An Introduction to Mathematical Ecology

3MC School on Quantitative Biology

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1 Modelling and Forecasting Ecological Dynamics

1.1 Objectives

The latest draft of this slide deck is posted on my website

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```
library(ggplot2)
```

1.0.1 Ecological Dynamics

- Competition and cooperation within species
  - logistic growth
  - Allee effects
- Competition and predation between species
  - predator-prey dynamics
  - intra-guild predation
- Host-Pathogen dynamics
- Biological Invasions
  - forest insect pests
  - agricultural pests and pathogens
  - animal diseases
  - biological control

1.0.2 Heterogeneity

- variation between individuals
  - age
  - size
  - behaviour
- variation in landscape
- variation over time

1.1 Model Design

1.1.1 Individual-based and Compartmental-based Models

- Compartmental models
  - The population is subdivided into *compartments* reflecting relevant heterogeneities.
  - The *state-space* of the system is the number of individuals in each compartment.
  - Individuals in the same compartment are interchangeable.
- Individual-based models:
  - The *state-space* of the model includes the state of each individual in the population.
  - Often more *natural* to develop, since the focus in describing the process at the level of the individual
  - Often more *complex* than necessary

1.1.2 Discrete-time vs Continuous-time Models

Many organisms have non-overlapping generations. Take, for example, an annual plant that does not survive through the winter, but produces seeds that germinate and sprout in the spring.

$$N_{t+1} = N_t \times \text{Fecundity} \times \text{Survival}$$

- Fecundity, or recruitment, is a number larger than one, for example, a ratio of offspring to adults, or juveniles to parents.
- Survival is a fraction, or a probability a given juvenile survives to the reproductive stage/age.

1.1.3 Age Structure

Or organisms may have life cycles that are synchronized, so taking a census of populations by age at fixed times makes sense.

$$
\begin{aligned}
N_{0,t+1} &= f_1 N_{1t} + f_2 N_{2t} + f_3 N_{3t} \\
N_{1,t+1} &= s_0 N_{0t} \\
N_{2,t+1} &= s_1 N_{1t} \\
N_{3,t+1} &= s_2 N_{2t}
\end{aligned}
$$

1.1.4 Size Structure

Many organisms are better described by size, not age.

$$
\begin{aligned}
N_{0,t+1} &= f_1 N_{1t} + f_2 N_{2t} + f_3 N_{3t} + (1 - p_0) N_{0t} \\
N_{1,t+1} &= (1 - p_1) N_{1t} + p_0 s_0 N_{0t} \\
N_{2,t+1} &= (1 - p_2) N_{2t} + p_1 s_1 N_{1t} \\
N_{3,t+1} &= (1 - p_3) N_{3t} + p_2 s_2 N_{2t}
\end{aligned}
$$

1.1.5 Stochastic vs Deterministic Modelling

Andersson and Britton (2012) raise several good arguments for using Stochastic models over deterministic models in Biology. Their interest is specific Epidemic Models, but the points pertain to Biology more generally.

1. The stochastic framework is the **most natural way to describe** most biological processes
2. Many phenomena of interest are inherently stochastic.
3. Quantifying our uncertainty in model elements, processes, and outcomes is necessary and inherently probabilistic.
4. Any forecast or assessment of the model is of little value without a corresponding assessment of its uncertainty.

1.1.6 Quantifying Uncertainty

An essential step in designing models is a *quantification of uncertainty.*

To be useful, a prediction must be accompanied by some measure of its accuracy and reliability.

Sources of uncertainty:

- uncertainty in model design, including
  - our understanding of the process being modelled,
  - our choice of model,
    - parameter selection, and
    - inherent variability in the process being modelled;
- uncertainty in observations,
  - measurement error.

1.1.7 Modelling Theory vs Modelling Data

- Modelling Theory
  - model hypothesized processes
  - compare predictions and observations
  - how well does theory explain observation?
