

Lecture 23: Populus Simulations of Predator-Prey Population Dynamics.

Lotka-Volterra Model

L-V model with density-dependent prey population growth

Theta-Logistic Model

Effects on dynamics of different functional response curves

This lab uses two models to simulate predator-prey population dynamics. One is the Lotka-Volterra model, which should be painfully familiar from Lecture 21. You will compare results from the Lotka -Volterra model (with density dependent and density-independent prey population growth) to results from the Theta-Logistic model. You should understand:

1. The changes in predator-prey dynamics that are seen as parameter values are changed within a single model.
2. The different assumptions involved in each model — the Theta-Logistic model is slightly more complex than the Lotka-Volterra model. It makes fewer simplifying assumptions, and therefore is more realistic.
3. Changes in predator-prey dynamics as different assumptions are incorporated.

For all the simulations:

1. The 'Populus' folder is in the 'Biology 405' folder on [\\hoppernew\labshare](http://hoppernew/labshare), as before.
2. There is a key at the back of the handout. Do each numbered exercise, answer the question(s) it asks, then compare your answers to the key before moving on to the next number.

A. *Lotka-Volterra Predation.*

The assumptions of the Lotka-Volterra predation model:

1. Prey grow exponentially in absence of predation.
2. No effect of intraspecific competition on predator.
3. No predator satiation.
4. Uniform habitat with no prey refuges.
5. Single-predator/single prey system.

Simulations:

1. Start Populus.
2. At the main menu, select 'Multi-Species Dynamics'
3. At the submenu, select 'Continuous Predator-Prey Models'.
4. Use the help to read the model notes. The equations are the same as in the lecture, but use different letters:

N = prey population size

r_1 = prey intrinsic rate of increase

C = prey taken per encounter
(C is e in lecture notes)

P = predator population size

d_2 = death rate of predator when prey absent

g = conversion of prey to predator biomass
(g is $f*e$ in lecture notes)

3. At the data entry panel, accept the **default values**:

$N_0 = 20$

$P_0 = 20$

$r_1 = 0.9$

$d_2 = 0.6$

$C_1 = 0.1$

$g = 0.5$

4. Run the simulation and examine the two output plots, which show identical information in two different ways. One shows numbers of predators (red) and prey (blue) vs time. The other shows number of predators (red isocline) vs number of prey (blue isocline) in a 'phase plane' plot. In the phase plane, time is implicitly advancing as you move along the yellow trajectory from the dot (start). An arrowhead shows the direction of the trajectory.

While viewing the predator-prey phase plane, examine the consequence of starting with different numbers of predators and prey. Do different initial numbers of predator and prey affect the dynamics? In what way (what *is* and what *isn't* affected)?

5. Press Esc to return to the data entry panel. Leaving other values constant, experiment by increasing the starvation rate (d_2) that predators suffer in the absence of prey. Recall that this number is subtracted from dP/dt , so a large value means an increasingly negative effect.

What two effects does increasing the predator's vulnerability to starvation have on predator-prey dynamics?

6. Now return to the default values (listed in 3) and experiment with increases in the intrinsic rate of increase for prey (r_1). This is the rate at which prey would increase (exponentially) in the absence of predation.

What effect does increasing the prey's intrinsic rate of increase have on predator-prey dynamics?

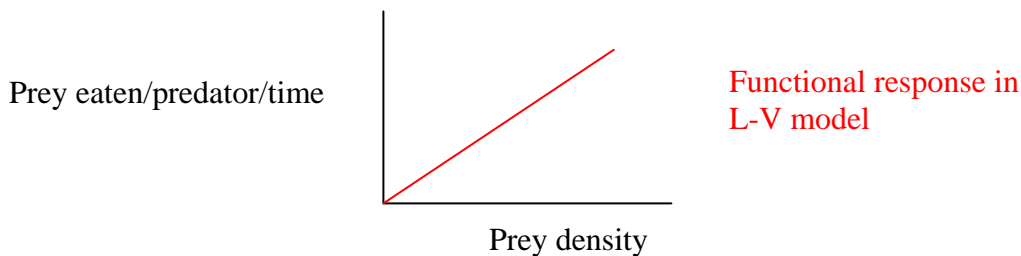
7. Now return to the default values and make prey growth density dependent, using the check box. What does this do to the isoclines? Why? What effect does increasing or

decreasing K have? How does the changed isocline due to density-dependence alter the dynamics?

