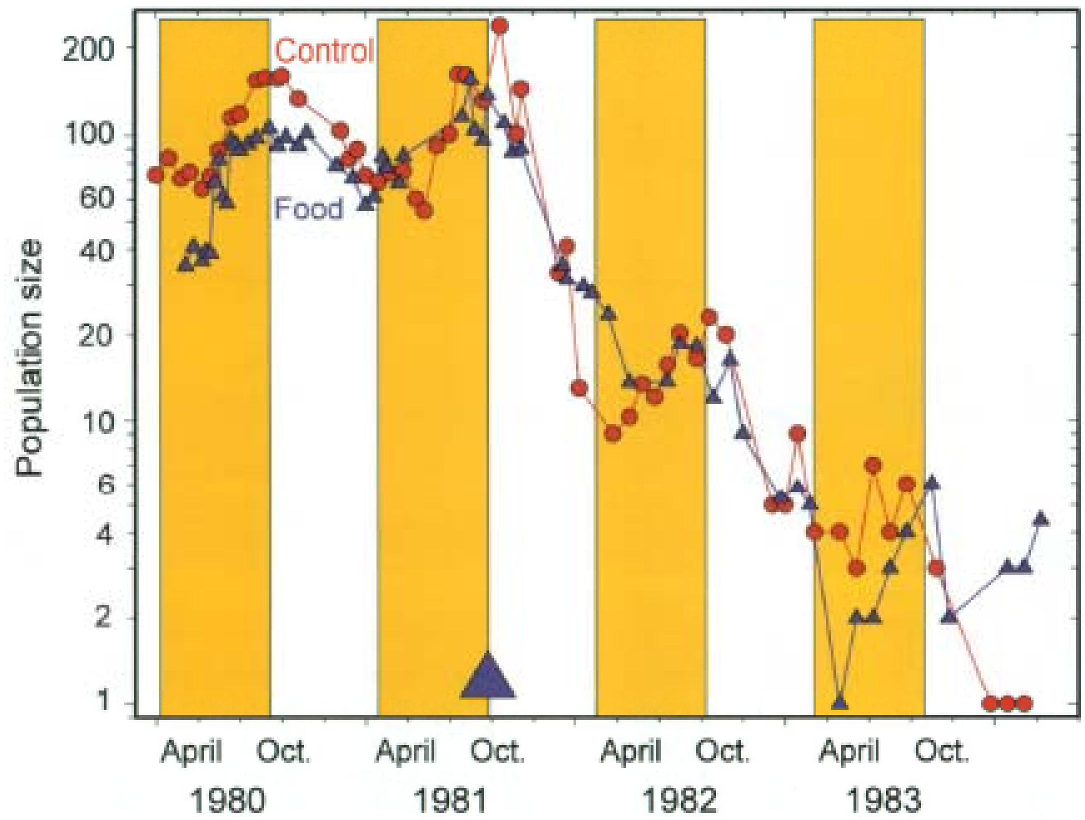


Figure 5. Changes in snowshoe hare numbers on control (1050, red) and food-supplemented (blue) areas during the population decline of 1981–1983 at Kluane, Yukon. The natural feeding experiment was begun in October 1981 (blue triangle). Summer months are shaded yellow. Data are from Krebs et al. (1985).

Krebs et al. (1995)

- Experimental test on 1 km^2 blocks, 2-3 replicates of:
  - control
  - food addition plots
  - predator exclusion plots, permeable to hares
  - fertilized plots
  - predator exclusion + food addition
- estimated density using mark-recapture and robust design
- estimated mortality using radio telemetry

