Handout on

Mathematics for EES students - Multidimensional processes

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November 2, 2023

Contents

References

[BTH05] M. Begon, C.R. Townsend, J.L. Harper (2005) *Ecology — From Individuals to Ecosystems, 4th Ed.* Blackwell Publishing

[K01] M. Kot (2001) *Elements of Mathematical Ecology* Cambridge University Press

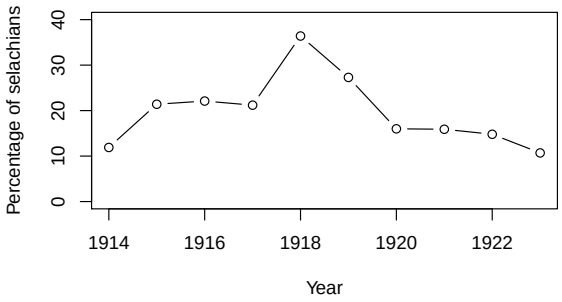
[C00] T.J. Case (2000) *An Illustrated Guide to Theoretical Ecology* Oxford University Press

We refer to Figures in Chapter 10 [BTH05]. I do not include these Figures in this handout for several reasons:

1. to keep the handout small in terms of megabytes and printed pages
2. to avoid copyright issues
3. as it is a good idea to read this and other chapters in the text book
4. as the figures can be downloaded from `http://www.blackwellpublishing.com/begon`

1 A simple predator–prey model by Volterra

Frequency of selachia (old name for sharks and simlar, mostly predators) on several italian fish markets observed by marine biologist Umberto D'Ancona:



- Almost no fishing from 1914 to 1918 (World War I)
- Why does reduced fishing lead to an increase of predators relative to prey?

D'Ancona's father in law, the mathematician Vito Volterra tried to answer this question with a simple mathematical model:

$N(t)$ prey population size

$P(t)$ predator population size

$$\begin{aligned} \frac{dN}{dt} &= rN - cPN \;=\; N \cdot (r - cP) \\ \frac{dP}{dt} &= bNP - mP \;=\; P \cdot (bN - m) \end{aligned}$$

- If $P \equiv 0$, N grows exponentially
- If $N \equiv 0$, P shrinks exponentially
- effects of N and P to each other are *mass-action* terms

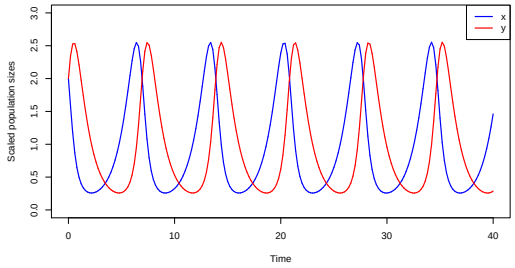
For the analysis, get rid of two parameters by changing variables:

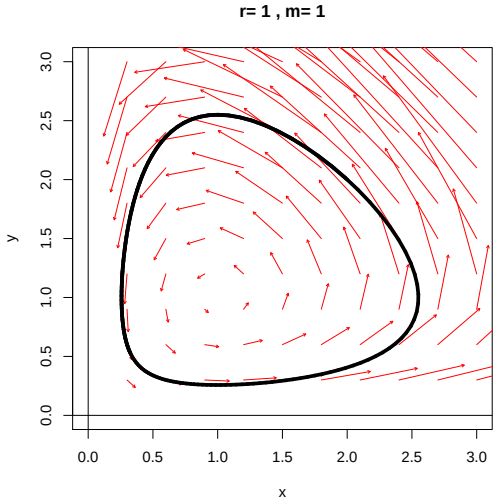
$$\begin{aligned} x &:= \frac{b}{m}N \\ y &:= \frac{c}{r}P \end{aligned}$$