B. Theta-Logistic Predation

The Lotka-Volterra model captures many features of predator-prey interactions, but it has some assumptions that are unrealistic. Two of these assumptions are dropped in the theta-logistic model:

- (1) Prey population growth is exponential in the absence of predation in the L-V model, but is logistic in this model.
- (2) The predator's functional response is linear, with no satiation point in the L-V model:



As will be discussed in lecture 22, an asymptotic (Types 1 & 2) or sigmoid (Type 3) functional response is more typical.

The Theta-Logistic predator prey model allows one to incorporate a functional response of type 1,2 or 3.

Growth rate of prey:

$$dN/dt = rN (1 - (N/K)^\theta) - fP$$

N = number of prey

r = intrinsic rate of increase for prey population

K = carrying capacity of prey population

Θ = (theta) exponent determining the force of density dependence

f = functional response of predator (described below in detail)

P = number of predators.

The term $1 - (N/K)^\theta$ is algebraically equivalent to $(K - N)/K$. So, this equation is the same as the (Verhulst-Pearl) logistic growth equation, with two modifications:

1. The density dependent term has an exponent θ (theta) added. When θ is large, it has the effect of making density dependence strong only when N is near K. When θ is

small, it has the effect of making density dependence strong even at low population density.

2. Predation slows growth as the number of predators (P) increases, with an impact that depends on the shape of the functional response curve (f).

The functions that are used for f are:

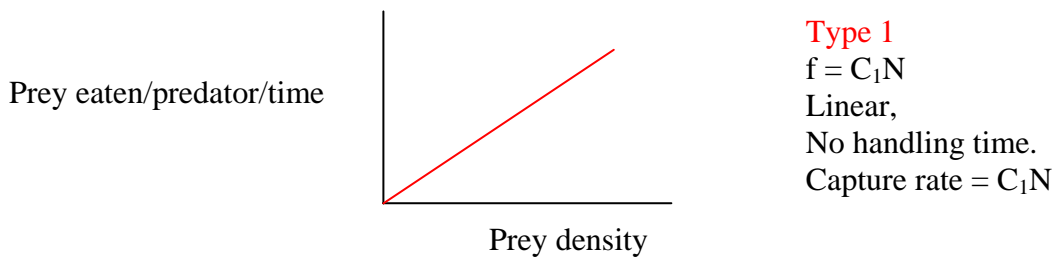
1. Type 1: $f = C_1N$
2. Type 2: $f = C_2N/(1 + h_2C_2N)$
3. Type 3: $f = C_3N^2/(1 + h_3C_3N^2)$