Krebs et al. 1995

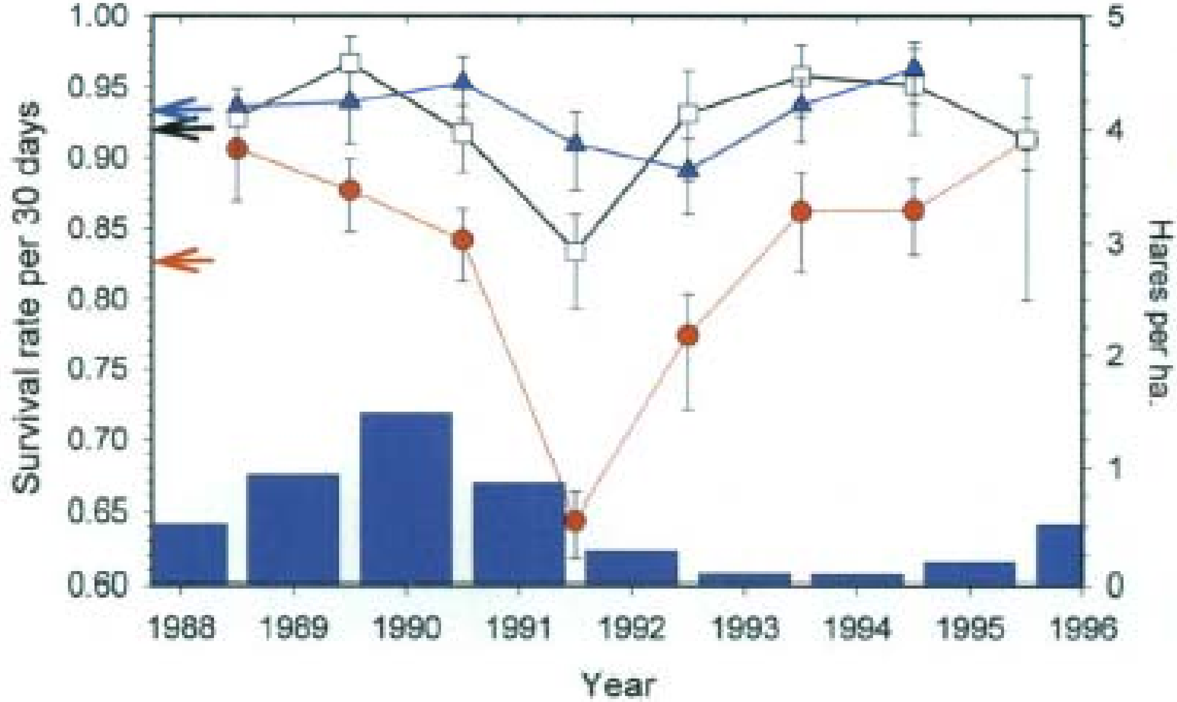


Figure 6. Changes in survival rates of snowshoe hare numbers on control and fenced areas during the population cycle of 1988–1996 at Kluane, Yukon. The arrows show the mean survival rate for each treatment. The survival rate per 30 days is averaged over each year, with 90% confidence limits, for radio-collared hares.

Decline resulted from decreased survival during peak and decline phases, most deaths caused by predation

Decline in survival mostly attributed to predator effect, small effect of food

Krebs et al. 1995

- Predator exclosure doubled density
- Food addition tripled density
- Combination increased density 11-fold
- Nutrients increased plant growth but not hare densities
- Non-additive effect suggested a three trophic level interaction generates the cycle

Spatial synchrony

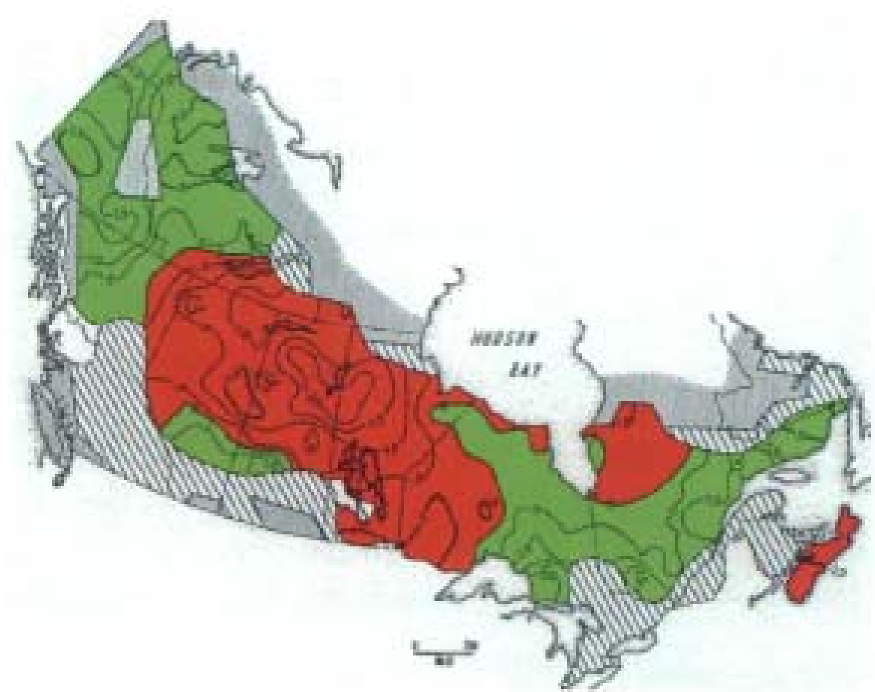


Figure 10. Synchrony in snowshoe hare cycles across Canada, 1931–1948, as measured by questionnaires (Chitty 1948, 1950). The average peak phase across Canada was scaled as 0.0, and the contour lines indicate peaks occurring earlier than average (red, negative contours) or later than average (green, positive contours). During this period, hare peaks were reached earliest in the central boreal region of northern Saskatchewan and Manitoba (Smith 1983).

