Modeling Population Dynamics in Biology:
Answers to questions

Rob J. de Boer
Theoretical Biology & Bioinformatics
Utrecht University
2018
This addendum provides short answers to the questions in the ebook “Modeling Population Dynamics in Biology”. Since many of the questions can be approached by using several different models, these answers are typically just one of the many possible answers. When you get stuck on a particular question, you may peek into its answer to obtain a hint enabling you to proceed. When you are done with a question, use this addendum to check if your own answer is correct and complete. Do not give up if your own answer is different, as it could very well be correct (and hopefully an even better). Please report errors and suggestions for improvement, for instance by emailing me Rob de Boer at r.j.deboer@uu.nl.

Ebook publicly available at:
http://tbb.bio.uu.nl/rdb/books/bm.pdf and ./bmAnswers.pdf
Answers to Chapter 2

Question 2.1. Red blood cells
Figure made with `blood.R`:

a. \( \frac{dN}{dt} = m - dN \).
b. See Panel (a)
c. See Panel (b)

Question 2.2. Pesticides on apples
Figure made with the previous version of Grind:

a. See Panel (a)
b. \( \bar{P} = \frac{\sigma}{\delta} \).
c. The model becomes \( \frac{dP}{dt} = -\delta P \) with the initial condition \( P(0) = \frac{\sigma}{\delta} \). Solving \( P(0)/2 = P(0)e^{-\delta t} \) yields \( t_{1/2} = \ln[2]/\delta \).
d. From \( \frac{dP}{dt} = 2\sigma - \delta P \) with \( \bar{P} = 2\sigma/\delta \), one obtains the same \( \ln 2/\delta \) days for the half life.
e. From 50 = \( \ln 2/\delta \) one obtains \( \delta = 0.014 \) per day.

Question 2.3. Bacterial growth
a. \( t = \ln[2]/r \).
b. From \( rB - kNB = 0 \) we neglect the trivial \( B = 0 \) solution to obtain \( N = \frac{r}{k} \).
c. The dimension of \( r \) is per hour. Since the total term \( kNB \) has dimension “number of bacteria per ml per hour”, the dimension of \( k \) should be “per neutrophil per ml per hour”. This can also be checked from the expression \( N = \frac{r}{k} \) that should be “neutrophils per ml” on the both the left- and right-hand side.
d. “bacteria per neutrophil per hour”. This is the maximum number of bacteria that one neutrophil can encounter and kill per hour.
e. \( N = \frac{r}{h + B} \), which is a straight line with slope \( r/k \), intersecting the vertical axis at \( N = \frac{rh}{k} \). This line is a nullcline: below this line \( dB/dt > 0 \), and above it \( dB/dt < 0 \).
f. $h$ has the dimension number of bacteria per ml. When $B = h$ the model is $dB/dt = rB - kN/2$, saying the neutrophils are killing at a rate $k/2$, i.e., half their half-maximal killing rate.

**Question 2.4. Physics**

a. The dimension of $v$ is m/s and that of $a$ is m/s$^2$.

b. For the plastic we write $dp/dt = k(t) = at + k(0)$, and the corresponding solution is $p(t) = \frac{1}{2}at^2 + k(0)t$.

c. No the amount of plastic will continue to increase at an accelerating rate.

**Answers to Chapter 3**

**Question 3.1. Carrying capacity**

a. The per capita birth rate is minimal when a population approaches its carrying capacity.

b. the per capita death rate is maximal when a population approaches its carrying capacity.

c. The individual well-being is expected to be best in an expanding population: the per capita birth rate is maximal and the per capita death rate is minimal.

d. With $dN/dt = rN[1 - N/(k\sqrt{N})] = 0$ one obtains the carrying capacity from $N/(k\sqrt{N}) = 1$ or $\sqrt{N} = k$ giving $\bar{N} = k^2$ which is still a finite carrying capacity, at which circumstances are poor. For the best circumstances the population has to remain below its carrying capacity.

**Question 3.2. Freitas**

a. No, the steady state of $dB/dt = m - dB$ is $\bar{B} = m/d$. In such a model the number of peripheral B cells remains proportional to the number of bone marrow precursors (which here determines $m$).

b. For instance with density dependent death, $dB/dt = m - dB(1 + eB)$, or with density dependent production, $dB/dt = m/(1 + eB) - dB$.

c. Yes clearly, in the absence of homeostasis the steady number of peripheral B cells is proportional to the number of bone marrow precursors.

d. No, it is accounting for a steady state, but not for density dependent population regulation.

**Question 3.3. Overfishing herring**

Figure made with the previous version of Grind:

(a) $dN/dt = f(N) = rN(1 - N/K)$, which is a parabola crossing the horizontal axis at $N = 0$ and $N = K$. See Panel (a).

b. The maximum of the function, $f(N) = rN - rN^2/K$, is found by setting its derivative, $\partial_N f = r - 2rN/K$, to zero. This delivers $\bar{N} = K/2$ (see Panel (a)). Substituting this maximum into the population growth function, one obtains the maximum population growth
of \( f(\hat{N}) = rK/4 \).

c. The optimal population size is that yielding maximum growth, i.e., \( N = K/2 \), and the total population growth, \( rK/4 \), could in principle be harvested.

d. \( dN/dt = rN(1 - N/K) - rK/4 \).

e. See Panel (b): at the maximum harvest there is a steady state where \( dN/dt = 0 \). Starting at \( N = K \) and allowing for this maximum harvest, one mathematically approaches this steady at \( N = K/2 \). This steady state is not structurally stable, however, as any disturbance of the population size, bringing it below the steady state at \( N = K/2 \), will let the fish go extinct.

f. One should never catch the maximum yield. This allows for a stable population size while the population is harvested. See Panel (c). The population remains vulnerable to extinction by large perturbations due to the saddle point at low population densities. In the computer practical you will revisit this problem and discover that by catching an optimal fraction of the population one can on average catch this maximum yield, without threatening the population with extinction.

**Question 3.4. Biofilm**

Figures made with the model *biofilm.R*:

![Graphs](image)

a. The function \( y = \frac{bx}{h+x} \) is an increasing saturation function intersecting the vertical axis in the origin, and the function \( y = d + ex \) is a straight line intersecting the vertical axis in \( y = d \); see Panel (a). When \( d < b \) these lines tend to intersect in two points, where the per capita birth rate equals the per capita death rate. The steady state at low population densities is unstable, and the one at high densities corresponds to the stable carrying capacity.

b. Because the birth function goes from quadratic to linear, and the death function from linear to quadratic, these tend to intersect three times: in the origin, at a low density and at a high density. See Panels (b) and (c) where (b) zooms in at low population densities.

c. We therefore find three steady states, with a stable origin and a stable carrying capacity, and a saddle point in the middle defining the population threshold corresponding to an Allee effect.

d. \( dB/dt = \frac{bB}{1+B/k} - \frac{dB}{1+B/h} \) where we have put the negative density dependence in the birth rate, and the Allee effect in the death rate. The per capita death rate is \( d \) when the population is small, decreases to \( d/2 \) when \( B = h \), and approaches zero when \( B \to \infty \).

**Question 3.5. Stem cells**

a. \( dS/dt = pS(1 - S/K) - dS \), where we could define a time scale of days, i.e., the dimension of \( p \) and \( d \) are \( d^{-1} \), and that of \( K \) is cells. No, the size of the substrate naturally limits the number of stem cells. This equation corresponds to the logistic growth model of Eq. (3.6).

b. Solving \( dS/dt = 0 \) gives the non-trivial solution \( \bar{S} = K(1 - \frac{d}{p}) \), which is smaller than \( K \) because sites are freed up by death.

c. Because the fraction \( S/K \) of the stem cells differentiates one obtains \( dD/dt = \frac{p}{K} S^2 - \delta D \).
d. The production rate is $\frac{R}{S} S^2$, which has the parabolic form of $y = ax^2$. Note that this despite the quadratic form this production has a correct dimension cells $d^{-1}$ because $p$ has a dimension $d^{-1}$, and $K$ cells. The production rate remains bounded, however, because there can be no more than $S = K(1 - \frac{d}{x})$ stem cells, i.e., the maximum production is $p(1 - \frac{d}{x})^2$ cells $d^{-1}$.

**Question 3.6. Generalized logistic growth**

a. The *per capita* growth term in the standard logistic equation is of the form $r(1 - N/K) = r - kN$, where $k = r/K$. Summing *per capita* birth and death rates of the form $b(1 - N/k_b)$ and $d(1 + N/k_d)$, respectively, also yields a *per capita* growth rate of the form $r - kN$, where $r = b - d$ and $k$ is a combination of all four parameters.

b. This would be a *per capita* birth rate of the form $b(1 - (N/k)^m)$, which is concave when $m > 1$ (like blue red line in Fig. 3.3c, and convex when $m < 1$ (like the green line in Fig. 3.3c). The concave shape would mean that the negative density dependence on the birth process kicks in at relatively high population densities, which would be realistic when resources become limiting only after the population has expanded. The convex shape would imply that effect of competition on the birth rate is steepest at low densities, which would be realistic for a population expanding spatially, and growing at its border. Thus, any positive value of $m$ seems legitimate.

c. The death rate would be of the form $d(1 + (N/k)^m)$, which for $m > 1$ would mean that the increase of *per capita* death rate keeps accelerating when the population expands. For $m < 1$ the increase of the *per capita* death rate decelerates with the population size. Both could be realistic and hence any positive value of $m$ seems legitimate.

**Question 3.7. Red blood cells**

Figure made with the model `epo.R`:

![Figure made with the model epo.R](image)

a. Because this should be a declining sigmoid function we start with a Hill function, $y = \frac{1}{1 + (B/h)^n}$. Since $y \to 0$ when $B \to \infty$ we need to add the minimum production rate, $s_0 \simeq 10^9$ cells kg$^{-1}$ d$^{-1}$, leading to something like $y = s_0 + \frac{1}{1 + (B/h)^n}$, and to define a maximum production rate of $s \simeq 10^{10}$ cells kg$^{-1}$ d$^{-1}$, we need to multiply the Hill function with a parameter, $s_1$, to obtain the end result: $f(B) = s_0 + \frac{s_1}{1 + (B/h)^n}$, where $s_0 \simeq 10^9$ and $s = s_0 + s_1 \simeq 10^{10}$ cells kg$^{-1}$ d$^{-1}$. See Panel (a).

b. $dB/dt = s_0 + \frac{s_1}{1 + (B/h)^n} - dB$, where $s_0 \simeq 10^9$, $s_1 \simeq 19 \times 10^9$, and $d = 1/120$.

c. In the absence of EPO the model simplifies to $dB/dt = s_0 - dB$, with steady state $\dot{B} = \frac{s_0}{d} = 1.2 \times 10^{11}$ cells kg$^{-1}$. To be able to run the model in the presence of EPO we have to think about the value of $h$, i.e., the RBC density where the effect of EPO is half maximal. Since the minimum RBC count is $\dot{B} = 1.2 \times 10^{11}$ cells kg$^{-1}$, and the effect of EPO should kick in well before this minimum is reached we set $h = 10^9$ cells kg$^{-1}$. Running the model for these parameters leads to a steady state of $\dot{B} = 1.77 \times 10^{11}$ cells kg$^{-1}$, i.e., there is a minor effect of EPO at the normal steady state.

d. Running the model after halving $s_1$ only leads to a 10% loss of the RBC in the blood, for $s_1 = 4.5 \times 10^9$ one finds $\dot{B} = 1.63 \times 10^{11}$ cells kg$^{-1}$.
Question 3.8. Regression to the mean

a. Since everything is random, the first expectation is that one should find not correlation between the per capita change, \( (N_{t+\Delta} - N_t) / N_t \), and the previous density, \( N_t \).

b. We nevertheless find a significant correlation. Although all \( N_t \) values are random, relatively small \( N_t \) values tend to create a large deviate \( N_{t+\Delta} - N_t \), which is subsequently “boosted” by dividing by a small \( N_t \) value. In statistics this is known as the “regression to the mean” phenomenon. Thus, testing for density dependence in a random time series is expected to lead to statistically significant evidence.

c. This “taught-experiment” illustrating the main message of the Shenk et al. [9] and Freckleton et al. [3] papers tells us that one needs to be careful when searching for evidence for density dependence in time-series data.