$C_1, C_2, C_3 = \text{constants}$

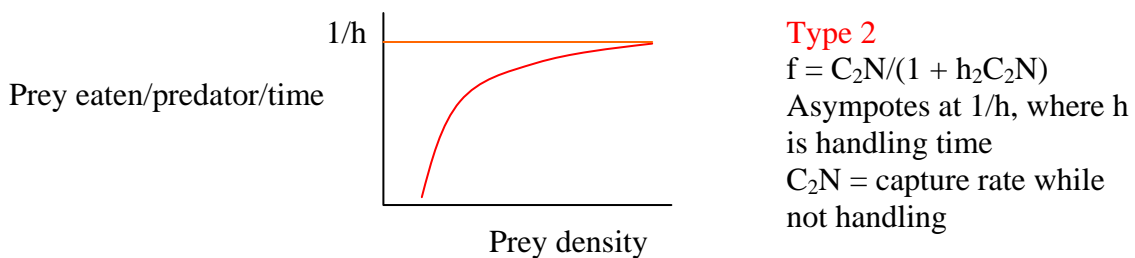
$N = \text{number of prey}$

$h = \text{handling time}$

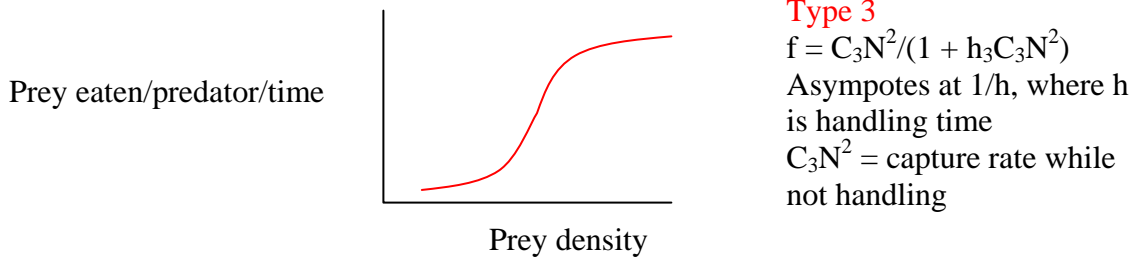
So Type 1 is a linear functional response (as in the L-V model):



Type 2 is asymptotic (accounting for satiation and handling time):



Type 3 is sigmoid, accounting for satiation, handling time, and prey switching:



Growth rate of predator

$$dP/dt = gP(f - D)$$

P = number of predators

f = functional response (described above)

D = number of prey needed for predator to replace itself

g = efficiency of conversion of prey eaten to predator offspring.

This is an exponential growth curve ($sP = a$ constant times population size), damped by the functional response of predators to prey (f), and a threshold amount of food (D) needed before individual predators contribute to population growth.

Simulations:

1. The Type 1 functional response is linear, as in the Lotka-Volterra model. Specify a Type 1 functional response, so that the major difference between this model and the L-V model is that prey growth is density dependent, rather than exponential, in the absence of predation.

Accept the **default values**:

This models a situation very similar to L-V model with default values, except that density dependent regulation of prey is taken into account.

How does the T-L result differ from L-V result? What does this imply about the likelihood of seeing predator-prey cycles in nature?

2. Keeping other variables at the defaults, increase (and decrease) the carrying capacity for the prey population. Use several values that allow K to vary widely. What effect does $\uparrow K$ have on population dynamics? Why?
3. Reset $K = 100$, and keep other variables at the defaults. Increase the intrinsic rate of increase for the prey (r), using several values between $r = 0.1$ and $r = 5$. What effect does $\uparrow r$ for the prey population have on population dynamics of predator and prey? Which assumption drives this result?
4. (a) Now change the functional response curve to Type 2, or open a second copy of the module, so you can directly compare results with Type 1 and Type 2 functional response curves.
 - (c) Reset all the parameters to default values. You can do this by just closing the module and re-opening it
 - (d) Carefully compare the results for Type 1 and Type 2 functional responses.
 - (e) With a Type 2 functional response, run a series of five simulations, increasing the number of prey needed for a predator to replace itself (make sure to move D through a range of values around 0.5-0.7) but leaving everything else constant.

How do the population dynamics change when the predator has a Type 2 functional response rather than Type 1? (*Note that, using default values, $C = 0.05$ and $h = 1$ for now*).

5. (a) Using the Type 2 functional response, set all the parameters back to defaults. Run the simulation (to give a standard to which you can compare the next result.)
 - (b) Now set $h = 2$, run the simulation and compare to the previous result.

What effect does a doubling of the handling time have on the maximum rate of prey intake per predator? The simulation doesn't directly give you the answer to this — you just have to think about it.