Traveling 2 year wave

<u>Hypotheses:</u>

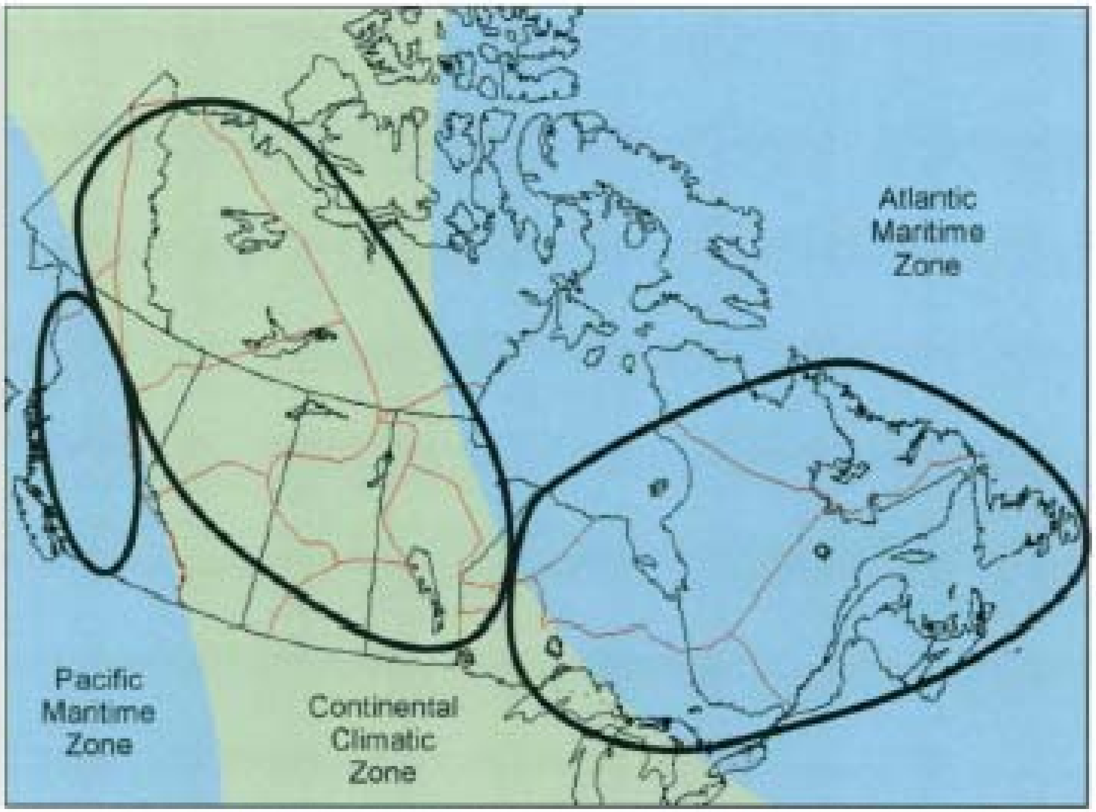
Weather**:** Sunspots, NAO

Dispersal: prey, predators

Sunspots don't explain regional differences in timing of peak

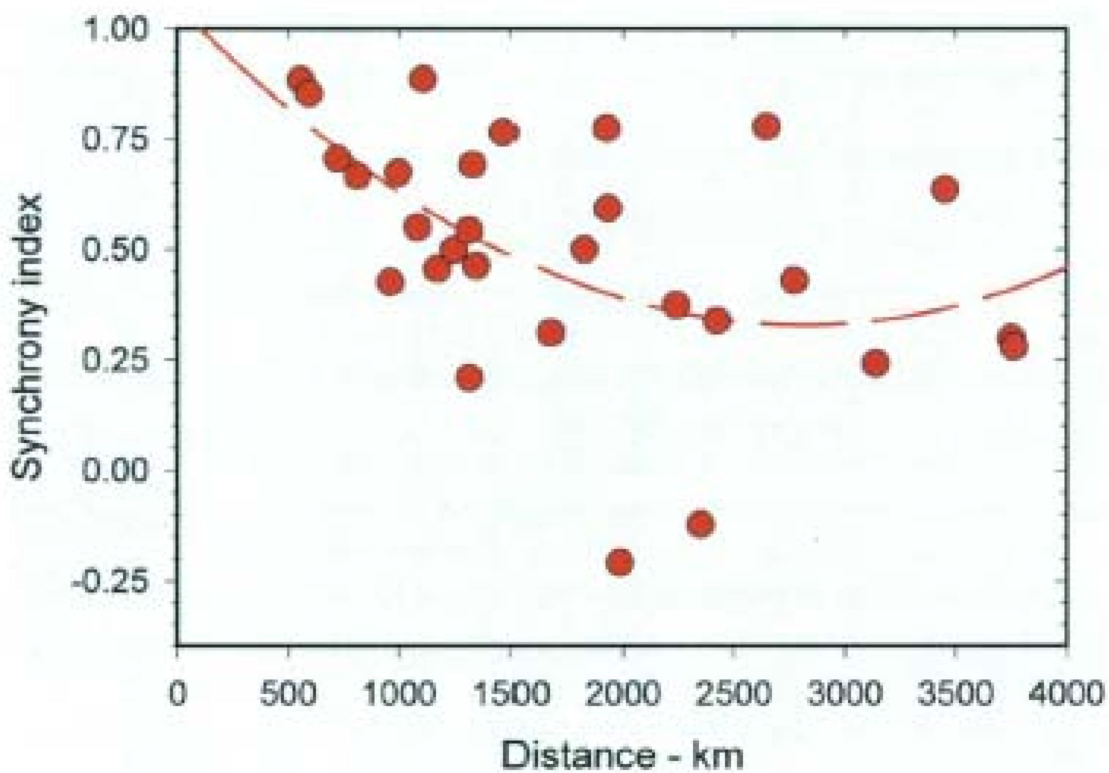
NAO defined climate regions do overlap with areas of hare, but mechanism(s) unclear

Figure 12. Climatic regions (shaded) within Canada, defined by the North Atlantic Oscillation. The large circles define the three regions within which the lynx cycle is most structurally similar. These regions fit within the independently defined climatic zones. Stenseth et al. (1999).