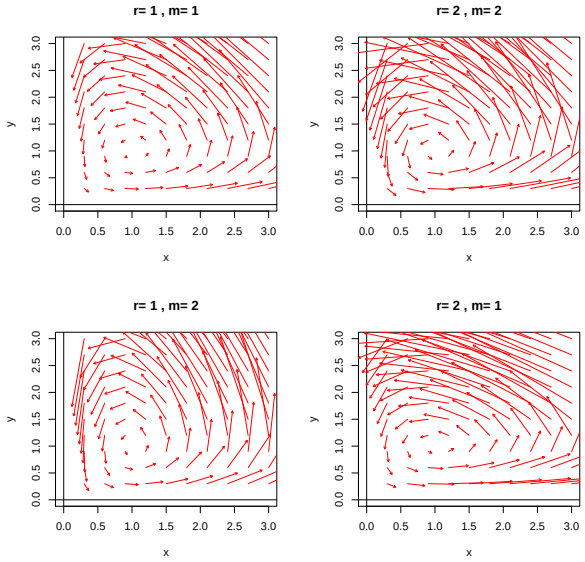
$\Rightarrow$

$$\begin{aligned} \frac{dx}{dt} &= r(1-y)x \\ \frac{dy}{dt} &= m(x-1)y \end{aligned}$$

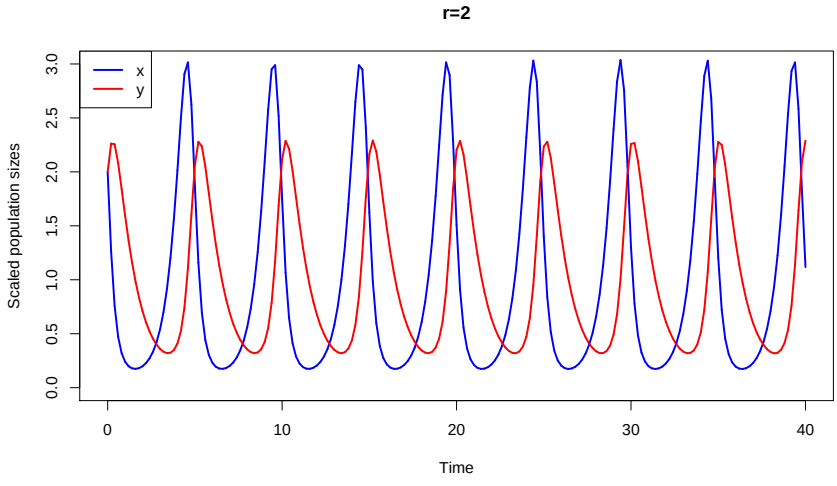
$$\frac{dx}{dt} = r(1-y)x$$
$$\frac{dy}{dt} = m(x-1)y$$







$$\frac{dx}{dt} = r(1-y)x \qquad \frac{dy}{dt} = m(x-1)y$$



Helpful for the anaylsis of the of the system are the

fixed points: points $\binom{x}{y}$ with

$$\frac{dx}{dt} = \frac{dy}{dt} = 0,$$

that is, if the system starts there, it will stay there

isoclines: lines (or curves) on which either

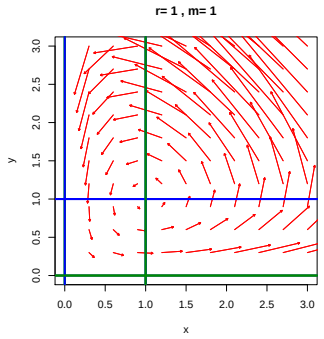
$$\frac{dx}{dt} = 0 \quad \text{or} \quad \frac{dy}{dt} = 0$$

or both

finding isoclines and fixed points

$$0 = \frac{dx}{dt} = r(1-y)x \quad \Rightarrow x = 0 \text{ or } y = 1$$

$$0 = \frac{dy}{dt} = m(x-1)y \quad \Rightarrow x = 1 \text{ or } y = 0$$



Fixed points:

$$\binom{x}{y} = \binom{0}{0}$$

and

$$\binom{x}{y} = \binom{1}{1}$$

Stability of these two fixed points

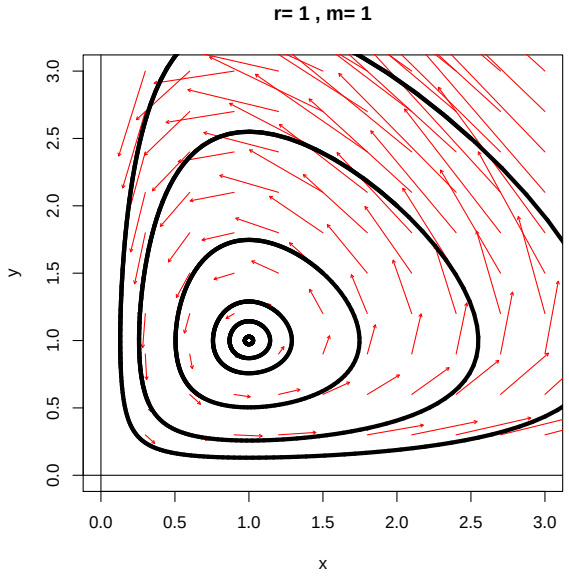
Remeber that *local stability* means that if the system starts very close to the fixed point it will move to it.

Is $\binom{0}{0}$ locally stable?

$$\left.\frac{dx}{dt}\right|_{\binom{0}{0}} \approx rx \qquad \left.\frac{dy}{dt}\right|_{\binom{0}{0}} \approx -my$$

It is not stable as it will be left into direction of x (leading to exponential growth of host population).

The mathematical analysis of $\binom{1}{1}$ is a bit more subtle, see [K01]. In fact, if the system starts close to this fixed point, it will stay on an orbit around it.



Note that on each orbit, movement is faster for higher values of x and y. Volterra could show that on each orbit, when averaged over time:

$$\text{average}(x) = 1 \quad \text{and} \quad \text{average}(y) = 1$$

Thus, for the average abundance of predator or prey over a longer time period, the starting point is irrelevant.

Average frequency in original scaling (fixed point):

$$\begin{aligned} \frac{dN}{dt} &= N \cdot (r - cP) \\ \frac{dP}{dt} &= P \cdot (bN - m) \end{aligned}$$

$$\text{average}(N) = \frac{m}{b}, \qquad \text{average}(P) = \frac{r}{c}$$

Back to our fish question - Model effect of fishing by additional death rate:

$$\begin{aligned} \frac{dN}{dt} &= N \cdot (r - cP - d) \\ \frac{dP}{dt} &= P \cdot (bN - m - d) \end{aligned}$$

$$\text{average}(N) = \frac{m + d}{b}, \qquad \text{average}(P) = \frac{r - d}{c}$$

Thus, Volterra's simple model even predicts that fishing increases the abundance of prey fish!

Maybe, this model can explain why the usage of pesticides sometimes leads to an increase in the abundance of the pest.

2 Should we believe in these oscillations?

Three questions:

1. Do we observe long-term oscillations in nature? See Figure 10.1 in [BTH05]

2. Does the model fit these data? E.g. in the hare–lynx system the hare is also prey of other predators and predator of its plant food. This makes thorough statistical analysis very complex. Time-series analysis of Lynx abundance given hare data fits well to predator–prey model. *GENERAL PROBLEM:* Data can never confirm that a model is valid! All models are wrong!

3. Are these osciallations still predicted if we make the model a bit more realistic?

2.1 Intraspecific competition

Lotka–Volterra model for predator–prey with intraspecific competition:

$$\begin{aligned} \frac{dN}{dt} &= N \cdot (r - cP - kN) \\ \frac{dP}{dt} &= P \cdot (bN - m - gP) \end{aligned}$$