  - robust, *extrapolate* to other scales, regions, …
- Modelling Data
  - descriptive
  - interpolation
  - near-term forecasting
  - no inference can be made on underlying processes

1.1.8 Incorporating Uncertainty and Variability

$$
\begin{aligned}
N_{0,t+1} &= f_1 N_{1t} + f_2 N_{2t} + f_3 N_{3t} + (1 - p_0) N_{0t} \\
N_{1,t+1} &= (1 - p_1) N_{1t} + s_0 p_0 N_{0t} \\
N_{2,t+1} &= (1 - p_2) N_{2t} + s_1 p_1 N_{1t} \\
N_{3,t+1} &= (1 - p_3) N_{3t} + s_2 p_2 N_{2t}
\end{aligned}
$$

Process Variability: f_i, p_i, s_i are nonlinear operators returning random variables.

Environmental or Temporal Variability: f_i, p_i, s_i are deterministic, but parameters are random variables.

Observation Uncertainty: not propagated through time

$$Y_{it} = N_{it} + \epsilon_{it}$$

1.2 Models and Forecasts

1.2.1 What is a Forecast?

The process of **predicting** the state of ecosystems, ecosystem services, and natural capital, **with fully specified uncertainties**, and is **contingent on explicit scenarios** for climate, land use, human population, technologies, and economic activity. Forecast state at future time from state at current time given assumptions about parameters. – (Clarke et al, 2001)

1.2.2 Forecasts for Policies, Strategies, and Decisions

- Serviceable Truths
- Working Hypotheses
- Actionable Intel

All models are wrong, but some models suggest *serviceable truths.*

1.2.3 Forecasting is part of the Scientific Method

- Observe
- Formulate Research Question
- Formulate Model and Hypothesis
- Predict/Forecast
- Gather data and test model/hypothesis
- Report and Repeat

2 Growth of a single population

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2.1 The Process-based ODE framework

2.1.1 The general *process-based* framework