Dispersal

- Hare movements limited, kilometers
- Lynx movements up to 1,100 km
- Great Horned Owls 265-1415 km



Hare and Lynx cycles

- Oscillations are related to predator-prey interaction, but important interactions across three trophic levels
- Predator movements probably important in maintaining synchrony over large areas

Definitions

- Direct effects

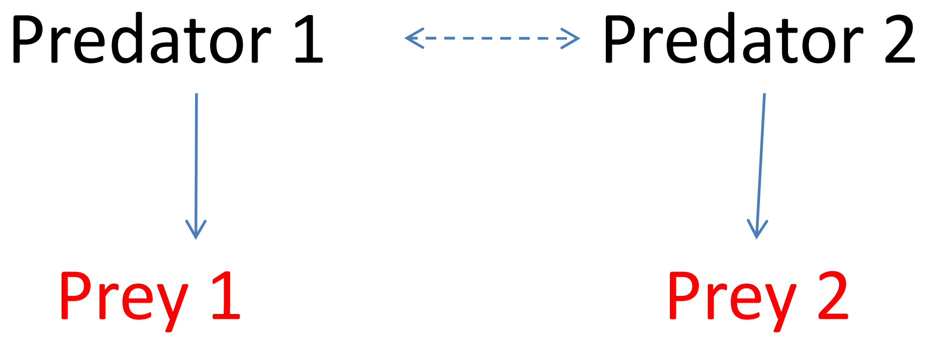
Predator

↓

Prey

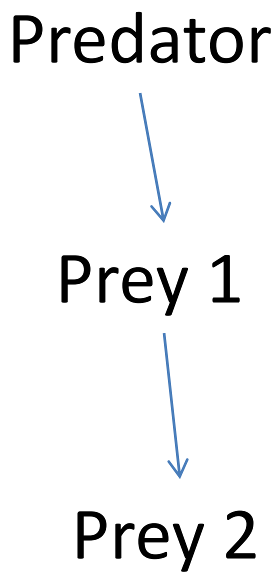
Definitions

- Indirect effects (e.g., competition among predators)



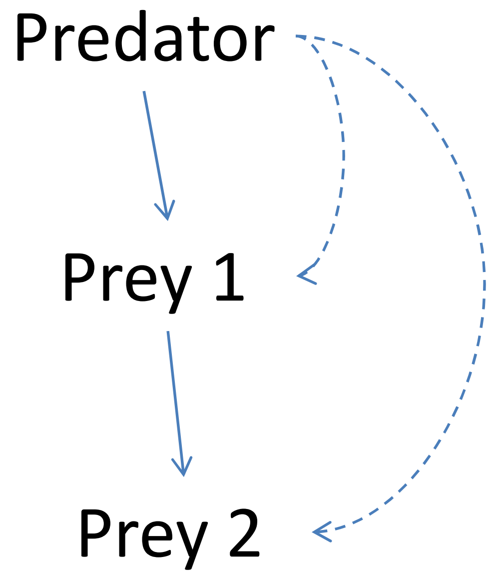
Definitions

- Trophic cascades
  - Direct effects



Definitions

- Trophic cascades
  - Direct effects
  - Indirect effects: altered mesopredator density, prey behavior—can lead to behavioral trophic cascades



Interspecific Killing in Predator Mammals:

Intraguild Predation

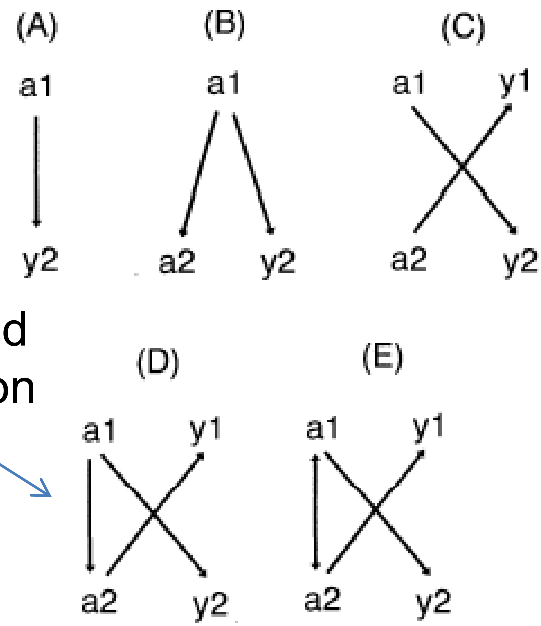


Figure 1: Patterns of interspecific killing in mammalian carnivores. Numbers denote species; letters denote adult (*a*) and nonadult (*y*) individuals. Arrows indicate direction from killer to victim. Asymmetrical age-structured patterns arise when species 1 may kill either (*A*) only nonadults or (*B*) both age classes of species 2. Mutual killings exist in which (*C*) both species kill nonadults of the other, (*D*) species 1 kills both adults and nonadults of species 2, but species 2 can only kill nonadults of species 1, and (*E*) both species kill each age class of the other.