non-trivial isoclines are given by

$$\begin{aligned} P &= \frac{r - kN}{c} \\ P &= \frac{bN - m}{g} \end{aligned}$$

If non-trivial equilibrium with $P \neq 0$ exists, it must be

$$(N, P) = \left(\frac{gr + cm}{cb + gk}, \frac{br - mk}{cb + gk} \right)$$

Thus, coexistence is only possible if $br > mk$ or, equivalently,

$$\frac{b}{m} > \frac{k}{r}$$

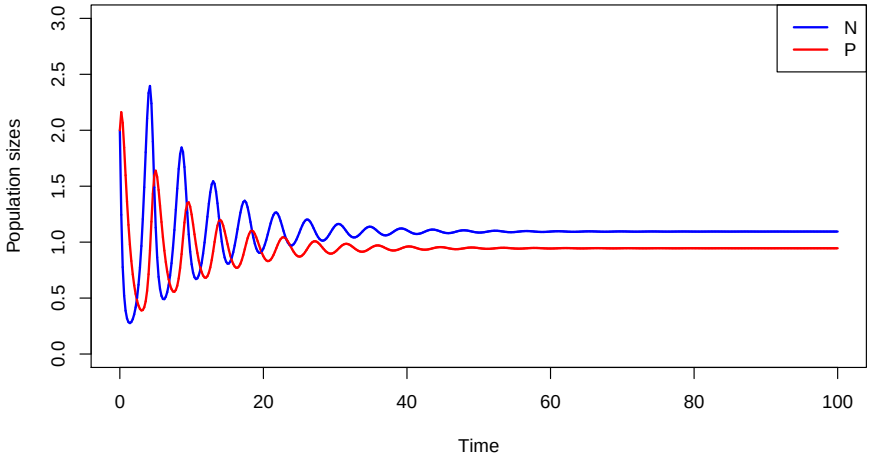
What if $P = 0$ and $N \neq 0$?
In this case

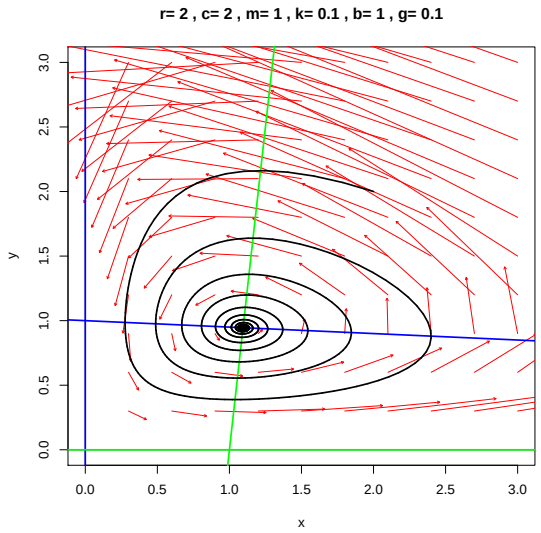
$$\frac{dN}{dt} = N \cdot (r - cP - kN) = N \cdot (r - kN)$$

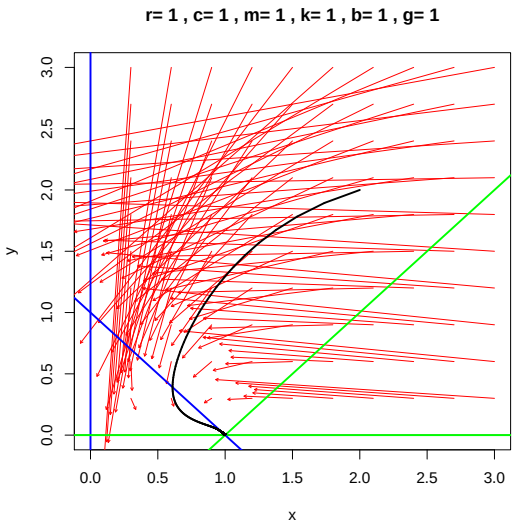
and we obtain the fixed point $N = r/k$.

Furthermore: If $0 < N < r/k$ then $\frac{dN}{dt} = N \cdot (r - kN) > N \cdot (r - k\frac{r}{k})) = 0$.
If $N > r/k$ then $\frac{dN}{dt} = N \cdot (r - kN) < N \cdot (r - k\frac{r}{k})) = 0$.

Therefore, $(N, P) = (r/k, 0)$ is a stable fixed point and $\frac{r}{k}$ is the *carrying capacity* of the prey population if there are no predators, that is the population size to which the system will converge, independently of the initial size $N > 0$.