Question 3.9. The Fisher equation

a. Starting at position 30, this code creates a wave traveling left- and right-wards. The wave traveling left-wards re-enters the space on the right.

b. If the Allee effect is sufficiently strong and the diffusion sufficiently show it should be possible to stop the wave. Try this!

c. The expected time between divisions in the Smith-Martin model is the sum of the length

Question 3.10. Cell division takes time

a. When \( t < \Delta \) the cells in the A-stage disappear at rate \( dA/dt = -(d + p)A \), whereas those in the B-phase obey \( dB/dt = pA - dB \). Since the two \( pA \) terms cancel each other, summing both delivers \( dN/dt = -dN \), which is a natural results because the cells can only die before the first divided cells appear at \( t = \Delta \). The model with a flexible delay gives very similar results because the \( B_i \) terms cancel each other when the \( dB_i/dt \) equations are summed and \( n \) is sufficiently large such that \( B_n \simeq 0 \).

b. \( dA/dt = -(p + d)A \) in the Smith-Martin model at early time points, i.e., the cells in the A-stage are declining until \( t = \Delta \). Running the Smith-Martin model for a short period of time readily confirms this.

c. The expected time between divisions in the Smith-Martin model is the sum of the length
of the A-stage and B-phase, i.e., $\frac{1}{p} + \Delta$. To compute the corresponding division rate, $p'$, in the simplest ODE model, we just take the inverse of this, i.e., $p' = \frac{1}{1/p + \Delta}$. We can only numerically test how this differs from the division rate, $r + d$, predicted by Eq. (3.19). For $p = 1$ and $\Delta = 0.5$ we obtain that $p' = 1/(1 + 0.5) = 2/3$ and that $r + d = 0.53 + d$. Thus, if the death rate, $d$, is small $p'$ overestimates the asymptotic division rate, and if $d$ is large $p' < r$.

d. The Smith-Martin model approaches the exponential growth model $\frac{dN}{dt} = rN$, which is not different from the $\frac{dN}{dt} = (p - d)N$ model when the parameters are set by Eq. (3.19). When the B-phase is short compared to the length of the A-stage the models will be very similar. The Smith-Martin model is therefore most appropriate for rapidly dividing cells with a division time dominated by the length of the B-phase. An example would be proliferating tumor cells, or lymphocytes during their clonal expansion phase [2].

Question 3.11. Life stages
Figure made with the old version of Grind:

a. For the larvae, $L$, and the adults, $A$, one could write

$$\frac{dL}{dt} = rA - mL - d1LA \quad \text{and} \quad \frac{dA}{dt} = mL - d2A,$$

where $m$ is the maturation of the larvae, and $r$ the reproduction of the adults.

b. The larval nullcline is solved from $dL/dt = rA - mL - d1LA = 0$ giving $A = \frac{mL}{r - d1L}$, which is zero when $L = 0$ and has a vertical asymptote at $L = r/d1$. The slope in the origin is computed from the derivative

$$\frac{m}{r - d1L} + \frac{md1L}{(r - d1L)^2} \quad \text{which for } L = 0 \text{ gives } \frac{m}{r}.$$

See Panel (a). For the adults $\frac{dA}{dt} = mL - d2A = 0$ gives $A = \frac{mL}{r - d2}$, which is a line with slope $m/d2$. If $m/d2 > m/r$ the two nullclines intersect in a non trivial stable steady state. Otherwise the origin is the only steady state (see Panel (b)).

c. Assuming a quasi steady state for the larvae, one has to solve $L$ from $dL/dt = 0$, giving $\hat{L} = \frac{rA}{m + d1A}.$

d. Substituting $\hat{L}$ into the adult equation gives $\frac{dA}{dt} = \frac{mrA}{m + d1A} - d2A$ for the quasi steady state model. This is one of the models with a density dependent birth rate (see Table 3.1).

e. From $A = (m/d2)L$ we get $dL/dt = (r' - m)L - dL^2$ where $r' = rm/d2$ and $d = d1m/d2$, which has the form of a logistic equation.

f. In many insect species the adults live much shorter than the larvae. Then $\frac{dA}{dt} = 0$ would be most realistic.
Question 3.12. Tumor growth
Figure made with the previous version of Grind:

\[ \text{Biomass (A)} \]

\[ \text{per capita growth} \]

\[ (b/d)^2 \]

\[ -d \]

\[ 0 \]

\[ 0 \]

\[ 2 \]

\[ 0 \]

\[ 0 \]

\[ -0.5 \]

\[ 0 \]

\[ 0.5 \]

\[ r = \sqrt{\frac{A}{c'}} = c' \sqrt{A}, \]

where \( c' \) is a new scaling constant. The total growth rate is proportional to the circumference \( 2\pi r \), which after substituting the radius becomes \( 2\pi c' \sqrt{A} = b \sqrt{A} \), where \( b \) is a “birth rate” that is proportional to the square root of the biomass. On the other hand, the total death rate is proportional to the total biomass. A simple model would therefore be \( \frac{dA}{dt} = b \sqrt{A} - dA \).

b. The carrying capacity is solved from \( b \sqrt{A} - dA = 0 \), or \( b - d \sqrt{A} = 0 \) giving \( A = (b/d)^2 \). There is a trivial steady state \( A = 0 \).

c. The per capita growth \( \frac{dA}{dt} = \frac{b}{\sqrt{A}} - d \). Which for \( A \to \infty \) approaches the horizontal asymptote \( -d \), which seems perfectly reasonable (see the Figure). However, for small population sizes, i.e., \( A \to 0 \), the per capita growth rate blows up, which is not a good property of the model.

Answers to Chapter 4

Question 4.1. Density dependent death
Figure made with the previous version of Grind:

\[ \frac{dA}{dt} = \frac{b}{\sqrt{A}} - d \]. Which for \( A \to \infty \) approaches the horizontal asymptote \( -d \), which seems perfectly reasonable (see the Figure). However, for small population sizes, i.e., \( A \to 0 \), the per capita growth rate blows up, which is not a good property of the model.

a. The per capita death rate is \( cN \): see Panel (a)

b. The net per capita growth rate is \( b - cN \): see Panel (b).
c. The steady state is \( \bar{N} = \frac{b}{c} \).

d. Because there is no generation time.

e. The derivative with respect to \( N \) is \( b - 2cN \). Substituting \( N = \frac{b}{c} \) yields \( \lambda = -b < 0 \). Thus the return time \( T_R = \frac{1}{b} \) is fully determined by the birth rate and is independent of the density dependent death rate \( c \).

**Question 4.2. Return time**

a. For \( \frac{dN}{dt} = f(N) = bN(1 - N/k) - dN \) there are two steady states, the origin \( \bar{N} = 0 \), and the carrying capacity \( \bar{N} = k(1 - d/b) = k(1/R_0) \). For the return time of the carrying capacity one computes the derivative \( \partial_N f(N) = b - d - 2bN/k \) and substitutes the steady state value to obtain

\[
\lambda = b - d - \frac{2b}{k} k(1 - d/b) = d - b \quad \text{and} \quad T_R = -\frac{1}{\lambda} = \frac{1}{b - d}.
\]

For \( \frac{dN}{dt} = g(N) = bN - dN(1 + N/K) \) there are also two steady states, the origin \( \bar{N} = 0 \), and the carrying capacity \( \bar{N} = k(b/d - 1) = k(R_0 - 1) \). For the return time of the carrying capacity one computes the derivative \( \partial_N g(N) = b - d - 2dN/k \) and substitutes the steady state value to obtain

\[
\lambda = b - d - \frac{2d}{k} k(b/d - 1) = d - b \quad \text{and} \quad T_R = -\frac{1}{\lambda} = \frac{1}{b - d}.
\]

Thus, in both models the return time decreases when the net rate of increase, \( r = b - d \), increases. This is just the \( r \) versus \( K \)-selected paradigm.

b. For \( \frac{dN}{dt} = f(N) = s - dN \) with steady state \( \bar{N} = s/d \), the derivative \( \partial_N f(N) = -d \), which immediately gives \( \lambda = -d \) and \( T_R = 1/d \).

c. The \( s \) and \( k \) parameters are not rates, but have dimension \([N \text{ time}^{-1}]\) and \([N]\), respectively. Because both depend on the units of the population size, one can always scale the population size such that \( s = 1 \) and \( k = 1 \). For instance, scaling the non-replicating population by its steady state, \( \bar{N} = s/d \), by defining a scaled population as \( n = \frac{s}{d}N \), and hence substitute \( N = \frac{s}{d}n \) in \( \frac{dN}{dt} = s - dN \), one obtains the scaled ODE

\[
\frac{s}{d} \frac{dn}{dt} = s - \frac{s}{d}dn \quad \text{or} \quad \frac{dn}{dt} = d - dn,
\]

see Section [13.5](#).

d. The ODE \( \frac{dN}{dt} = s(1 - N/k) - dN \) can be written as \( \frac{dN}{dt} = s - (s/k + d)N = s - \delta N \), where \( \delta = s/k + d \). This is of the same form as \( \frac{dN}{dt} = s - dN \), and hence the return time is given by \( R_T = \frac{1}{\delta} = \frac{1}{(s/k + d)} \). The parameter \( s \) is now part of the return time because \( s/k \) is a rate.