- Direct effects: Mortality rates can be high: 43-68%
- May limit population size of one predator or cause local extinction
- Indirect effects
  - Space use (Cheetahs)
  - Temporal segregation
  - Prey (victim) populations

Palomares and Caro 1999

Soule et al 1988; Crooks and Soule 1999

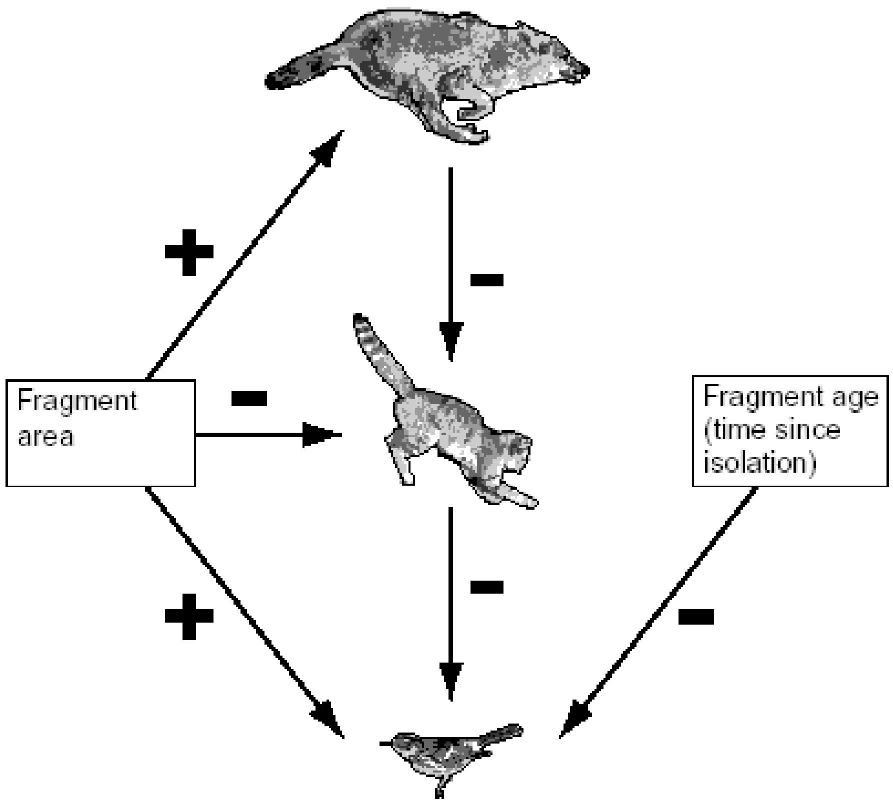


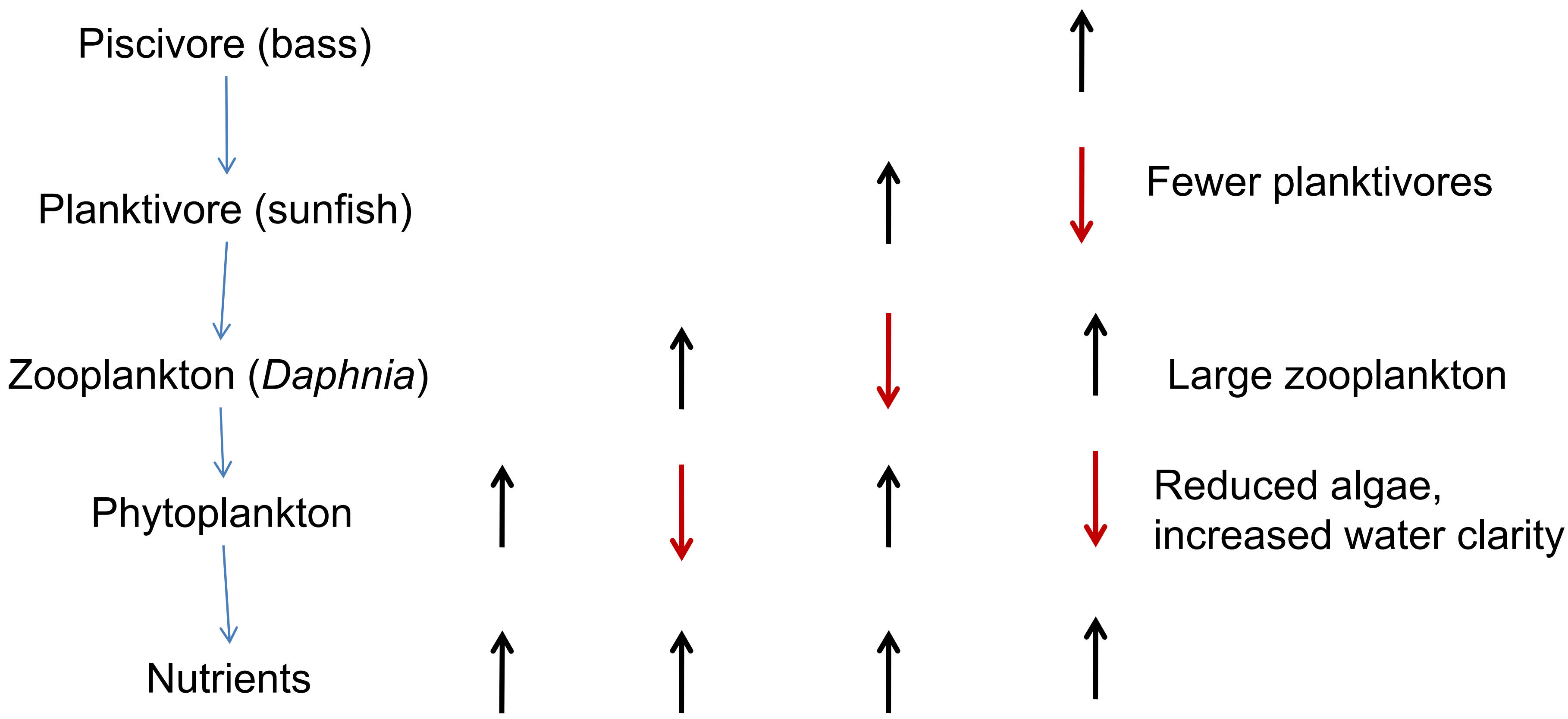
Figure 1 Model of the combined effects of trophic cascades and island biogeographical processes on top predators (for example, coyote), mesopredators (domestic cat) and prey (scrub-breeding birds) in a fragmented system. Direction of the interaction is indicated with a plus or minus.