N
P
Population sizes
0.0
0.5
1.0
1.5
2.0
2.5
3.0
0
20
40
60
80
100
Time


r= 2 , c= 2 , m= 1 , k= 0.1 , b= 1 , g= 0.1
y
x
0.0
0.5
1.0
1.5
2.0
2.5
3.0


r= 1 , c= 1 , m= 1 , k= 1 , b= 1 , g= 1
y
x
0.0
0.5
1.0
1.5
2.0
2.5
3.0

2.2 When predators eat only as much as they can

Model for type II functional response:

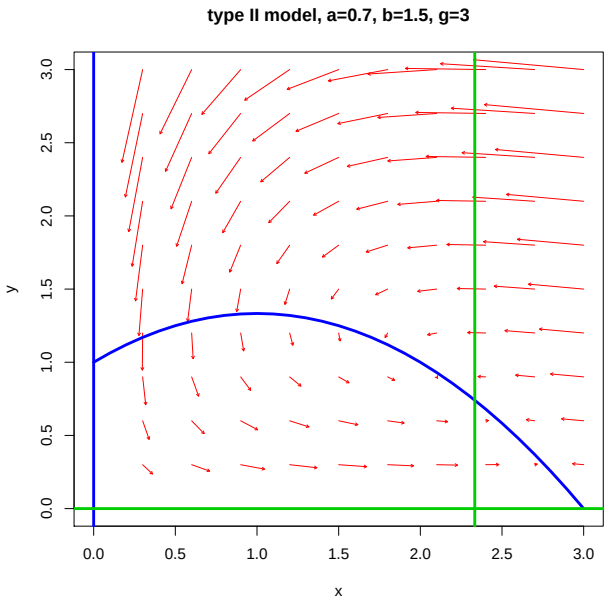
$$\begin{aligned} \frac{dN}{dT} &= rN\left(1-\frac{N}{K}\right)-\frac{cNP}{a+N} \\ \frac{dP}{dT} &= \frac{bNP}{a+N}-mP \end{aligned}$$

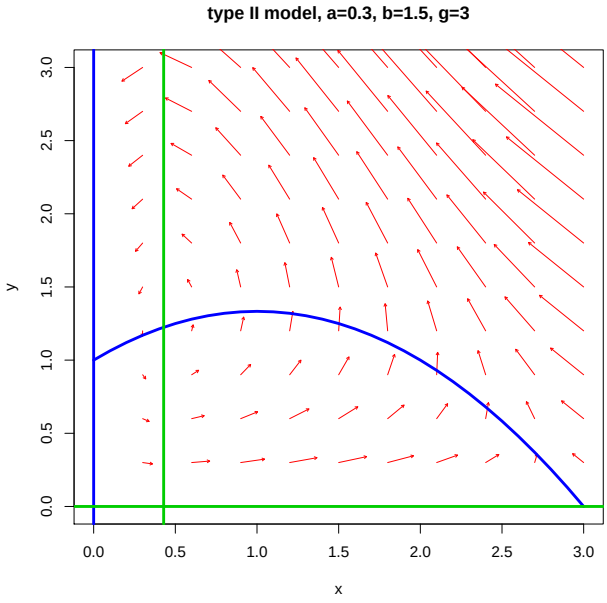
rescaling the variables and setting

$$N=ax \quad P=r\frac{a}{c}y \quad T=\frac{1}{r}t \quad \alpha=\frac{m}{b} \quad \beta=\frac{b}{r} \quad \gamma=\frac{K}{a}$$

leads to

$$\begin{aligned} \frac{dx}{dt} &= x\cdot\left(1-\frac{x}{\gamma}-\frac{y}{1+x}\right) \\ \frac{dy}{dt} &= \beta\cdot\left(\frac{x}{1+x}-\alpha\right)\cdot y \end{aligned}$$