**Question 4.3. Whales**

Figures made with the model `whales.R`.
To develop a proper model for the whales we have to consider three biological processes: birth, death, and the likelihood of finding a male. Write a model for the number of females, \( N \), in the population, and assume that there is a similar number of males (the true population size would therefore be similar to \( 2N \)). The probability that an individual female finds a male should increases with the number of males, and approach one at large densities of males. A simple saturation function \( p = \frac{N}{(h + N)} \), where \( p \) is the probability, and \( h \) is the population size at which there is a 50% probability of finding a male. Note that a sigmoid Hill function would be inappropriate here because at low densities this probability should increase linearly with the density. To allow for a carrying capacity we have to include negative density dependence in either the birth or the death terms.

a. Assuming density dependent birth one would write something like

\[
\frac{dN}{dt} = \frac{bN}{1 + N/k} \frac{N}{h + N} - dN , \tag{A.4.1}
\]

and assuming density dependent death one could write

\[
\frac{dN}{dt} = bN \frac{N}{h + N} - dN(1 + (N/k)^2) , \tag{A.4.2}
\]

and in reality one could have a combination of the two.

b. The population birth rate (in red) and the death rate (in blue) of Eq. (A.4.1) is depicted in Panel (a). Those of Eq. (A.4.2) are shown in Panel (b).

c. The population growth rates are shown in Panels (c) and (d). The basins of attraction are defined by the intersections by the black line located at \( \frac{dN}{dt} = 0 \).

**Answers to Chapter 5**

**Question 5.1. Sketch the per capita birth rate**
Figure made with the file `birth.R`:

![Graph](image)

**a.** Plotting $y = \frac{b(R_T - cN)}{h + R_T}$ as a function of $N$ needs to be done in several steps. First, $y = 0$ when $N = \frac{R_T}{c}$, i.e., when all nutrients are contained in the cells. At low population densities the population approaches the birth rate $y = \frac{bR_T}{h + R_T}$, and when the saturation constant, $h$, is much smaller than the total resource density, $R_T$, this will approach the maximum birth rate $b$. When $N$ increases the per capita birth rate will decrease. Since the function is of the form $y = b\left(1 - \frac{h}{h + R_T - cN}\right)$, one can see that there is a vertical asymptote at $N = \frac{h + R_T}{c}$, which is located beyond the point, $N = \frac{R_T}{c}$, where $y = 0$. We find the horizontal asymptote by first writing $y = \frac{bR_T(N - c)}{h + R_T(N - c)}$, and then taking the limit $N \to \infty$ to find that $y \to b$. We therefore obtain the concave hyperbolic function depicted above.

**b.** This concave shape is what we considered most realistic in Chapter 3. For instance see Fig. 3.5c.

**Question 5.2. Neutrophils**

Figure made with `neutrophils.R`:

![Graphs](image)

**a.** In Panel (a) the uninfected state is unstable and there is a stable state corresponding to a chronic infection. In Panel (b) the uninfected state is stable, and small infections cannot grow.

**b.** Compare Panel (a) with (b): $\frac{r}{k} < \frac{s}{d}$, which translates the previous condition $kN > r$ now
translates into $\frac{ks}{d} > r$.

c. See Panels (c)–(e). Panel (d) is like (b), and small infections cannot grow. In Panel (e) is like Panel (a), with one stable state corresponding to a chronic infection. In Panel (c) the point $(B, \bar{N}) = (0, s/d)$ is a stable node, the steady state marked by the open circle is a saddle point, and the the steady state marked by the bullet is a stable node.

d. The condition for control is that $\frac{s}{d}$ is larger than the maximum of the parabola (which can be computed by substituting $B = (K - h)/2$ into the equation for the nullcline $N = \frac{r}{d}(h + B)(1 - B/K)$). Additionally, saturated killing creates a threshold density of bacteria above which the bacteria can no be controlled, which corresponds to the saddle point indicated by the open circle in Panel (b).

e. Writing $\frac{dB}{dt} = rB \frac{B}{a + B}(1 - B/K) - \frac{kNB}{h + B}$ makes hardly anything changes around the steady states (see Panels f and g). This partly because small populations of bacteria were already controlled by the neutrophils, i.e., $(B, \bar{N}) = (0, s/d)$ was already stable. Secondly, because the bacteria have no death rate the Allee effect is “weak”, i.e., in the absence of neutrophils small bacteria populations do not decline but grow slowly (which may in fact be realistic).

f. The large transient output from the bone marrow tends to overcome the threshold of the previous model.

Question 5.3. Lotka-Volterra models

a. $dT/dt = rT(1 - T/K) - kTN$ and $dN/dt = aTN - dN$ for the tumor, $T$, and natural killer cells, $N$, respectively.

b. $dS/dt = rS(1 - S/K) - \beta SI$ and $dI/dt = \beta SI - dI$ for the susceptible individuals, $S$, and infected individuals, $I$, respectively.

c. The natural killer cells probably have a maximum killing rate, and a maximum rate of activation, which would change the model to $dT/dt = rT(1 - T/K) - \frac{KTN}{h + T}$ and $dN/dt = \frac{aTN}{h + T} - dN$ (see Chapter 7). The SI model is frequently written as $dS/dt = rS(1 - S/K) - \frac{\beta SI}{S + I}$ and $dI/dt = \frac{\beta SI}{S + I} - dI$, because $\frac{I}{S + I}$ is the fraction of infected individuals in the population (see Chapter 6). This is more natural when the susceptible individuals tend to meet an average number of other people, irrespective of their health status.

Question 5.4. Desert

Figures made with the previous version of Grind:

a. If there is no vegetation one sets $V = 0$ to obtain $dW/dt = a - cW$ with the steady state $\bar{W} = a/c$

b. If there is twice the amount of rain the parameter $a$ becomes $2a$, which means $\bar{W} = 2a/c$.

c. The steady state is now solved from the system $dW/dt = dV/dt = 0$. Since $V = 0$ cancels from $dV/dt = 0$ one obtains the steady state $\bar{W} = e/d$ from the vegetation equation. This is independent of rain and evaporation!

d. The steady state remains $\bar{W} = e/d$ and all the extra water ends up in the vegetation.
The vegetation nullcline is solved from $dV/dt = dWV - eV = 0$ which means that $V = 0$ and $W = e/d$. The water nullcline is solved from $dW/dt = a - bWV - cW = 0$ or $a - cW = bWV$, i.e., $V = \frac{a}{bW} - \frac{c}{b}$, which is a decreasing hyperbolic function with horizontal asymptote $V = -(c/b)$ and vertical asymptote $W = 0$. There are two possibilities: See Panel (a) and (b). The vector field shows steady state $\tilde{W} = a/c$ without a vegetation is an unstable saddle in Panel (a) and is stable in Panel (b). For the non-trivial steady state in Panel (a) we can derive the full Jacobian

$$J = \begin{pmatrix} -b\tilde{V} - c & b\tilde{W} \\ -b\tilde{V} & -b\tilde{W} \end{pmatrix},$$

because $\tilde{W} = \frac{e}{\bar{W}}$, and giving $\text{tr}J = -b\tilde{V} - c < 0$ and $\det J = 0 + bd\tilde{W}\tilde{V} > 0$. One can also retrieve the graphical Jacobian from the local vector field, i.e.,

$$J = \begin{pmatrix} - & - \\ + & 0 \end{pmatrix}$$

also giving $\text{tr}J < 0$ and $\det J > 0$.

Both methods agree that the non-trivial steady state in Panel (a) is stable.

**f.** Increased rainfall increases $a$, which will move the water nullcline up and to the right. Since the vertical vegetation nullcline is unaffected, the amount of water in the soil remains the same, and the vegetation increases.

**Question 5.5. Phages and bacteria**

a. The `lagvalue(tlag)` function returns the values of all 5 variables at time $t - \Delta$.

b. The `fig2B0` data correspond to bacterial growth in the absence of phages, and the `fig2B` data is with phages.

c. Fitting the first data provides a very similar estimate for the consumption rate, $v$.

d. Yes, this looks like a good fit, and the parameter estimates are similar. Since the resistant bacteria are growing slower than predicted, it would have been better to also estimate a fitness cost.

e. No the data are equally well described with an ODE model without a fixed time delay? The value of the eclipse time, $1/\lambda$, is much longer now because it is exponentially distributed.

f. The model has no death rate of the bacteria and no clearance of the phages. Given the short time scale of the experiment this is probably not important.

**Question 5.6. Return time**

We calculate the return time of the non-trivial steady state of the Lotka Volterra model considering both density dependent birth and density dependent death. For simplicity we do this for the case where this equilibrium is a stable spiral point. To save time we first write the model in a general form and compute the return time for this general model. The two cases of density dependent birth and death can then be “substituted” into the general form. A general form of the Lotka Volterra model is

$$\frac{dR}{dt} = rR - \gamma R^2 - aRN \quad \text{and} \quad \frac{dN}{dt} = caRN - \delta N .$$

a. For the return time of the general form we first solve the non-trivial steady state by setting $dN/dt = 0$ and $dR/dt = 0$, which gives

$$\bar{R} = \frac{\delta}{ca} \quad \text{and} \quad \bar{N} = \frac{r}{a} - \frac{\gamma}{a} \bar{R} = \frac{r}{a} - \frac{\gamma\delta}{ca^2} ,$$

respectively. The Jacobian of the general model is

$$J = \begin{pmatrix} r - 2\bar{\gamma}\bar{R} - a\bar{N} & -a\bar{R} \\ ca\bar{N} & ca\bar{R} - \delta \end{pmatrix} = \begin{pmatrix} -\frac{\gamma\delta}{ca} - \frac{\delta}{c} \\ \frac{\delta}{c} \end{pmatrix} .$$
where \( cr - \gamma \delta / a > 0 \) because \( caN > 0 \). The trace of this matrix is negative, i.e., \( \text{tr} = -\frac{\gamma \delta}{2ca} \), and the eigenvalues of this Jacobian are given by

\[
\lambda_\pm = \frac{\text{tr} \pm \sqrt{\text{tr}^2 - 4 \det}}{2} = -\frac{\gamma \delta}{2ca} \pm \frac{\sqrt{D}}{2},
\]

where \( D = \text{tr}^2 - 4 \det \) is the discriminant of the matrix (and “det” the determinant). Since we are considering a spiral point, the eigenvalues have to be complex, implying that the discriminant \( D < 0 \). The imaginary part of the eigenvalues defines the period of the dampened oscillation, and the real part how fast its amplitude grows or contracts, i.e., the return time depends on the real part only. Thus, for the return time we consider the real part, \( \text{Re}(\lambda) = -\frac{\gamma \delta}{2ca} \), to obtain a return time

\[
T_R = \frac{-1}{\text{Re}(\lambda)} = \frac{2ca}{\gamma \delta} = \frac{2}{\gamma} \frac{1}{\bar{R}}.
\]

Thus, the return time is independent of the net rate of increase, \( r \), depends on the density dependence parameter, \( \gamma \), and is inversely related to the steady state of the resource.