- Southern California canyons
- Trophic cascade:
- Mesopredator release
  - Fragmentation extirpates coyotes
  - Cats increase
  - Birds decline
- Island Biogeograpy
  - Fragment size
  - Age since isolation

Soule et al 1988; Crooks and Soule 1999

- Direct effects of coyotes: 21% scats with cats; 25% R.T. cats killed by coyotes
- Indirect effects mediated by humans:
  - 46% of owners restricted cat behavior when owners believed coyotes were in area
  - Cat densities well above K, subsidized by cat food: 20 Ha fragment w/ ~100 residences harbors ~ 35 outdoor cats vs. 2-4 native predators
  - Estimated annual cat predation per 20 ha fragment:
    - 840 rodents (67% native)
    - 525 birds (95% native)
    - 595 lizards (100% native)
  - High local extinction rates, "Ecological Traps"

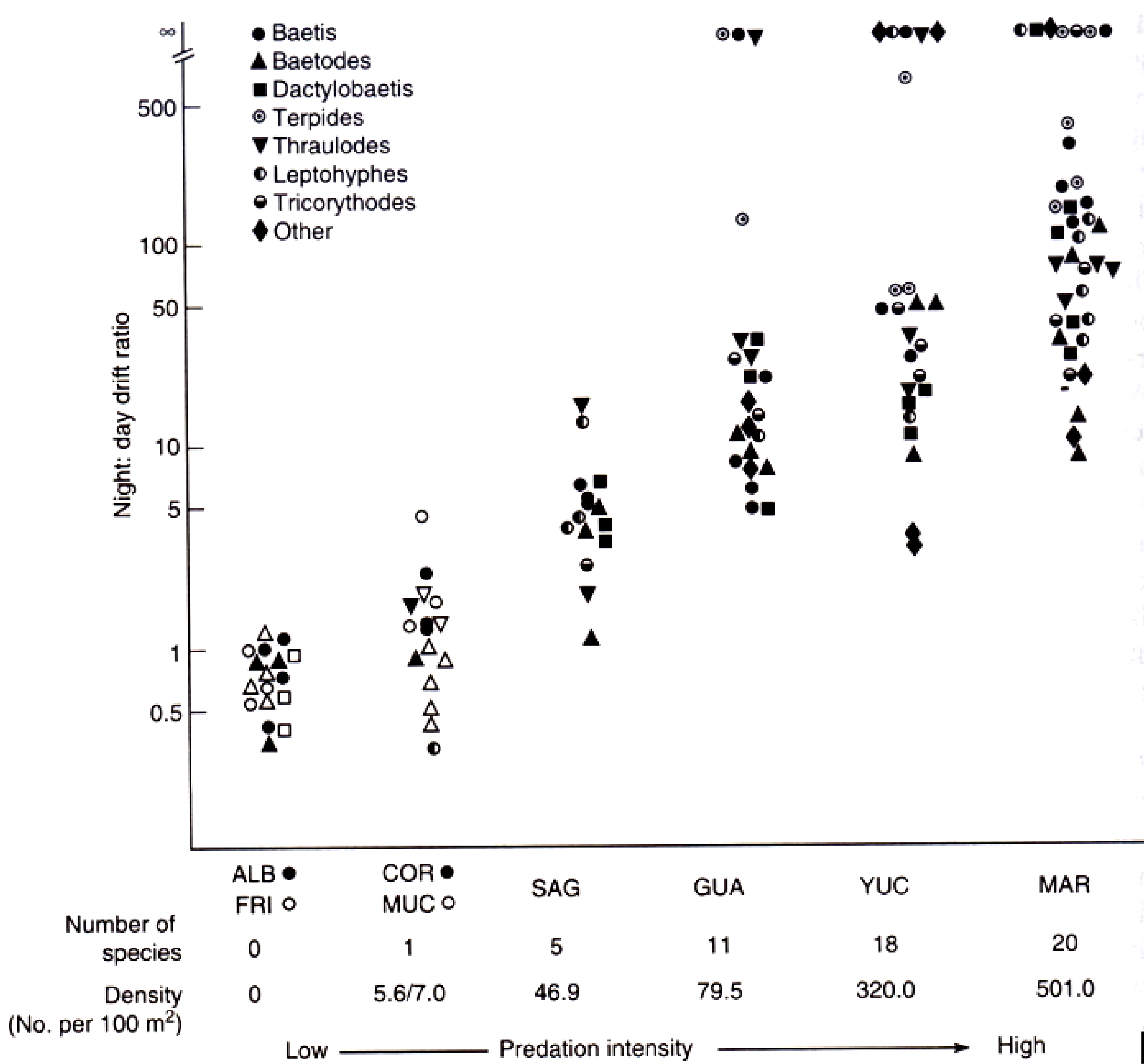
Trophic cascades in mesotrophic-moderately eutrophic temperate lakes