$$\frac{dN}{dt} = B(t, N) - D(t, N)$$

- $N(t)$ is the population at time t
- continuous state: $N \in \mathbb{R}$
  - interpret N as a density or expected population
- continuous time: $t \in \mathbb{R}$
- $B(t, N)$ is the birth rate at time t and population N
- $D(t, N)$ is the death rate at time t and population N

Objective: study solutions of the ODE and their dependence on initial conditions and parameters.

2.1.2 Exponential Growth

Suppose the birth and death rates are both independent of time and proportional to the population size.

$$\frac{dN}{dt} = bN - dN$$

with $N(0) = N_0$.

- The constants b and d are referred to as the *per-capita* birth and death rates.
- The solution grows exponentially at rate $b - d$ if $b > d$, or decays if $b < d$.

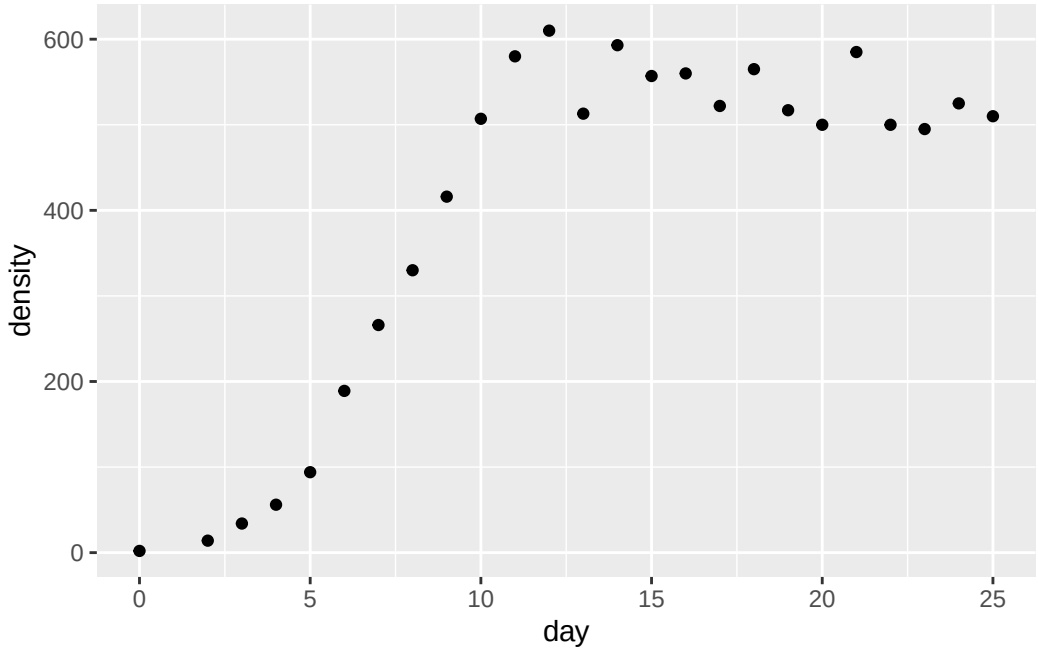
2.2 Logistic Growth

2.2.1 Density Dependent Birth and Death Rates

In a classic series of experiments, Gause collected counts of *P. Aurelia* over several weeks (Gause 2019; De Vries et al. 2006)

```
aurelia = data.frame(
    day = c(0,2:25),
    density = c(2,14,34,56,94,189,266,330,416,507,580,610,513,593,557,560,522,565,517,500,585
    )

ggplot(aurelia,aes(x=day,y=density)) + geom_point()
```



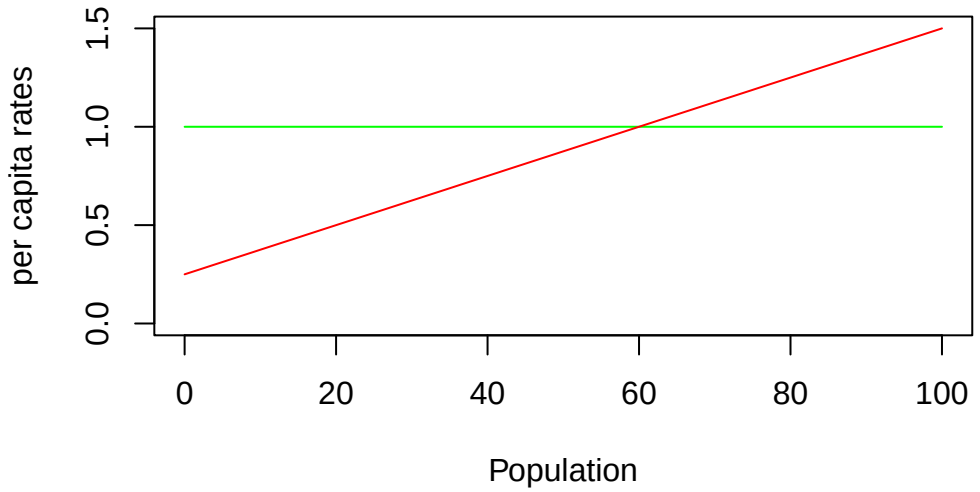
The simplest explanation for the *levelling-off* of the counts is that competition or interference between individuals results in a reduction in fecundity and/or survival as density increases.

2.2.2 Logistic Growth

$$\frac{dN}{dt} = bN - (d + aN)N$$

```
b = 1
d = .25
K = 60
a = (b-d)/K
pcBirth = function(N) rep(b,length=length(N))
pcDeath = function(N) d + a*N
N = seq(0,100,length=100)
```

```
plot(N,pcBirth(N),type='l',col='green',ylab = "per capita rates", xlab = "Population",ylim =
lines(N,pcDeath(N),type='l',col='red')