b. We write the model with density dependent birth as

\[
\frac{dR}{dt} = bR(1 - R/k) - dR - aRN = bR - bR^2/k - dR - aRN,
\]

which in the general form means that \( r = (b - d) \) and \( \gamma = b/k \). To obtain the return time of the non-trivial steady state of this model, we only need to substitute \( \gamma = b/k \) into the general expression for the return time, because the return time is independent of \( r \), and because \( \bar{R} \) came from \( dN/dt = 0 \), which has not changed. We obtain that

\[
T_R = \frac{2}{b} \frac{k}{\bar{R}} = \frac{2cak}{b \delta},
\]

where \( k/\bar{R} \) is a ratio of resource densities (i.e., \( k \) is the density at which the birth rate become zero). Note that the dimension is correct: \( k/\bar{R} \) is dimensionless and \( 2/b \) has the dimension time. Thus, the return time of this density dependent birth depends on the birth rate parameters, \( b \) and \( k \), and not on the density independent death rate, \( d \).

c. We write the model with density dependent death as

\[
\frac{dR}{dt} = bR - dR(1 + R/k) - aRN = bR - dR - dR^2/k - aRN,
\]

which in the general form means that \( r = (b - d) \) and \( \gamma = d/k \). Now we substitute \( \gamma = d/k \) into \( T_R \) and obtain that

\[
T_R = \frac{2}{d} \frac{k}{\bar{R}} = \frac{2cak}{d \delta},
\]

where \( k/\bar{R} \) is another ratio of resource densities (i.e., \( k \) is the density at which the death rate doubles). Now the return time depends on the density dependent death rate parameters, \( d \) and \( k \).

d. In both cases the return time is determined by a self-dampening effect of the resource onto itself, i.e., \( \text{Re}(\lambda) = -(\gamma/2)\bar{R} \). Increasing the birth rate, or the death rate, decreases the return time because it speeds up the dynamics around the steady state. Increasing \( k \) increases the return time because it weakens the density dependent regulation. Weakening the consumer, i.e., increasing \( \bar{R} \), decreases the return time because that also increases the self-dampening effect of the resource.
Answers to Chapter 6

Question 6.1. SARS

a. First count the total number of infected patients $I(t)$. $R_0 = 3$ in two weeks means that $\beta = 1.5$ per week. For a time scale of weeks the model therefore is $dI/dt = 1.5I - 0.5I = I$. The equation to solve is $3 \times 10^9 = I(0)e^{rt}$, where $r = (\beta - \delta) = 1$, and where one starts with one infected individual, i.e., $I(0) = 1$. Solving $3 \times 10^9 = e^t$ yields $t = 22$ weeks for the time required to have $I(t) = 3 \times 10^9$.

One could argue that it is more interesting to calculate the time required to have killed half of the population, but this is more difficult. For that one also should keep track of the total number of dead individuals $dD/dt = \delta I$. With $I(t) = e^{(\beta - \delta)t}$ and $D(0) = 0$ the solution of $dD/dt = \delta e^{(\beta - \delta)t} = D(t) = \frac{\delta e^{(\beta - \delta)t-1}}{\beta-\delta}$. Solving $I(t) + D(t) = 3 \times 10^9$ for $\beta = 1.5$ and $\delta = 0.5$ per week gives a total time of $t = 21$ weeks. The difference is small because the number of dead patients approaches a fixed fraction $\frac{\delta}{\beta-\delta} = 0.5$ of the total number of patients that are alive.

b. No, it will go slower because the epidemic will limit itself by depleting the number of susceptibles. Thus it is much better to use an SI model. Because the SARS epidemic is so much faster than the human birth and death rates, Eq. (6.1) would simplify to

$$\frac{dS}{dt} = -\beta IS \quad \text{and} \quad \frac{dI}{dt} = \beta IS - \delta I .$$

You can use the sir.R model to study how rapid SARS would spread in this SI model. Another improvement of the model that would slow down the epidemic is to allow for an incubation period, and use the SEIR model.

Question 6.2. SIR model

Figure made with the model sir.R:

\begin{align*}
a. & \quad R_0 = \frac{\beta}{(\delta + r)} \quad \text{and} \quad r_0 = \beta - \delta - r . \\
b. & \quad \text{Setting} \quad \frac{dI}{dt} = \frac{\beta SI}{S + I} - (\delta + r)I = 0 \quad \text{gives} \quad \frac{\beta S}{\delta + r} = I + S , \\
& \quad \text{or} \quad I = S(R_0 - 1), \quad \text{which is a line through the origin with slope} \quad R_0 - 1. \quad \text{For the other nullcline we set} \\
& \quad \frac{dS}{dt} = s - dS - \frac{\beta SI}{S + I} = 0 \quad \text{giving} \quad (s - dS)(S + I) = \beta SI \quad \text{or} \quad I = \frac{sS - dS^2}{(\beta + d)S + s} .
\end{align*}
which defines a line that is too unpleasant to sketch by hand. Better use the \texttt{sir.R} model (see the Panel (a)).

c. The fact that the \( \frac{dI}{dt} = 0 \) nullcline goes through the origin means that the epidemic can grow when the susceptible population is extremely small (see the upward arrow near the origin). This is an unpleasant consequence of using the fraction of infected individuals in the number of daily encounters: at low population densities the number of individuals encountered should actually go to zero. Thus, this problem should be solved by realizing that the infection term should depend on the expected number, \( n \), of individuals encountered per day, and the fraction, \( f = \frac{I}{S+I} \), of infected individuals among them. This frequency dependent model only deals with the latter by making the rate at which a susceptible individual is infected directly proportional to the fraction, \( f \), of infected individuals. If one were to write that the expected number of individuals encountered per day should be a saturation function of the population density, e.g., \( n = \frac{S+I}{h+S+I} \), and that the infection rate should be proportional the fraction of infected individuals encountered, i.e., \( fn = \frac{I}{h+S+I} \), we obtain from \( \frac{dI}{dt} = \beta SI \frac{S+I}{h+S+I} - (\delta + r)I = 0 \) that the nullcline, \( B = S(R_0 - 1) - h \), is intersecting the horizontal axis at \( h = \frac{h}{R_0-1} \) (see Panel (b)).

\textbf{Question 6.3. Measles}

a. On a logarithmic scale the epidemic first grows linearly and then contracts.

\textbf{b.} \( s[*I*]<-\exp(coef(f)[1]) \) sets \( I(0) \) to \( e^i \) where \( i \) is the intercept.

\textbf{p["beta"]<-1 + coef(f)[2] \} sets \( \beta \) to one plus the slope because \( r_0 = \beta - r = \beta - 1 \).

c. The fit looks reasonable, but not perfect. Starting from different initial guesses similar estimates are obtained. Because starting from different initial guesses similar estimates are obtained the parameter seem identifiable.

d. Parameters have quite large confidence ranges.

e. Since \( N = S + I + R \) is not changing over time, this basically scales the \( \beta \) parameter, and nothing should change. However, the frequency dependent formulation better separates the parameters \( S(0) \) and \( \beta \) from each other, which may facilitate the fitting and hence narrow down the confidence intervals.

\textbf{Answers to Chapter 7}

\textbf{Question 7.1. Michaelis Menten}

a. From the conservation equation one obtains that the concentration of freely available enzyme is given by \( E = E_0 - C \). From the reaction scheme one derives for the complexes \( \frac{dC}{dt} = k_1 ES - (k_{-1} + k_2)C \), which after substituting the conservation equation becomes

\[ \frac{dC}{dt} = k_1(E_0 - C)S - (k_{-1} + k_2)C. \]

For the formation of product one simply writes \( \frac{dP}{dt} = k_2C \).

b. To solve \( \frac{dC}{dt} = 0 \) we first collect all the terms containing \( C \):

\[ \frac{dC}{dt} = k_1 E_0 S - (k_1 S + k_{-1} + k_2)C. \]

Because \( \frac{dC}{dt} = 0 \) we obtain \( k_1 E_0 S = (k_1 S + k_{-1} + k_2)C \), or

\[ C = \frac{k_1 E_0 S}{k_1 S + k_{-1} + k_2} = \frac{E_0 S}{K_m + S} \quad \text{where} \quad K_m = \frac{k_{-1} + k_2}{k_1}. \]

Thus, \( C \) as a function of \( S \) looks like our standard Hill function \( y = \frac{x}{h+x} \).
c. By defining $K_m$ the simplification was already done. This means the the product equation can be written as $\frac{dP}{dt} = \frac{k_2 E_0 S}{K_m + S}$

d. The beautiful trick of adding $\frac{dC}{dt} = 0$ to $\frac{dS}{dt}$ readily simplifies the substrate equation into $\frac{dS}{dt} = -k_2 C$. Filling in the quasi steady state expression for $C$ gives $\frac{dS}{dt} = -\frac{k_2 E_0 S}{K_m + S}$

**Question 7.2. Parameters**

The biological interpretation and dimension of the parameters are:

a. 1. $a_1$: Maximal per capita growth rate (1/t)
   2. $K$: Carrying capacity (numbers or biomass).
   3. $b_1$: Maximal per capita catch rate (1/t).
   4. $c_1$: Population density $R$ where $N$ catches/feeds at its half maximal rate (numbers or biomass).
   5. $a_2$: per capita death rate (1/t).
   6. $b_2$: Maximum per capita birth rate (1/t).
   7. $c_2$: Population $R$ where $N$ grows at half its maximum rate (numbers or biomass).

b. $b_2 = \alpha b_1$ where $\alpha$ is the conversion factor. If population sizes are measured in biomass the normal trophic conversion factor is $\alpha = 0.1$, i.e., typically there is a 90% loss between trophic levels. If the population sizes are measured in numbers this could be anything because small consumers could feed on a large resource.

c. $c_1 = c_2$ means that the growth of the consumer is proportional to what it eats. $c_1 > c_2$ means that the growth rate saturates earlier than the catching rate, which is to be expected when the birth rate of the consumer saturates as a function of its consumption; see Eq. (7.16). $c_1 < c_2$ seems strange and means that the catching rate is saturated earlier than the growth rate.

**Question 7.3. Dampened oscillations**

Figures made with hiv.R:

(a)

(b)

a. The mass-action model is shown in Panel (a) and for large values of the saturation constants the extended model indeed has a very similar behavior (the target cells, $T$, are called “C” in the figure because “T” also means true in R).

b. The behavior of the saturated model for low saturation constants is depicted in Panel (b). Comparing Panel (a) with (b) we see that the oscillations are dampened by the “Beddington” interaction terms.

c. Yes, if both populations are small their encounters should be proportional to the product of their densities, and in this regime the Beddington term approaches the mass-action term. When only one of the populations is large the Beddington term approach a normal saturation function, whereby the process is limited by the smallest population. All of this seems very reasonable.
d. Yes, the trajectories of the Beddington model approach the steady state asymptotically, whereas those of the Lotka-Volterra model approach it by dampened oscillations. The latter steady state is a stable spiral and the former a stable node.