Biomanipulation

- Difficult to remove nutrients
- Relatively easy to manipulate top predators
- Lake Mendota, Wisconsin
  - Biomanipulation increased biomass of top predator (walleye) at same time as die-off of planktivore (cisco)
  - Correlated reduction in algae
  - However, high top predator biomass could not be maintained b/c unanticipated angling pressure

Drift of stream invertebrates and predators:



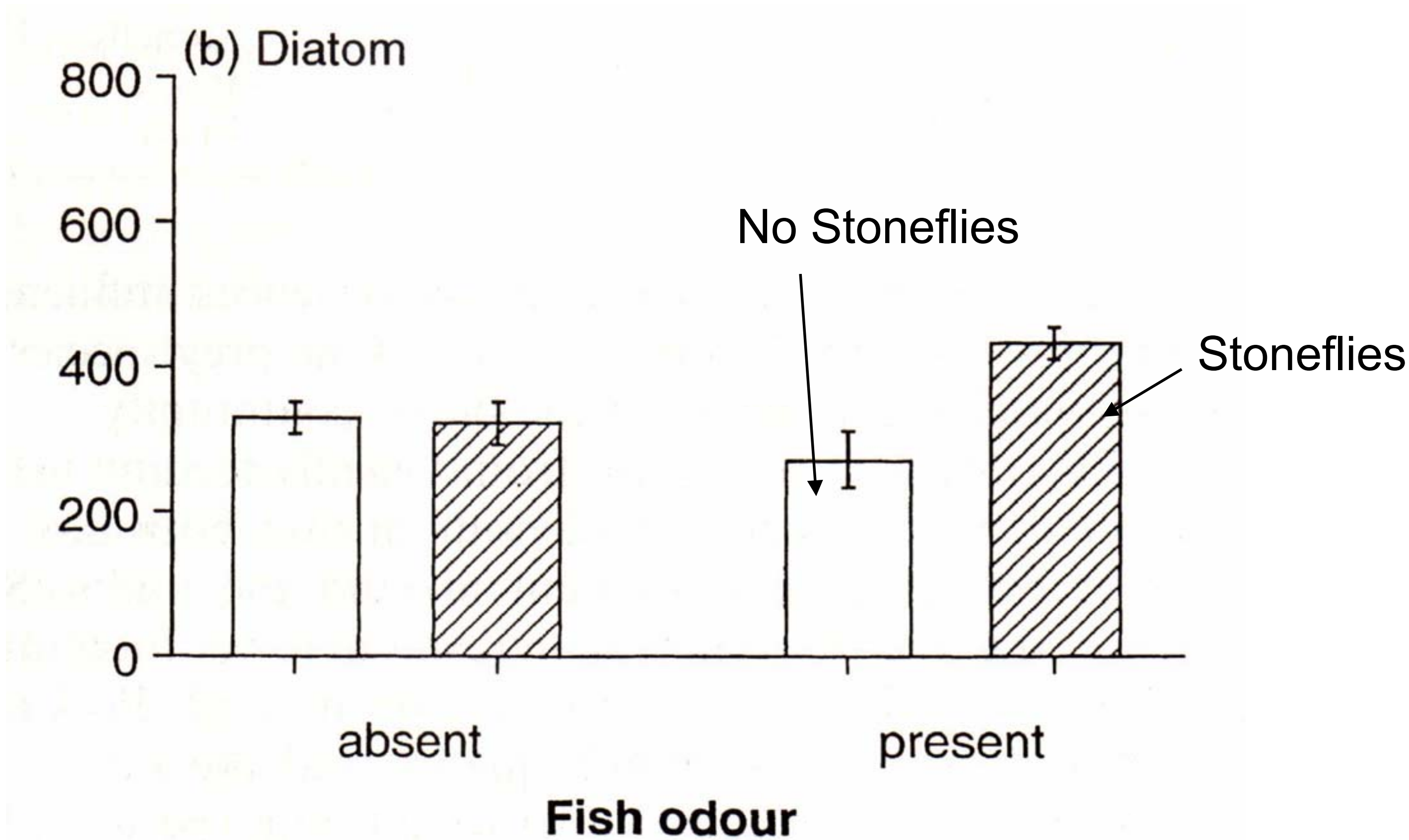
Flecker 1992

Baetis response to trout cues

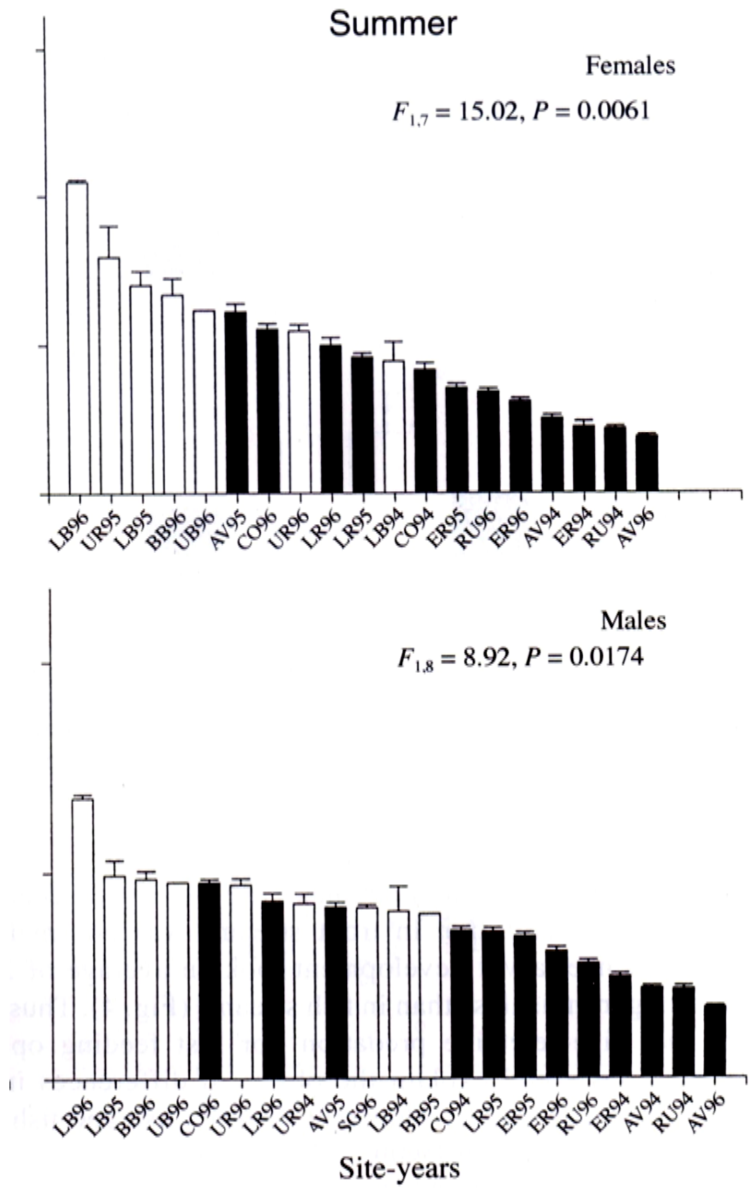
- Trout cues reduce daytime feeding
- Trout cues increase drift at night
- Increased trout cue concentration increases drift in small larvae and inhibits drift in large larvae

Cowan and Peckarsky 1994
McIntosh et al 1999