```



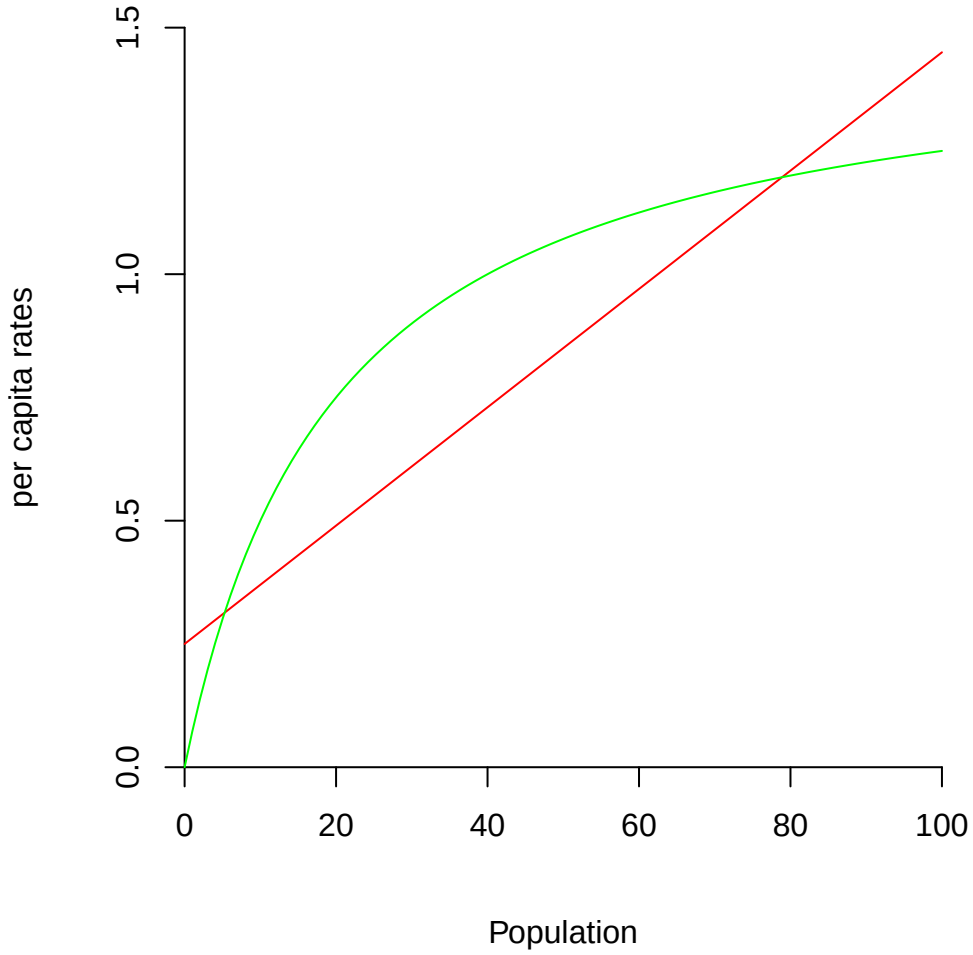
2.3 Allee Effects

2.3.1 Allee Effects

$$\frac{dN}{dt} = \frac{bN^2}{c+N} - (d + aN)N$$

```
b = 1.5
d = .25
K = 60
Na = 10
a = 0.012
c = 20
pcBirth = function(N) b*N/(c+N)
pcDeath = function(N) d + a*N
N = seq(0,100,length=100)
plot(NULL,
     ylab = "per capita rates",
     xlab = "Population",
     xlim = c(0,100),
     ylim = c(0,1.5),
     xaxt="n",yaxt="n",frame.plot=FALSE
     )
axis(1,pos=0)
```

```
axis(2,pos=0)
lines(N,pcDeath(N),type='l',col='red')
lines(N,pcBirth(N),type='l',col='green')
```



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3 Interactions between species

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3.1 Predator-Prey Dynamics

3.1.0.1 Volterra's question

Alfred Lotka (PNAS 1920 6(7):410-415) and Vito Volterra (1926) independently proposed a simple predator-prey model. Lotka was motivated by previous work on rhythmic chemical reactions and oscillations in populations. Volterra was motivated by a question posed by Umberto D'Ancona, a marine biologist who later became Volterra's son-in-law. D'Ancona was curious if Volterra could use mathematical modelling to explain why the proportion of predatory fish in catches increased when fishing effort decreased during the First World War, and then decreased again when fishing resumed.

3.1.1 The Lotka-Volterra Model {.t}

- The classic Lotka-Volterra model is based on a few simple assumptions.

1. Prey grow exponentially at per capita rate r in absence of the predator.
2. Prey are killed at a constant per capita rate a per predator.
3. Each prey killed due to predation gives rise to c predators.
4. Predator death is at a constant per capita rate m.

- The original model consists of a pair of differential equations.