**Question 7.4. Curvature**

Figure made with `hyper.R`:

![Curvature Figure](image)

a. Panel (a) shows that for $H = h/(1 - \gamma/2)$ all curves cross at $R = h$, and that the curvature changes smoothly from a conventional saturation function to a discontinuous Holling type-I function. This therefore seems a very useful functional response when data deviate from the usual saturating functions.

b. Panel (b) and (c) show two cases with a stable limit cycle around a stable steady state. Note that it is not possible for the consumer nullcline to intersect at the right-hand side of the maximum of the resource nullcline.

c. It is somewhat disturbing that a somewhat steeper curvature can completely change the behavior of the model. The devil is apparently in the details, which is unfortunate because we typically do not worry about the curvature and just choose a convenient function.

**Question 7.5. Ratio-dependent predation**

Figures made with `ratio.R`:

![Ratio-dependent Predation Figure](image)

a. This model has the same two regimes as models based upon the Beddington functional response, with a limited-consumer scenario in Panel (a), and a humped consumer nullcline with a stable steady state in Panel (b), and with an unstable steady state in Panel (c). Panel (c) reveals that the behavior of the model is problematic as all trajectories approach the origin, which is an unstable steady state. Like in the question on the SIR model, this is a consequence of the consumer nullcline going through the origin.

b. No, by increasing $K$ in Panel (b) one never finds a Hopf bifurcation.

**Question 7.6. Eutrophication**
Figures made with the previous version of Grind:

(a)

(b)

(a) For the algae, $A$, and zooplankton, $Z$, one writes

$$\frac{dA}{dt} = rA(1 - A/K) - bZA^2 + A^2$$ and $$\frac{dZ}{dt} = cbZA^2 + A^2 - dZ(1 + eZ),$$

where $e$ is the extra death due to intra-specific competition. The nullcline for the algae has been constructed in the text. For the zooplankton one obtains from $dZ/dt = 0$ that $Z = 0$ or

$$cbZA^2 + A^2 - dZ = 0$$ or

$$Z = \frac{cb}{de} \frac{A^2}{h^2 + A^2} - \frac{1}{e},$$

which is a sigmoid function intersecting the vertical axis at $Z = -1/e$ and the horizontal axis at $A = h/\sqrt{R_0 - 1}$, where $R_0 = cb/d$. When $e = 0$ the $Z$-nullcline is a vertical line.

(b) The carrying capacity, $K$, of the algae will depend on the total amount of nutrients that are available for the algae. Studying eutrophication therefore corresponds to increasing $K$.

(c) There are many possibilities, see Panel (a) and (b). The effect of eutrophication corresponds to moving along a sigmoid zooplankton nullcline from the lowest to the highest algae nullcline. Steady states may stabilize or destabilize, and appear or disappear.

(d) Models suggest that changing a single parameter can have various different effects, depending on the precise initial circumstances. It is difficult to generalize, and reliable predictions are nearly impossible to make. A model plays the important role of suggesting various possible outcomes; possibly including undesired outcomes.

**Question 7.7. Luckinbill**

Figures made with previous version of Grind:
a. The oscillatory behavior suggests a Monod saturation
\[
\frac{dP}{dt} = aP(1 - P/K) - \frac{bDP}{h + P} \quad \text{and} \quad \frac{dD}{dt} = \frac{cDP}{h + P} - dD.
\]

b. Increasing the viscosity of the medium decreases the likelihood of meeting prey, which corresponds to increasing the h parameter; see Panel (b). Halving the concentration of food decreases the K parameter; see Panel (c).

c. See Panels (a)–(c).

d. The agreement between model and data seems perfect; a simple Monod saturated functional response provides a good explanation.

e. Formally the populations cannot go extinct in the model; the noise in the data would require stochasticity in the model.

**Question 7.8. Filter feeders**

Figures made with the previous version of Grind:

a. For the phytoplankton, P, and the copepods, C, one could write
\[
\frac{dP}{dt} = aP(1 - P/K) - b_1PC \quad \text{and} \quad \frac{dC}{dt} = b_2 \min(P,L)C - dC,
\]
where L is the phytoplankton level at which the copepods are saturated.

b. Solving \(\frac{dP}{dt} = 0\) one obtains \(P = 0\) or
\[
a - \frac{aP}{K} = b_1C \quad \text{or} \quad C = \frac{a}{b_1} \left(1 - \frac{P}{K}\right),
\]
which is a Lotka Volterra type prey nullcline. Solving \(\frac{dC}{dt} = 0\) gives \(C = 0\) and \(b_2 \min(P,L) - d = 0\). For \(P < L\) this yields \(P = \frac{d}{b_2}\) and for \(P > L\) there is no solution. The case \(P = L\) we do not consider because it is not generic. Thus, the P-nullcline exists when \(\frac{d}{b_2} < L\). See Panel (a). The non trivial steady state is stable because the graphical Jacobian
\[
J = \begin{pmatrix} - & - \\ + & 0 \end{pmatrix} \quad \text{has a} \quad \text{tr}J < 0 \quad \text{and} \quad \text{det} J > 0.
\]
The carrying capacity \(P = K\) is stable when \(\frac{d}{b_2} > L\) because then \(\frac{dC}{dt} < 0\) everywhere.

c. The return time is calculated from a neighborhood stability close to a steady state. The threshold \(L\) has not changed the properties of the steady state, so there is no difference in the return time.

d. When \(P > L\) the vertical arrows in the vector field remain constant. Trajectories in this region therefore move less slowly upward than when \(L \to \infty\). This slows the trajectory down and changes its angle.
e. The mussels would correspond to a normal Holling type I functional response

\[ \frac{dP}{dt} = aP(1 - P/K) - b_1C \min(P,L) \quad \text{and} \quad \frac{dC}{dt} = b_2C \min(P,L) - dC \]

corresponding to the following nullclines

\[ \begin{align*}
C &= \frac{aP}{b_1} \left(1 - \frac{P}{K}\right) \quad \text{and} \quad P = 0 \quad \text{when} \ P < L \\
C &= \frac{aP}{b_1L} \left(1 - \frac{P}{R}\right) \quad \text{otherwise}.
\end{align*} \]

See Panel (b).

**Question 7.9. Exponential functional response**

Figure made with the previous version of Grind:

![Exponential functional response graph](image)

**a.** For \( R \to \infty \) the functional response \( (1 - e^{-\ln(2)R/h}) \to 1 \), which means that at high resource densities the per capita consumer consumption is a resource per unit of time.

**b.** Since one can scale time by the natural rate of increase \( r \), the resource density by its carrying capacity, and the consumer by the \( a \) parameter, the generic form of both models is:

\[ \frac{dR}{dt} = R(1 - R) - \frac{NR}{h + R} \quad \text{and} \quad \frac{dR}{dt} = R(1 - R) - N(1 - e^{-\ln(2)R/h}), \]

which has only one parameter \( h \). Panel (a) shows the nullclines for \( h = 0.1, 0.2, 0.4, 0.8 \) and \( h = 1.6 \). The nullclines intersect when \( R = h \) because the functional response then equals 0.5. There is no qualitative difference between the two sets of nullclines, i.e., we expect similar behavior for these two models.

**Question 7.10. Wolves**

Figure made with the previous version of Grind:

![Wolves graph](image)

There are many different possibilities. For instance, let \( R \) be the prey, and \( W \) be the wolves:
a. One could define $\hat{R} = RW/(c + W)$ as the number of prey that can be caught, i.e., if there are enough wolves ($W \gg c$) all prey can be caught ($\hat{R} \to R$). Taking $\hat{R}$ through a normal Monod saturation gives

$$f(R, W) = \frac{\hat{R}}{h + \hat{R}} = \frac{RW}{hc + hW + RW}$$

$$\frac{dR}{dt} = rR(1 - R/K) - \frac{aRW^2}{hc + hW + RW} \quad \text{and} \quad \frac{dW}{dt} = \frac{aRW^2}{hc + hW + RW} - dW,$$

with $R_0 = a/d$.

b. To sketch the predator nullcline one solves

$$\frac{aRW}{hc + hW + RW} = d \quad \text{or} \quad W = \frac{hc}{R(R_0 - 1) - h},$$

which has a vertical asymptote at $R = h/(R_0 - 1)$ and a horizontal asymptote at $W = 0$. The only intersection with the vertical axis ($R = 0$) is at the negative value $W = -c$. The prey nullcline is not so easy to sketch. We have drawn it with Grind in the picture above, where it looks like a parabola. From the vector field one can see that the carrying capacity is stable. This is an Allee effect because the wolves cannot invade in small numbers. The upper non-trivial steady state is stable when the intersection points is located at the right hand side of the top of the parabola. The lower intersection point is a saddle point, with a separatrix defining the Allee effect.

Alternatively, one could use a mass action predation term and write

$$\frac{dR}{dt} = rR(1 - R/K) - \frac{aRW^2}{c + W} \quad \text{and} \quad \frac{dW}{dt} = \frac{aRW^2}{c + W} - dW.$$

One could even define $f(R, W) = \frac{R}{a(1-cW)+R}$ as a phenomenological functional response that decreases the saturation constant when the number of wolves increases (and use a maximum function to prevent that $1 - cW$ becomes negative).

**Question 7.11. Saturation in consumers**

Figure made with the previous version of Grind:

![Graphs](image)

The prey nullcline is solved from

$$r(1 - R/K) = \frac{aN}{h + N} \quad \text{or} \quad R = K \left(1 - \frac{a/rN}{h + N}\right),$$

which is an inverse Hill function intersecting the vertical $R$-axis at $R = K$. If $a/r < 1$ one obtains a “limited predation” nullcline with an asymptote at $R = K(1 - a/r)$; see Panel (a).
Otherwise the nullcline intersects the horizontal $N$-axis $N = h/(a/r - 1)$; see Panel (b). The consumer nullcline is solved from
\[
\frac{aR}{h + N} = d \quad \text{or} \quad R = (d/a)(h + N),
\]
which is a straight line with slope $d/a$ that intersects the vertical axis at $R = dh/a$. For the non-trivial steady states in both panels we derive the Jacobian
\[
J = \begin{pmatrix}
- & + \\
- & - \\
\end{pmatrix}
\]
giving $\text{tr}J < 0$ and $\text{det}J > 0$, i.e., they are stable.

Answers to Chapter 8

Question 8.1. Food chain
a. For $N = M = 0$ one finds $\bar{R} = s/d$. For $M = 0$ one solves $R = d/b$ from $dN/dt = 0$ and then $\bar{N} = s/d - r/b$. When all three species are present, one solves $\bar{N} = e/c$ from $dM/dt = 0$, then $\bar{R} = s + be/c$ from $dR/dt = 0$, and finally $\bar{M} = (b\bar{R} - d)/c$ from $dN/dt = 0$.

b. Yes, the steady state of $R$ only depends on its source when the length of the chain is odd.

e. \[J = \begin{pmatrix}
(p + d) & 0 & 0 & 0 & \ldots & 0 \\
2p & -(p + d) & 0 & 0 & \ldots & 0 \\
0 & 2p & -(p + d) & 0 & \ldots & 0 \\
& \vdots & & & & \\
0 & \ldots & 0 & 0 & 2p & d
\end{pmatrix}, \tag{A.8.3}
\]
with a characteristic equation corresponding to Eq. (8.11).