Stoneflies & trout cues decrease grazing on algae (behavioral trophic cascade):



Baetis size at emergence in natural populations:



Peckarsky et al. 2001

Whole-stream manipulation

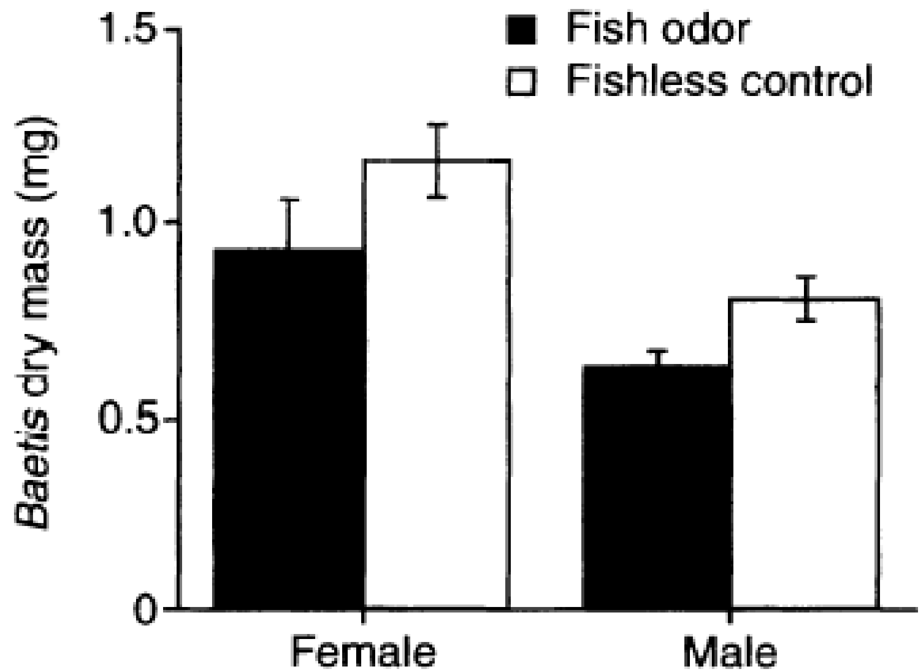


FIG. 1. Dry mass (mean ± 1 SE) of mature (black wing-pad) female and male *Baetis* larvae was lower in streams with brook trout chemicals added (solid bars) compared to control streams with only fishless water added (open bars). Data are for the summer generation, 1999.

Indirect effects of trout cues > direct effects of trout mortality on fitness:

- A demographic model suggest that removing trout mortality would increase fitness by 38.8% (λ natural population: 1.993 vs. 2.765), while removing the indirect negative effects of trout on growth would increase fitness by 114.0% (λ=4.264)

McPeek and Peckarsky 1998