$$\frac{dN}{dt} = rN - aNP,$$
$$\frac{dP}{dt} = caNP - mP.$$

- Here N and P are the prey and predator densities, respectively.

3.1.2 Lotka-Volterra Analysis

$$\frac{dP}{dN} = \frac{(caN - m)P}{(r - aP)N}$$

Separating variables and integrating shows the solutions are level curves of

$$\Psi(N, P) = \frac{ca}{r}N - \frac{m}{r}\log\left(\frac{ca}{r}N\right) + \frac{a}{r}P - \log\left(\frac{a}{r}P\right)$$

3.1.3 The Volterra Predator Prey Model

In 1931, Volterra published an analysis of a predator-prey model with a prey carrying capacity:

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right) - aNP, \tag{1}$$

$$\frac{dP}{dt} = caNP - mP. \tag{2}$$

These equations represent a similar vector field to the original Lotka-Volterra model. The N-nullcline is no longer horizontal, but has the equation $aP = r - rN/K$. The interior equilibrium is at $N = m/a$, $P = \frac{r}{a}\left(1 - \frac{m}{aK}\right)$, which is positive only if $K > \frac{m}{a}$.

3.1.4 The Rosenzweig-MacArthur Model

Rosenweig and MacArthur (1963) are credited with the addition of a hyperbolic functional response to the classic predator-prey model.

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right) - \frac{aNP}{1 + bN},$$

$$\frac{dP}{dt} = c\frac{aNP}{1 + bN} - mP.$$

3.1.5 The Functional Response

Turchin (2003) provides an excellent discussion of the various responses of predators to changes in prey populations. Consider a general predation model of the form

$$\frac{dN}{dt} = NF(N) - H(N, P)P,$$

$$\frac{dP}{dt} = G(N, P)P - mP.$$

The three functions are usually referred to as

- the net per capita growth rate F, which is assumed independent of predation,
- the ***functional response*** H, which is the rate individual predators kill prey, and
- the ***numerical response*** G, which models the dependence of the per capita predator growth rate on population densities.

3.1.6 Holling's Functional Response Types

Holling (1959) introduced a simple classification of functional responses of predators into type I, II, or III. Mathematically, type I and II are similar, and represent a saturating response.

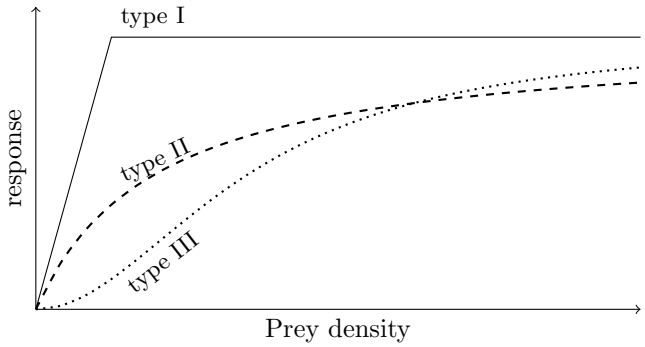


Figure 1: Holling's Functional Response types

3.2 Competition

3.2.1 The Classic Volterra Competition Model

$$\begin{aligned}\frac{dN_1}{dt} &= N_1\left(1 - a_{11}N_1 - a_{12}N_2\right),\\ \frac{dN_2}{dt} &= N_2\left(1 - a_{21}N_1 - a_{22}N_2\right).\end{aligned}$$

Two species in competition:

- a_{ii} measures within-species competition, $i \in \{1, 2\}$.
- a_{ij} measures between-species competition, $i \in \{1, 2\}$, $j \neq i$.

3.3 Virus Dynamics

3.3.1 Classic In-host infection model

$$\begin{aligned}&\text{susceptible host cell:} && \frac{dT}{dt} = \lambda - \mu T - \beta TV\\ &\text{infected host cell:} && \frac{dI}{dt} = \beta TV - \delta I\\ &\text{free virus:} && \frac{dV}{dt} = pI - cV - \beta_v TV\end{aligned}$$

. . .

Infection-free equilbrium: $(T_o = \frac{\lambda}{\mu}, 0, 0)$

. . .