Watch the simulation in the R script!
isocline $\frac{dx}{dt} = 0$:

$$x = 0 \qquad y = (1+x)\left(1 - \frac{x}{\gamma}\right)$$

isocline $\frac{dy}{dt} = 0$:

$$y = 0 \qquad x = \frac{\alpha}{1-\alpha}$$

non-trivial fixed point is stable if and only if

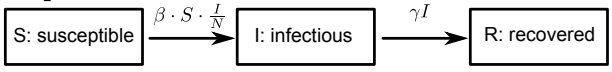
$$\frac{\alpha}{1-\alpha} > \arg\max_x (1+x)\left(1 - \frac{x}{\gamma}\right)$$

See fig 10.6 to 10.12 in [BTH05]
Other reasons why oscillations can be damped:

- spatial structure: local oscillation are averaged
- role of stochasticity?

3 The SIR model revisited

A simple version of the SIR model

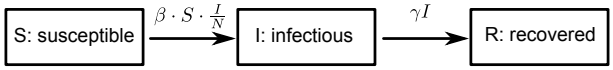


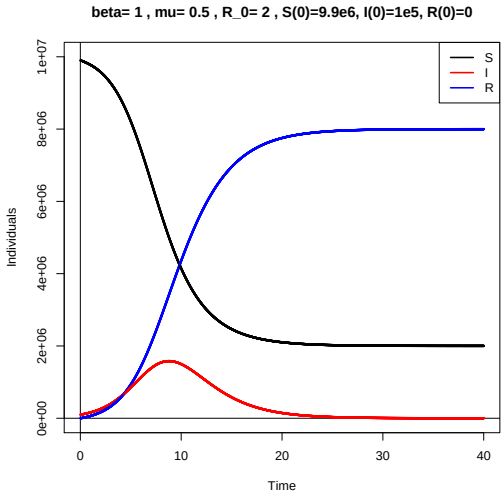
$$N = S + R + I \quad , \qquad \frac{dS}{dt} = -\frac{\beta \cdot S \cdot I}{N} \quad , \qquad \frac{dI}{dt} = \frac{\beta \cdot S \cdot I}{N} - \gamma \cdot I \quad , \qquad \frac{dR}{dt} = \gamma \cdot I$$

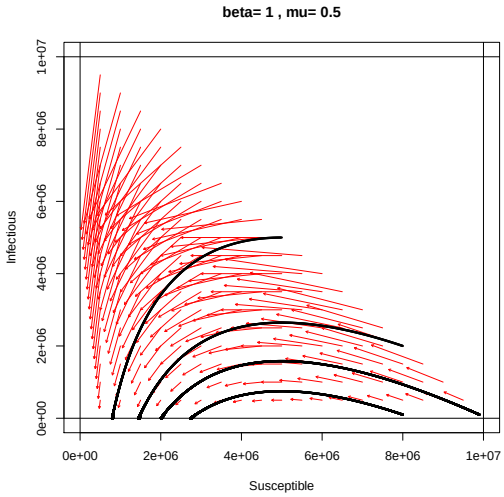
$\mathcal{R}_0 = \beta/\gamma$ is the basic reproduction rate, that is, the average number of other individuals infected by one infected indivdual when (almost) everybody is susceptible.

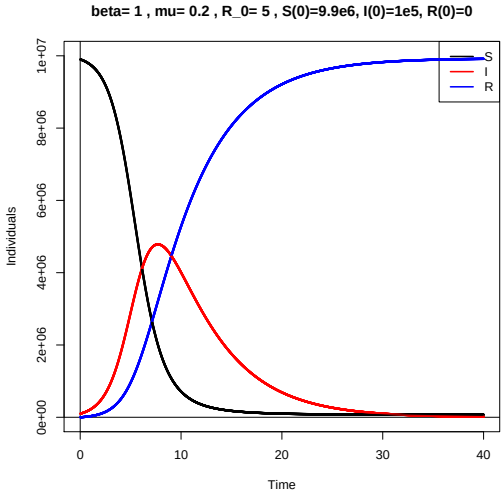
As N is constant and $R(t) = N - S(t) - I(t)$, the model is effectively 2-dimensional, as each state is defined by $(S(t), I(t))$.