Question 8.2. Triangular Jacobian
Since $dN_0/dt$ only depends $N_0$, and $dN_i/dt$ only depends on $N_{i-1}$ and $N_i$, the Jacobi matrix is of the triangular form
\[
J = \begin{pmatrix}
-(p + d) & 0 & 0 & 0 & \ldots & 0 \\
2p & -(p + d) & 0 & 0 & \ldots & 0 \\
0 & 2p & -(p + d) & 0 & \ldots & 0 \\
& \vdots & & & & \\
0 & \ldots & 0 & 0 & 2p & d
\end{pmatrix}, \tag{A.8.3}
\]

Question 8.3. Accumulating mutations
a. Mathematically this would seem appropriate, and it is similar to Eq. (8.9).

b. The problem with a cascade like this is that the variables described by ODEs are continuous, whereas actual cell numbers cannot become lower than a single cell. Solving this cascade, either mathematically or numerically, would immediately populate all the $N_i$ equations, and hence deliver very small densities into the equations for the senescent and leukemic cells at very early time points. For the senescent cells this is not a problem because they die and disappear, but since the leukemic cells have a growth rate could be much faster than the division rate of the progenitor cells, they will start to expand much earlier than expected.

c. Note that the vector in the leukemia.R document is indexed from 1 to \text{ndiv} (and not from 0 to \text{n}), and that R allows one to write all the ODEs for $dN_i/dt$ as a single (fast) vector operation. The leukemic cells appear way too early in this model.

d. This model violates the constraint that size of a population of cells should be described by an integer number. When populations are large this is typically not a problem, but small populations should be described by stochastic models describing the behavior of individual cells. This problem is also known as the atto-fox problem. When $pN_i < 1$ one should define...
this term as the probability that a single cell divides and delivers exactly two daughter cells in the next generation. The formal procedure to do this is called a Gillespie simulation \[6\] in which every term of the model is translated into an event happening with a probability depending on the current population densities. Stochastically executing individual events on the basis of these probabilities, the population densities will only change by a single cell at a time. The ODEs basically describe the average that is expected from running a large number of Gillespie simulations. If you like this we could turn this model into a project.

e. No the Smith-Martin model would only delay the formation of the leukemic cells by \( n \times \Delta \) days, i.e., by the total time spend in the B-phase division, which short even if cells divide once per year.

Question 8.4. Chaos

Figures made with the previous version of Grind:

(a) \( N \)

(b) \( N \)

a. See Panel (a). Yes, for their values of \( b_1 \) the steady state is unstable.
b. See Panel (b). Yes, the unstable steady state around which the trajectory cycles is located above the top-consumer nullcline, and we expect the average consumer density to be higher than the top-consumer nullcline.
c. Use Grind for the last 3 items.

Question 8.5. Detritus

Figure made with the previous version of Grind:

(a) \( N \)

(b) \( N \)

A simple model would be

\[
\frac{dR}{dt} = [bF - d_R - c_1 N]R, \quad \frac{dN}{dt} = [c_1 R - d_N - c_2 M]N \quad \text{and} \quad \frac{dM}{dt} = [c_2 N - d_M]M,
\]

where \( F = K - R - N - M \). This shows that the \( dN/dt \) and \( dM/dt \) equations do not change. The 3-dimensional nullclines of the consumer, \( N \), and the top-consumer \( M \) therefore stay the
same. That of the resource is solved from \([b(K - R - N - M) - d_R - c_1 N] = 0\), which gives a negative linear relation in each of the sides of the 3-dimensional phase space. The plane will look like a triangle (Panel a) that moves vertically when \(K\) is changed (Panel b).

For \(N = M = 0\) one now obtains \(\bar{R} = K - d_R/b\), which increases linearly with the total amount of nutrients, \(K\), in the system. When \(N > 0\) and \(M = 0\), one solves \(R = d_N/c_1\) from \(dN/dt = 0\), and from \([b(K - \bar{R} - N) - d_R - c_1 N] = 0\) one solves that
\[
\bar{N} = \frac{c_1 b K - b d_N - c_1 d_R}{c_1(b + c_1)}
\]
which increases linearly with \(K\), and becomes positive when \(K > (b d_N - c_1 d_R)/(c_1 b)\). When \(N > 0\) and \(M > 0\) one again solves \(\bar{N} = d_M/c_2\) from \(dM/dt = 0\), \(\bar{T}\) from \(dT/dt = 0\), and \(\bar{R}\) from \(dR/dt = 0\). The steady state resource density again only depends on \(K\) when the food chain has an odd length.

**Answers to Chapter 9**

**Question 9.1. Migration**

Figure made with the previous version of Grind:

(a)  
(b)  
(c)  

\[ a. \] The model would become
\[
\frac{dN_1}{dt} = i + r_1 N_1(1 - N_1 - \gamma_1 N_2) \quad \text{and} \quad \frac{dN_2}{dt} = i + r_2 N_2(1 - N_2 - \gamma_2 N_1)
\]

\[ b. \] Given that \(i \ll 1\) one obtains the nullclines in Panels (a)–(c).

\[ c. \] From the vector field one can see that the steady states close to the carrying capacity are stable. The steady state in the middle of Panel (a) is stable, whereas that in the middle of Panel (c) is unstable.

\[ d. \] In Panel (a) there is normal coexistence. In the other Panels there is no true competitive exclusion. However, at the steady state near the carrying capacity the density of the rarest species is very low.

**Question 9.2. Co-existence by trade-offs?**

\[ a. \] No this is not an appropriate model for substitutable resources because the birth rate increases with every non-essential resource that is added to the ecosystem. Consumers are expected to approach their maximal birth rate at sufficiently high densities of just one resource if these are non-essential.
b. One could argue that this would become a model for essential resources when the birth rates, $\beta_{ij}$, on the individual resources are made smaller than the death rates, $\delta_i$. Consuming a combination of resources then becomes essential, but this interpretation remains somewhat contrived.

c. One can define a trade-off by adding terms like $c_{12} \leftarrow c - c_{11}$; $c_{21} \leftarrow c - c_{22}$; $c_{31} \leftarrow c - c_{32}$ to the model, which defines a total consumption rate, $c$, that is the same for all consumers, and play with the other consumption rates. For substitutable resources defined by Eq. (9.21) (in the file additive.R), one indeed finds that the three consumer nullclines intersect in one steady state in a Tilman diagram spanned up by two resources, but this requires that all other parameters like the saturation constants and the death rates are also the same. For essential resources defined by Eq. (9.23) (in the file essential.R), defining this trade-off is not sufficient to let the three consumer nullclines intersect in one steady state.

d. The result seems rather artificial: it is not based upon an appropriate model, and requires unreasonable parameter constraints. This would be a good project to study further.

Question 9.3. Non-equilibrium co-existence
Figure made with the previous version of Grind:

\[ f(R) \]

\[ R \]

\[ N_2 \]

\[ N_1 \]

\[ R \]

(a)  (b)

\[ \frac{dL}{dt} = rA - dL(1 + eL) - mL \quad \text{and} \quad \frac{dA}{dt} = mL - \delta A, \]

where we assume density dependent death by competition between the larvae. The steady
state can be solved by first setting \( \frac{dA}{dt} = 0 \) delivering \( A = mL/\delta \). Substituting this into \( \frac{dL}{dt} = 0 \) gives

\[
L = \frac{1}{e} \left[ \frac{m}{d} \left( \frac{r}{\delta} - 1 \right) - 1 \right], \quad \bar{A} = \frac{m}{\delta} L,
\]

which requires \( \alpha = r/\delta > 1 \) and \( m(\alpha - 1)/d > 1 \).

b. Adding two predators changes the model into

\[
\frac{dL}{dt} = rA - dL(1 + eL) - mL - c_1LN_1, \quad \frac{dA}{dt} = mL - \delta A - c_2AN_2,
\]

\[
\frac{dN_1}{dt} = (c_1L - d_1)N_1 \quad \text{and} \quad \frac{dN_2}{dt} = (c_2A - d_2)N_2.
\]

Solving the steady state of the latter two gives \( \bar{L} = d_1/c_1 \) and \( \bar{A} = d_2/c_2 \). Substituting this into \( \frac{dL}{dt} = 0 \) and \( \frac{dA}{dt} = 0 \) gives

\[
\bar{N}_1 = \frac{rd_2}{c_2d_1} - \frac{m}{c_1} \frac{1 + ed_1}{c_1} \quad \text{and} \quad \bar{N}_2 = \frac{Md_1}{c_1d_2} - \frac{\delta}{c_2}.
\]

Since one can always choose parameters such that \( \bar{N}_1 > 0 \) and \( \bar{N}_2 > 0 \) co-existence is possible.

**Question 9.5. Gradients**

Figure made with the previous version of Grind:

(a) In gradient 1 there is a sharp transition, as if there is a abrupt change in the environmental conditions in sample seven. In gradient 2 it seems that environmental conditions change more gradually.

(b) Turn the nullclines in the direction of the arrows, while keeping the carrying capacities the same. See Panel (a)-(f).

(c) Both environmental gradients can be gradual. If this gradient affects the inter-specific competition the gradual change in the environment can give rise to the sharp cline in gradient 1 or to the continuous transition in gradient 2.
d. The samples have some variation that could be due to noise.

**Question 9.6. Density dependent birth rate**

Figure made with the previous version of Grind:

![Diagram](image)

**a.** $R_0 = b/d$ or $R_0 = \frac{b}{\frac{h}{a} + a}$, depending on its definition.

**b.** The QSS of the resource is $R = 1 - aN$ which when substituted into

$$\frac{aR}{H + aR} \quad \text{gives} \quad \frac{1 - aN}{H - aN}$$

where $H = 1 + h/a$, which is larger than one. The new consumer equation becomes

$$\frac{dN}{dt} = b \frac{1 - aN}{H - aN} - d \quad N.$$ 

c. The maximum birth rate is $b/H = \frac{ab}{a+h}$. Hence $R_0 = \frac{b}{\frac{h}{a} + a}$, which is the same as the second answer in a.

d. To sketch the per capita birth rate as a function of $N$ we first consider the function $y = \frac{1-aN}{H-aN}$ knowing that $H > 1$. For $N = 0$ this delivers $y = 1/H$, and for $y = 0$ we find $N = 1/a$. A horizontal asymptote is found by dividing numerator and denominator by $N$, i.e., $y = \frac{1/N-a}{H/N-a}$, and letting $N \to \infty$ to find that $y \to 1$. A vertical asymptote is located at $N = H/a$. Because $H > 1$ we know that the intersections with the horizontal and vertical axis fall below the asymptotes. See Panel (a). Finally we multiply (scale) the whole function with the birth rate $b$ to obtain the sketch in Panel (b).

e. This concave shape is what we considered most realistic in Chapter 3. For instance see Fig. 3.5c.