$$\frac{d}{dt}\begin{pmatrix} T \\ I \\ V \end{pmatrix} = \begin{pmatrix} -\mu & 0 & -\beta T_o \\ 0 & -\delta & \beta T_o \\ 0 & p & -(c+\beta_v T_o) \end{pmatrix}\begin{pmatrix} T-T_o \\ I \\ V \end{pmatrix}$$

. . .

$$\mathcal{R}_0 = \frac{p\beta T_o}{\delta(c+\beta_v T_o)}$$

3.3.2 Classic In-host infection model

- The Jacobian matrix has two diagonal blocks.
- The upper left block relates to the invariant infection-free axis, $(T, 0, 0)$.
- The lower right block relates to the invasion of the infection-free equilibrium by the virus.

$$\begin{pmatrix} -\delta & \beta T_o \\ p & -(c+\beta_v T_o) \end{pmatrix} = \begin{pmatrix} 0 & 0 \\ p & 0 \end{pmatrix} - \begin{pmatrix} \delta & -\beta T_o \\ 0 & c+\beta_v T_o \end{pmatrix}$$

The basic reproduction number of the virus is the leading eigenvalue of

$$\begin{pmatrix} 0 & 0 \\ p & 0 \end{pmatrix}\begin{pmatrix} \delta & -\beta T_o \\ 0 & c+\beta_v T_o \end{pmatrix}^{-1}$$

$$\mathcal{R}_0 = \frac{p\beta T_o}{\delta(c+\beta_v T_o)}$$

3.3.3 Virus Dynamics with a Single Eclipse Stage

susceptible host cell:	$\frac{dT}{dt} = \lambda - \mu T - \beta TV$
eclipse-stage:	$\frac{dE}{dt} = \beta TV - \alpha E$
infectious-stage:	$\frac{dI}{dt} = \alpha E - \delta I$
free virus:	$\frac{dV}{dt} = pI - cV - \beta_v TV$

3.3.4 Virus Dynamics with Two Eclipse Stages

$$
\begin{aligned}
&\text{susceptible host cell:} && \frac{dT}{dt} = \lambda - \mu T - \beta TV \\
&\text{eclipse-stage-1:} && \frac{dE_1}{dt} = \beta TV - \alpha E_1 \\
&\text{eclipse-stage-2:} && \frac{dE_2}{dt} = \alpha E_1 - \alpha_2 \\
&\text{infectious-stage:} && \frac{dI}{dt} = \alpha E_2 - \delta I \\
&\text{free virus:} && \frac{dV}{dt} = pI - cV - \beta_v TV
\end{aligned}
$$

3.3.5 Virus Dynamics with Three Eclipse Stages

$$
\begin{aligned}
&\text{susceptible host cell:} && \frac{dT}{dt} = \lambda - \mu T - \beta TV \\
&\text{eclipse-stage-1:} && \frac{dE_1}{dt} = \beta TV - \alpha E_1 \\
&\text{eclipse-stage-2:} && \frac{dE_2}{dt} = \alpha E_1 - \alpha_2 \\
&\text{eclipse-stage-3:} && \frac{dE_3}{dt} = \alpha E_2 - \alpha_3 \\
&\text{infectious-stage:} && \frac{dI}{dt} = \alpha E_3 - \delta I \\
&\text{free virus:} && \frac{dV}{dt} = pI - cV - \beta_v TV
\end{aligned}
$$

3.3.6 Virus Dynamics with Four Eclipse Stages

$$
\begin{aligned}
\text{susceptible host cell:} \quad & \frac{dT}{dt} = \lambda - \mu T - \beta TV \\
\text{eclipse-stage-1:} \quad & \frac{dE_1}{dt} = \beta TV - \alpha E_1 \\
\text{eclipse-stage-2:} \quad & \frac{dE_2}{dt} = \alpha E_1 - \alpha_2 \\
\text{eclipse-stage-3:} \quad & \frac{dE_3}{dt} = \alpha E_2 - \alpha_3 \\
\text{eclipse-stage-4:} \quad & \frac{dE_4}{dt} = \alpha E_3 - \alpha_4 \\
\text{infectious-stage:} \quad & \frac{dI}{dt} = \alpha E_4 - \delta I \\
\text{free virus:} \quad & \frac{dV}{dt} = pI - cV - \beta_v TV
\end{aligned}
$$

3.3.7 Virus Dynamics with a Distributed Eclipse Stage

A common approach to modelling eclipse phases and staged progression is to use a *linear chain* of exponentially distributed stages. These appear in simple ode models as

$$
\begin{aligned}
T' &= \lambda - \mu T - \beta TV \\
I' &= AI + \beta TV \\
V' &= PI - cV - \beta_v TV
\end{aligned}
$$

where I, T and V are densities of infected cells, susceptible target cells and virus. With the simple one-stage model, I is a scalar. In the n-stage model, I is a vector of length n, A is an $n \times n$ *progression* matrix and p is a vector of *budding rates*.