With a type 2 functional response, what effect does a doubling of the handling time have on the population dynamics?

6. Return parameters to default values, still using a Type 2 functional response. The default value for the carrying capacity of the prey population is $K = 100$. Run a series of simulations in which K varies while everything else is held constant.
 - (a) Reduce K in a series of runs.
 - (b) Increase K in a series of runs.

What effect does altering the carrying capacity of the prey have on the population dynamics? Decreasing K ? Increasing K ?

7. Experiment on your own to explore the effects of altering the capture rate (C) with a type 2 functional response. Then explore the effects of altering θ . When θ is small, the

prey population is affected by density dependence even when prey numbers are low.
When θ is large, density dependence is weak until prey numbers are high.

Key for predation simulations.

A.4. Altering initial numbers of predators and prey changes the amplitude of the cycles, but does not shift the basic pattern of cycles in which predator density lags prey density by $1/4$ cycle.

A.5. Increasing predator's vulnerability to starvation (r_2) has two effects:

- Increases the predator's isocline (the density of prey below which the density of predators decreases). That is, the predator isocline shifts to right.
- Increases the amplitude of cycles.

A.6 Increasing the prey's intrinsic rate of increase has two effects:

- Increases the prey's isocline (the density of predators below which the density of prey decreases). That is, the prey isocline shifts upward.
- Increases the amplitude of cycles.

A.7. By adding intraspecific competition to the prey dynamics, you cause the predator isocline to have a negative slope. That is, fewer predators are needed to limit the prey as the prey reaches high density, because the prey are limiting themselves via intraspecific competition for resources. As 'bottom-up' regulation gets stronger, 'top-down' regulation gets weaker.

B.1. In the Lotka Volterra model, cycles continue indefinitely. In the Theta-Logistic model, similar conditions produced 'damped' cycles, in which the amplitude decreases with each successive cycle, so the trajectory spirals in to a stable equilibrium.

This implies that, for cases in which prey are affected by predation **and** by intraspecific competition (density-dependence), cycles might be transient. Adding a carrying capacity for prey to the model leads to stable equilibrium where the L-V model predicted endless cycles.

This is one possible explanation for the general rarity of long-term predator-prey cycles in nature.

B.2. Increasing the carrying capacity of the prey population allows cycles to persist for a longer period. As $K \uparrow$, the prey's population growth remains almost exponential (density-independent) for a longer period, so the model behaves more like the L-V model than when K is small.

In the extreme, with K set at, say, 1000 or 2000, then the prey isocline is virtually horizontal, as in the L-V model, and cycles will persist for a loooooong time. You can prove this to yourself by setting the simulation to 'run to stable state' with $K = 1000$.

Low carrying capacity for prey = strong density dependent limitation of prey = steep negative slope of the prey isocline = quick elimination of cycles.

High carrying capacity for prey = weak density dependent limitation of prey = small negative slope of the prey isocline = long persistence of cycles.

B.3. Increasing the intrinsic rate of increase for the prey (while leaving the carrying capacity constant) has no effect on the equilibrium density of prey, which you might have expected. Instead, predator numbers increase to harvest the increased food, and the only effect is an increase in the equilibrium density of predators.

This result is driven by the assumption that predators are never satiated, and require no handling time to catch/kill/eat prey. With a Type I functional response (and constant S), predators just keep benefiting from the increase in prey availability, and never reach an upper limit.

B.4. Switching the functional response to Type 2 changes the shape of the prey isocline. With Type 1, the prey isocline is linear, with a negative slope. With Type 2, the prey isocline is no longer linear — it is a decelerating function, but the new prey isocline is above the old isocline (or equal to it) for all prey densities. In other words, *it requires a larger number of predators to reduce the prey population's growth rate (dP/dt) to 0*, when the predator has a type 2 functional response.