**f.** The QSS now equals $R = 1/(1 + aN)$ which gives a per capita birth rate of $\frac{b}{1 + \frac{h}{a} + hN}$ which is convex. Again the devil is in the details, as the shape of the consumers density dependence depends on the nature of the resource.

**Question 9.7. Tilman’s competition model**

Figures made with the previous version of Grind:
We can study the difference between the two parameter settings by studying the Jacobian of $N$. The red $dN_1/dt = 0$ nullcline has a vertical part at $R_1 = b_{11}d_1/b_{11}c_{11}$ where the first resource is limiting, and a horizontal part at $R_2 = b_{22}d_2/b_{22}c_{22}$ where the second resource is limiting. Above and on the right hand side of this nullcline $dN_1/dt > 0$ (which is “arbitrarily” indicated by horizontal arrows). The blue $dN_2/dt = 0$ nullcline as a horizontal part located at $R_2 = h_{12}d_2/h_{22}c_{22}$, and a vertical part located at $R_1 = h_{12}d_1/h_{11}c_{11}$. The upwards arrows indicate the region where $dN_2/dt > 0$. The derivatives of the resources are not (yet) defined because this diagram is based upon Eq. (9.32) only. Both resources are essential because the horizontal and vertical parts of these nullclines define the minimum amounts the species require for growth. Two qualitatively different examples with intersecting nullclines are given in Panels (a) and (b). When these nullclines fail to intersect there is no resource density ($R_1, R_2$) where $dN_1/dt = dN_2/dt = 0$.

The nullclines in Panels (c) and (d) correspond to Tilman diagrams of (a) and (b), respectively. All Panels were made by assuming that $N_1$ consumes more of resource one, whereas $N_2$ specializes on $R_2$, i.e., $c_{11} = c_{22} = 0.5$, and $c_{12} = c_{21} = 0.25$. All Panels have the same birth and death rates ($b_1 = b_2 = 0.5$, $d_1 = d_2 = 0.1$), and we have located the intersection point at the same resource densities by setting

$$\frac{h_{11}d_1}{b_{11}c_{11}} = \frac{h_{21}d_2}{b_{21}c_{21}} = \frac{h_{22}d_2}{b_{22}c_{22}} = \frac{h_{12}d_1}{b_{12}c_{12}} = 0.4,$$

i.e., $h_{11} = h_{22} = 1$, and $h_{21} = h_{12} = 0.1$ in Panel (a) and $h_{11} = h_{22} = h_{21} = h_{12} = 0.5$ in Panel (b). The steady state is then located at $\bar{R}_1 = \bar{R}_2 = 0.4$ and $\bar{N}_1 = \bar{N}_2 = 2$ (this can be studied with the file tilman.r). In Panels (a & c) each species therefore consumes most of the resource it requires most. In Panels (b & d) they require the same amount of each resource, but they consume them at different rates. The former leads to stable co-existence (Panel c), the latter to a “founder controlled” phase plane with an unstable steady state (Panel d).

c. We can study the difference between the two parameter settings by studying the Jacobian of the 4-dimensional system. Since $N_1$ consumes more of $R_1$ and $N_2$ more of $R_2$ we obtain that $\partial R^t_1/\partial N_1 = -c_{11}\bar{R}_1 = -0.5 \times 0.4 = -0.2$ and that $\partial R^t_1/\partial N_2 = -c_{21}\bar{R}_1 = -0.25 \times 0.4 = -0.1$. For resource two this is just the other way around. The local effect of a specialized consumer
on its resource is thus 2-fold larger than that of the other consumer. This is the same in both parameter settings. The effect of the resources on the consumers can be read by combing the graphical Jacobian with the full Jacobian. In Panel (a) where the steady state is located at the vertical part of the \( \frac{dN_1}{dt} = 0 \) nullcline and hence \( R_1 \) is limiting, a small increase of \( R_1 \) will increase \( \frac{dN_1}{dt} \), i.e., \( \frac{\partial N'_1}{\partial R_1} = (b_1c_{11}/h_{11})\bar{N}_1 = (0.5 \times 0.5/1)\bar{N}_1 = 0.25 \times 2 = 0.5 \), whereas \( \frac{\partial N'_1}{\partial R_2} = 0 \) because \( R_2 \) is not limiting (and we stay on the \( \frac{dN_1}{dt} = 0 \) nullcline if \( R_2 \) is increased). For the second consumer this is just the other way around. Conversely, in Panel (b) the steady state is located at the horizontal part of the \( \frac{dN_1}{dt} = 0 \) nullcline and hence \( R_2 \) is limiting, a small increase of \( R_1 \) will not affect \( \frac{dN_1}{dt} \), i.e., \( \frac{\partial N'_1}{\partial R_1} = 0 \) whereas \( \frac{\partial N'_1}{\partial R_2} = (b_1c_{12}/h_{12})\bar{N}_1 = (0.5 \times 0.25/0.5)\bar{N}_1 = 0.25 \times 2 = 0.5 \). For the second consumer this is just the other way around. Thus, in Panels (a & c) the species that consumes most of \( R_1 \) is also limited by \( R_1 \), whereas in Panels (b & d) the species that consumes most of \( R_1 \) is limited by \( R_2 \). The former is a stable situation and the latter is not (see Section 9.5 and [10, 11]).

**Question 9.8. Fitness**

**a.** Writing out Eq. (9.12) explicitly, and combining parameters

\[
R^*_i = \frac{h_i/c_i}{b_i/d_i - 1} = \frac{H_i}{r_i - 1},
\]

where \( H_i = h_i/c_i \) and \( r_i = b_i/d_i \), we have a simple expression for which species wins (i.e., the one with the lowest \( R^*_i \)). Writing

\[
\hat{R}_0 = \frac{b_i}{d_i} \frac{c_i}{h_i + \hat{R}} = r_i \frac{\hat{R}}{H_i + \hat{R}} = r_i \frac{1}{H_i/\hat{R} + 1},
\]

we can solve for \( r_i \) and write Eq. (A.9.4) in terms of \( \hat{R}_0 \):

\[
R^*_i = \frac{H_i}{\hat{R}_0(H_i/\hat{R} + 1) - 1} \quad \text{where} \quad \hat{R} = \frac{s}{d}.
\]

The species with the lowest fitness \( \hat{R}_0 \) can therefore be the superior competitor when its \( H_i \) is sufficiently smaller than that of the other competitors. In conclusion, \( \hat{R}_0 \) does not uniquely identify the superior competitor, and the critical resource density, \( R^* \), remains the best indicator.

**b.** The model competition.R provides an example where an \( r \)-selected species, with the lowest \( R_0 \) and carrying capacity, outcompetes a \( K \)-selected species.

**Answers to Chapter 10**

**Question 10.1. Invasion criterion**

Figures made with the previous version of Grind:
a. See Panel (a)
b. Since $N_1$ and $N_3$ do not compete the model simplifies to

$$\frac{dN_1}{dt} = rN_1(1-N_1-\alpha N_2) \quad \frac{dN_2}{dt} = rN_2(1-N_2-\alpha N_1-\alpha N_3) \quad \text{and} \quad \frac{dN_3}{dt} = rN_3(1-N_3-\alpha N_2)$$

c. Because $N_2 \approx 0$ the steady states before invasion is $\bar{N}_1 = \bar{N}_3 = 1$ and $dN_2/dt \simeq r N_2(1-2\alpha)$. For invasion one requires $dN_2/dt > 0$ or $1-2\alpha > 0$ giving that $\alpha < 1/2$.

d. Since $N_2$ has an overlap of one with itself the total overlap with the other species should be less than the overlap with itself.

e. For $N_2 = 0$ the nullclines of $N_1$ and $N_3$ are perpendicular lines at $N_1 = 1$ and $N_3 = 1$, respectively. The $N_2$ nullcline intersects the $N_1$ and the $N_3$ axis at $1/\alpha$. At the critical invasion point the $dN_2/dt = 0$ nullcline should go exactly through the point $N_2 = 0$ and $N_1 = N_3 = 1$. See Panel (b) When $N_2$ can invade the $dN_2/dt = 0$ nullcline will intersect at larger $N_1 = N_3$ values, and there will be a stable 3-dimensional steady state.

Question 10.2. Control by parasites

a. Define $T = S + I$ as the total population size of susceptible and infected birds, and write

$$\frac{dS}{dt} = bT(1-T) - dS - \beta SI \quad \text{and} \quad \frac{dI}{dt} = \beta SI - \delta I$$

b. The $R_0$ of the birds is $b/d$ and the carrying capacity is $K = 1 - 1/R_0$.

c. The $R_0$ of the parasites is $R'_0 = \beta K/\delta$.

d. $S = \delta/\beta = K/R'_0$.

e. Defining $O$ as the other species one could write $dS/dt = bT(1-T-O) - dS - \beta SI, dI/dt = \beta SI - \delta I$ and $dO/dt = bO(1-T-O) - d_0 O$, with $d_0 > d$. Whenever $b(1-T)/d_0 > 1$ the other species can invade.

f. Thus, if the infection is sufficiently harmful, i.e., $\bar{T} \ll K$, the other species can invade despite its lower fitness.

g. If each species is sufficiently down-regulated by its parasite the resource density can stay high and many species can be maintained.

Question 10.3. Monopolization

a. Yes, since most competition situations are “founder controlled”, species that grow faster are more likely to outcompete the species that grow slower.

b. No, one would still have that species will survive in a few patches just because they arrived there earlier, or in greater numbers, than other species.
Question 10.4. Symbiosis

Figures made with the previous version of Grind:

(a) A simple model makes the birth rate a saturation function of the other species and assumes density dependent death:

\[
\frac{dN_1}{dt} = N_1 \left[ \frac{b_1 N_2}{h + N_2} - d_1(1 + e_1 N_1) \right] \quad \text{and} \quad \frac{dN_2}{dt} = N_2 \left[ \frac{b_2 N_1}{h + N_1} - d_2(1 + e_2 N_2) \right].
\]

The \( \frac{dN_2}{dt} = 0 \) nullcline is given by

\[
N_2 = \frac{1}{e_2} \left[ \frac{R_0 - \frac{N_1}{h + N_1}}{1} \right],
\]

where \( R_0 = b_2/d_2 \). This is a saturation function starting at \( N_2 = -1/e_2 \) when \( N_1 = 0 \). See Panel (a).

(b) Let \( N_1 \) be the saprophyte:

\[
\frac{dN_1}{dt} = N_1 \left[ \frac{b_1 N_2}{h + N_2} - d_1(1 + e_1 N_1) \right] \quad \text{and} \quad \frac{dN_2}{dt} = N_2 \left[ b_2 - d_2(1 + e_2 N_2) \right].
\]

(c) The other species could merely increase the birth rate:

\[
\frac{dN_1}{dt} = N_1 \left[ b_1 + \beta_1 N_2 \frac{b_1}{h + N_2} - d_1(1 + e_1 N_1) \right] \quad \text{and} \quad \frac{dN_2}{dt} = N_2 \left[ b_2 + \beta_2 N_1 \frac{b_2}{h + N_1} - d_2(1 + e_2 N_2) \right],
\]

where \( \beta_i \) is the maximum birth rate due to the presence of the symbiont, and \( b_i \) is the maximum birth rate in the absence of the symbiont.