Shiny demo

3.3.8 Classic Immune Model: Cellular

$$
\begin{aligned}
\text{susceptible host cell:} \quad & \frac{dT}{dt} = \lambda - \mu T - \beta TV \\
\text{infected host cell:} \quad & \frac{dI}{dt} = \beta TV - \delta I - bIB \\
\text{free virus:} \quad & \frac{dV}{dt} = pI - cV \\
\text{immune cells:} \quad & \frac{dB}{dt} = aIB - mB
\end{aligned}
$$

(See Ciupe and Heffernan 2017; Murase, Sasaki, and Kajiwara 2005)

3.3.9 Classic Immune Model: Humoral

$$
\begin{aligned}
&\text{susceptible host cell:} && \frac{dT}{dt} = \lambda - \mu T - \beta TV \\
&\text{infected host cell:} && \frac{dI}{dt} = \beta TV - \delta I \\
&\text{free virus:} && \frac{dV}{dt} = pI - cV - \beta_v TV - bVB \\
&\text{immune cells:} && \frac{dB}{dt} = aVB - mB
\end{aligned}
$$

. . .

Infection-free equilibrium $(T_o = \lambda/\mu, 0, 0, 0)$.

$$
\begin{pmatrix}
-\mu & 0 & -\beta T_o & 0 \\
0 & -\delta & \beta T_o & 0 \\
0 & p & -c - \beta_v T_o & 0 \\
0 & 0 & 0 & -m
\end{pmatrix}
$$

3.3.10 Classic Immune Model: Humoral – Immune Response

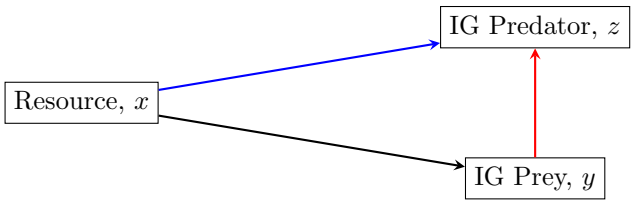
$$
\begin{aligned}
&\text{susceptible host cell:} && \frac{dT}{dt} = \lambda - \mu T - \beta TV \\
&\text{infected host cell:} && \frac{dI}{dt} = \beta TV - \delta I \\
&\text{free virus:} && \frac{dV}{dt} = pI - cV - \beta_v TV - bVB \\
&\text{immune cells:} && \frac{dB}{dt} = aVB - mB
\end{aligned}
$$

Immune-free equilibrium $(T_i, I_i, V_i, 0)$.