A points (s, i) is a fixed point if and only if $i = 0$.









$$N = S + R + I \quad , \qquad S' = -\frac{\beta \cdot S \cdot I}{N} \quad , \qquad I' = \frac{\beta \cdot S \cdot I}{N} - \gamma \cdot I \quad , \qquad R' = \gamma \cdot I$$

- Even for the simple SIR differential equations there is no simple closed-form solution
- But we can derive an equation for $S(\infty) = \lim_{t\to\infty} S(t)$, i.e. number of never-infected.

$$(1) \quad S' = -\frac{\beta \cdot S \cdot I}{N} \quad , \qquad (2) \quad I' = \frac{\beta \cdot S \cdot I}{N} - \gamma \cdot I \quad , \qquad (3) \quad R' = \gamma \cdot I$$

$$\begin{aligned}
(1) \quad &\Rightarrow \quad \frac{\beta}{N} I(t) = -\frac{S'(t)}{S(t)} = -\frac{d \ln(S(t))}{dt} \quad \Rightarrow \quad \frac{\beta}{N} \int_0^\infty I(t)dt = -(\ln S(\infty) - \ln(S(0))) \\
(1)\&(2) \quad &\Rightarrow \quad S'(t) + I'(t) = -\gamma I(t) \\
&\Rightarrow \quad \gamma \int_0^\infty I(t)dt \;=\; [-S(\infty) - I(\infty)] - [-S(0) - I(0)] \;=\; S(0) + I(0) - S(\infty)
\end{aligned}$$

Together, this implies:

$$\ln\left(\frac{S(0)}{S(\infty)}\right) = \frac{\beta}{N} \int_0^\infty I(t)dt = \frac{\beta}{\gamma N} \cdot (S(0) + I(0) - S(\infty)) = \frac{\mathcal{R}_0}{N} \cdot (S(0) + I(0) - S(\infty))$$

Given $S(0)$, $I(0)$, N (or $R(0)$) and the basic reproduction rate $\mathcal{R}_0$, there is a unique solution $S(\infty)$ for

$$\ln\left(\frac{S(0)}{S(\infty)}\right) = \frac{\mathcal{R}_0}{N} \cdot (S(0) + I(0) - S(\infty)).$$

Note: If $R(0) = 0$ and thus $S(0) + I(0) = N$, the above equation simplifies to

$$\ln\left(\frac{S(0)}{S(\infty)}\right) = \mathcal{R}_0 \cdot \left(1 - \frac{S(\infty)}{N}\right).$$

Conclusion: The parameter that really matters is the basic reproduction rate $\mathcal{R}_0$.

Literature on the SIR model and its extensions

References

[KM27] W.O. Kermack, A.G. McKendrick (1927) Contributions to the mathematical theory of epidemics *I. Proc. R. Soc. A* 115:700–721 (reprinted in *Bulletin of Mathematical Biology* 53 (1991) 33–55).

[CB09] G. Chowell, F. Brauer (2009) The Basic Reproduction Number of Infectious Diseases: Computation and Estimation Using Compartmental Epidemic Models In: G. Chowell et al. (eds.), *Mathematical and Statistical Estimation Approaches in Epidemiology*, Springer.

4 Conclusions

General aims of modelling in ecology and evolutionary biology:

- understand ecological mechanisms theoretically/mathematically
- but also intuitively/biologically
- how intuitive and theoretical predictions correspond to each other
- how robust these predictions are against violations of basic assumptions
- check in a rigorous way whether an intuitive explanation really make sense
- theoretical models (but rather probabilistic ones) are the basis of data analyses

Some of the thing you should be able to explain:

- Meaning of differential equations and how to do calculations
- Meaning of each parameter in Lotka-Volterra equations and their generalizations
- How to add a certain mechanisms to Lotka-Volterra equations
- How to calculate isoclines and fixed points
- Local stability of fixed points and how to analyse it
- What can damp oscillations in predator–prey dynamics
- Consequences of different functional response assumptions
- Model assumptions and predictions of the simple SIR model.
- General aims of mathematical modelling in biology