What is the effect of this new prey isocline on the population dynamics? That depends on where the predator isocline crosses the prey isocline. This is illustrated by the series of runs with increasing D , which shifts the predator isocline to the right.

- At low prey density, the isoclines cross where the prey isocline is flat, relative to the type 1 prey isocline. Consequently, cycles persist longer with type 2 functional response than with type 1.
- At high prey density, the isoclines cross where the prey isocline is steep, relative to the type 1 prey isocline. Consequently, cycles persist a shorter time with type 2 functional response than with type 1.

B.5. Setting $h = 2$ represents a doubling of the handling time, which reduces the maximum rate at which prey can be captured ($1/h$) from 1.0 to 0.5 prey captured per predator per time period.

The population dynamics previously showed damped cycles that reached a stable equilibrium fairly quickly. Doubling the handling time shifted the dynamics back to stable cycles!

Important point: Predator - prey cycles are predicted by the L-V equations as a consequence of fairly unrealistic assumptions (exponential prey growth, no satiation of predator). However, predator - prey cycles are also predicted for some sets of parameter values in more realistic models. The model you just ran incorporates logistic growth in the prey population (realistic) and a type 2 functional response (also realistic), and it exhibits stable predator-prey cycles.

B.6. Reducing the carrying capacity of the prey gives the prey isocline a steeper negative slope (which makes sense). A steep prey isocline at the point of intersection means that the trajectory cycles for a short time before reaching stable equilibrium.

If the carrying capacity of the prey drops below D (the number of prey needed for a predator to replace itself), then the last predator dies and the prey population stabilizes at K .

Increasing K has the opposite effect. The prey isocline becomes flatter as $K \uparrow$, so the populations cycle for longer periods. As $K \uparrow$, eventually a threshold is crossed, and the predator-prey trajectory shows stable cycles (at $K = 1.835$). With $K > \text{threshold}$, the trajectory spirals outward rather than inward. (The threshold is at $K = 1.835$, for this set of parameter values, but there is nothing magical about this number — the threshold would fall at a different value of K for another set of parameter values).

Important point: With realistic assumptions, predator-prey cycles can be fragile. A given set of parameter values will lead to cycles. However, a small shift in the carrying capacity (e.g. a slightly better than average year, in terms of food available to prey) can nudge the dynamics into an outward spiral. Outward spirals are inherently unstable; either the predator or the prey population will eventually strike zero. If the prey strike zero first, then the predator will follow (unless it can switch to other prey). If the predator strikes zero first, then the prey will go to K .

A common argument for the rarity of cycles in nature is that the conditions that cause cycles will also lead to extinctions, with only a small shift in ecological conditions. In other words, the conditions that favor cycles are similar to conditions that predict local extinction of predator, or local extinction of prey followed by local extinction of predator.

B.7.

(A) Increasing θ allows the prey population to grow almost exponentially for a longer time before density dependent effects slow dN/dt . Therefore, the prey isocline moves up as θ increases. However (if the carrying capacity is not changed) then the prey isocline still falls to zero at the same point on the X-axis ($dN/dt = 0$ at $N=K$, regardless of the shape of the isocline). These two effects combine to make the prey isocline increasingly 'humped' as θ increases. Because this increases the slope of the prey isocline at its point of intersection with the predator isocline, increasing θ causes cycles to persist. (And increases the amplitude of the cycles). This **reiterates a point seen earlier**: increasing density dependence in prey population growth tends to eliminate cycles.

(B) Increasing the capture rate:

- Shifts the predator isocline to the left. In other words, fewer prey are needed for the predator population to maintain a positive growth rate ($dP/dt > 0$).
- Prolongs cycling. Eventually, the system settles into a repeating series of cycles if C is large enough. This is a way of modeling stronger predation pressure without initially increasing the number of predators (though P may later increase as a result).

That is, you are modeling a situation in which each predator has a bigger impact on prey (per capita).