$$
\begin{pmatrix}
* & * & * & 0 \\
* & * & * & 0 \\
* & * & * & -bV_i \\
0 & 0 & 0 & aV_i - m
\end{pmatrix}
$$

3.4 Intra-Guild Predation

3.4.1 A Simple Intraguild Predation Model



$$\dot{x} = x(1-x) - xy - \alpha_2 xz,$$

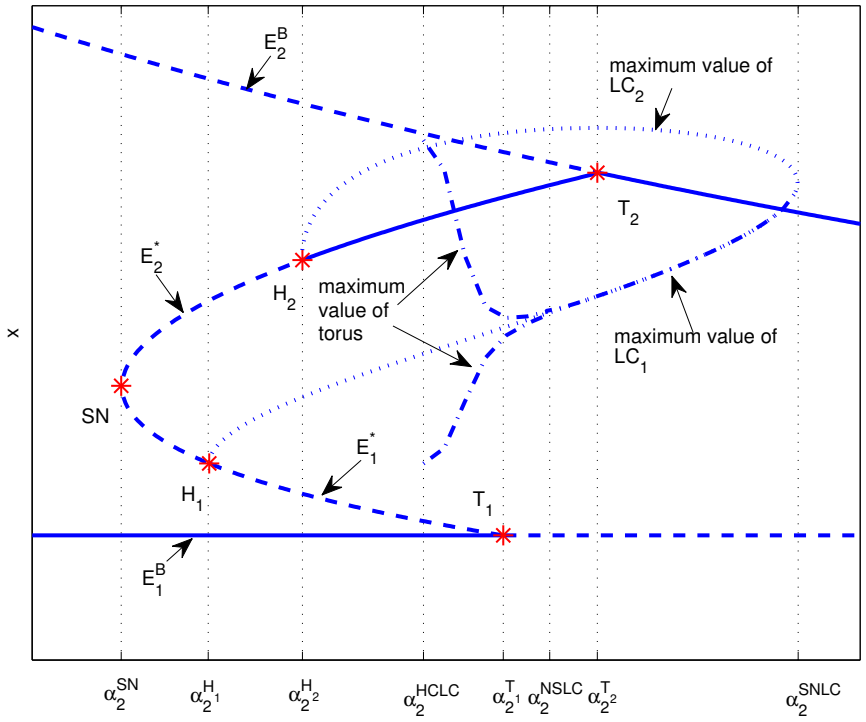
$$\dot{y} = \sigma_1 xy - \frac{yz}{1+\beta_3 y} - \mu_1 y,$$

$$\dot{z} = \frac{\sigma_2 \alpha_2 xz}{1+\beta_2 x} + \frac{\sigma_3 yz}{1+\beta_3 y} - \mu_2 z.$$

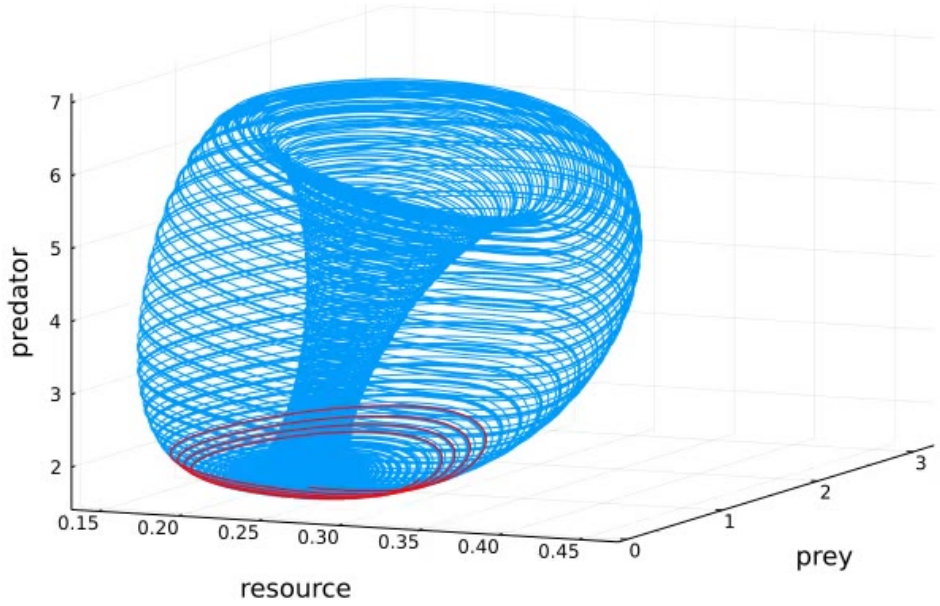
- Type II interaction between IG prey and IG predator (β_3)
- Saturating numerical response of IG predator to resource ($\alpha_2,\ \beta_2$)

(See Hu (2014), Shu et al. (2015))

3.4.2 Intraguild Predation: bifurcation diagram



3.4.3 The Stable Torus



images/igptorus.mp4

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