(d) Yes, just make sure that \( R_{0i} = b_i/d_i < 1 \) in the absence of the other species, and \( (b_i + \beta_i)/d_i > 1 \) to enable growth in the presence of the symbiont. Panel (c) depicts the typical phase space when \( R_{0i} > 1 \).

Question 10.5. Infinite Niche-matrix

(a) The partial derivatives of the off-diagonal elements

\[
\partial_{N_j} N_i - \sum_j A_{ij} N_i N_j \quad \text{are} \quad 0, 0, \ldots, -A_{ij} N_i, 0, \ldots.
\]

Because all populations have the same steady state, \( \bar{N} \), they become \( -\alpha \bar{N}, -\alpha^4 \bar{N}, -\alpha^9 \bar{N} \). The partial derivatives on the diagonal

\[
\partial_{N_i} N_i - \sum_j A_{ij} N_i N_j \quad \text{are} \quad 1 - 2 \bar{N} - \sum_{j \neq i} A_{ij} \bar{N}
\].
and hence the Jacobian is:

\[
J = \begin{pmatrix}
... & -\alpha \bar{N} & 1 - 2\bar{N} - \sum_{j \neq i} A_{ij} \bar{N} & -\alpha \bar{N} & -\alpha^4 \bar{N} & -\alpha^9 \bar{N} & ... \\
... & -\alpha^4 \bar{N} & -\alpha \bar{N} & 1 - 2\bar{N} - \sum_{j \neq i} A_{ij} \bar{N} & -\alpha \bar{N} & -\alpha^4 \bar{N} & ... \\
... & ... & ... & ... & ... & ... & ...
\end{pmatrix}
\]

Moving one of the $2\bar{N}$ on the diagonal into the sum we obtain

\[
J = \begin{pmatrix}
... & -\alpha \bar{N} & 1 - \bar{N} - \sum A_{ij} \bar{N} & -\alpha \bar{N} & -\alpha^4 \bar{N} & -\alpha^9 \bar{N} & ... \\
... & -\alpha^4 \bar{N} & -\alpha \bar{N} & 1 - \bar{N} - \sum A_{ij} \bar{N} & -\alpha \bar{N} & -\alpha^4 \bar{N} & ... \\
... & ... & ... & ... & ... & ... & ...
\end{pmatrix}
\]

Finally because $\bar{N} = 1/\sum A_{ij}$ all diagonal elements can be simplified as $-\bar{N}$, i.e.,

\[
J = \begin{pmatrix}
... & -\alpha \bar{N} & -\bar{N} & -\alpha \bar{N} & -\alpha^4 \bar{N} & -\alpha^9 \bar{N} & ... \\
... & -\alpha^4 \bar{N} & -\alpha \bar{N} & -\bar{N} & -\alpha \bar{N} & -\alpha^4 \bar{N} & ... \\
... & ... & ... & ... & ... & ... & ...
\end{pmatrix}
\]

b. The Jacobian is equal to $-\bar{N}A$, where $A$ is the interaction matrix. The signs of the eigenvalues of the Jacobian are equal to those of the interaction matrix.

**Question 10.6. Random Jacobian**

a. At low connectivities the characteristic equation will tend to be defined by the trace, i.e., $\lambda = -1$.

b. For large $n$ Eq. \ref{10.11} holds, for small $n$ the dominant eigenvalue tends to be $\lambda = -1$.

c. Change the assignments in the for loops and see what happens.

d. Change the definition of the diagonal elements by diag() into a for loop setting a fraction of them to $-1$.

**Answers to Chapter 11**

**Question 11.1. Biomanipulation**

Figures made with the previous version of Grind:

\[\text{(a)}\]

\[\text{(b)}\]

a. For $F = 0.15$, $h = 1$, $k = 10$, $m = 0.4$ and $p = 0.5$ the phase space is given by Panel (a), which has three non-trivial steady states. By decreasing the carrying capacity the upper two states disappear.

b. Changing the carrying capacity $k$ yields the bifurcation diagram of Panel (b). The heavy solid line depicts stable steady states, the light solid line unstable steady states, and the dots are $Z$ values where the limit cycle crosses a Poincaré plane located at an average $A$ value.
c. There is a transcritical bifurcation at $k = 4$, a saddle-node bifurcation at $k \approx 9$, a Hopf bifurcation at $k \approx 11.5$, and another saddle-node bifurcation at $k \approx 19.5$. The stable limit cycle that is born at the Hopf bifurcation dies by a so-called “global bifurcation” around $k = 12$ when it glues with the stable manifold of the saddle point in the middle.

Answers to Chapter 12

**Question 12.1. Paradox of enrichment**

a. Scale the density of the algae at which the birth rate vanishes to $k = 2$ and scale time by their expected life span such that $d_1 = 1$ (which implies a time scale of about one week). We could give the algae a maximum rate of increase of $b - d_1 = 1$ by setting $b = 2$ per week. Because the carrying capacity $K = k(1 - 1/R_0)$ (see Table 3.1) we obtain that $K = 1$. Because the saturation of the functional response probably occurs at prey densities below the carrying capacity, it seems wise to set $h \ll K$, e.g., $h = 0.1$. We could scale the predator biomass such that the trophic conversion factor becomes $c = 1$, and let us give the predators a 2-fold longer life span, i.e., $d_2 = 0.5$. To give the predator an $R_0 = ce/d_2 = e/0.5 > 1$ we could set $e = 0.6$ such that the initial growth rate of the predator at high prey densities is about 0.1, i.e., 10-fold slower than the algae. For these values the predator nullcline is located at $h/(R_0 - 1) = 0.1/(0.6/0.5 - 1) = 0.5$, which is just at the right hand side of the maximum of the prey nullcline at $(K - h)/2 = 0.45$.

b. Different possibilities for the location of the predator nullcline, without changing that of the prey, can be made by changing the death rate of the predator.

c. The carrying capacity can be changed by altering the density $k$ at which the birth rate of the algae vanishes.

d. First settle into a non-trivial steady state by giving proper initial values and then issuing the `f<-newton()` command. Then call `continue(f, x="k", xmin=0.1, xmax=5, y="N")` to define a horizontal axis (where we avoid $k = 0$ because the model is dividing by $k$), and we keep the predator on the vertical axis.

e. Replace the death rate of the predators by $d_2(1 + \epsilon N)$.

f. This indeed delivers a phase plane resembling that of consumer-resource model with a sigmoid functional response.

**Question 12.2. Fishing herring**

The first thing to think about is the parameters of the model. For instance, one could consider the Herring population in the North sea, and realize that the population will have a carrying capacity amounting to an enormous number of individuals, or an enormous amount of biomass. Fortunately, one can always scale the population density in a model by the carrying capacity of the population. Thus, we can set the carrying capacity, $k = 1$, realizing that $H = 1$ actually corresponds to the Herring population at carrying capacity in the North sea. The next parameter is the natural rate of increase, $r$. We first need to define a time-scale, and for a Herring population with a yearly reproduction cycle, a time-scale of years seems a proper choice. If $t$ is measured in years we can think of a growth rate per year, and using our biological intuition about fish or the size of Herring, it seems obvious that a growth rate of 1% per year seems slow and that they will not easily grow faster than 100% per year. Thus, setting $r = 0.1$ per year or $r = 0.2$ per year seem reasonable choices. One can actually check this by studying the recovery rate of a crashed Herring population in the absence of fishing: setting $H = 0.01$ and $Q = 0$, and run the model for a few decades to test how long it takes for the population to recover and approach its carrying capacity. Once you think you have found realistic parameters, you can start on the rest of the exercise.
a. Starting at the carrying capacity, and setting \( Q = rk/4 \) to study the impact of this maximum yearly harvest, one finds that the population approaches \( H = k/2 \) in the absence of noise. However, the population will always go extinct if there is enough noise.

b. Now the population will not go extinct.

c. At the steady state \( dH/dt = rH(1 - H/k) - fH = 0 \), or \( \bar{H} = k(1 - f/r) \), the total harvest is \( f \bar{H} \). Taking the derivative, \( \partial_f \), of \( f \bar{H} \), and setting that to zero gives \( k - (2k/r)f = 0 \) or \( f = r/2 \). Substituting that into \( \bar{H} \) gives \( \bar{H} = k/2 \), i.e., half of the carrying capacity.

d. The population will no longer go extinct. Even noise on the “optimal” \( f \) will not drive the population to extinction.

e. The optimal harvest \( f \bar{H} \) at \( f = r/2 \) is \( rk/4 \), which is equal to \( Q \). Thus, catching a fraction of the Herring population on average allows for the same maximum harvest, but is much more robust. Note that a shortcut to the same result is to see that this optimum is reached when the harvest function, \( fH \), crosses the growth function, \( rH(1 - H/k) \), in its maximum \( rk/4 \) at \( H = k/2 \).

Question 12.3. Fitting the Gause data from 1934

a. Yes, the fit looks reasonable and starting with the estimates of Gause [5] we obtain very similar estimates for the two growth rates and carrying capacities.

b. For Paramecium aurelia we obtain \( \alpha \simeq 1.05 \) and for \( P. caudatum \) we obtain \( \beta \simeq 0.64 \). However, this does not mean that \( P. aurelia \) suffers more from \( P. caudatum \) than the other way around because some parameters remain to be divided by the—quite different—carrying capacities. This can easily be checked by calling \( \texttt{plane}(xmax=110,ymax=110,eps=-0.01) \) for the estimated parameters, revealing that the nullclines fail to intersect, and that \( P. aurelia \) is the strongest competitor.

c. Since \( P. aurelia \) suffers more from \( P. caudatum \) than from itself, it could be that the species are competing for more than one essential resource, and that \( P. caudatum \) consumes more than \( P. aurelia \). However, also note that \( \alpha \simeq 1 \) and that we could be over-interpreting the fact that \( \alpha > 1 \) (see below).

d. Fortunately we find similar results, but this is at least partly due to the fact that by going step wise, and by using Gause’s estimates, we have such a good initial guess. Try other initial guesses to test how much this depends on the guess.

e. Yes, given a good initial guess the confidence intervals suggest that all parameters are identifiable. The confidence intervals for \( \alpha \) and \( \beta \) do overlap, and hence we cannot conclude that \( \alpha > \beta \). Additionally the confidence interval for \( \alpha \) includes \( \alpha = 1 \), so we have indeed over-interpreted the estimate that \( \alpha > 1 \).

f. The more mechanistic model explains the data at least equally good, with similar growth rates, and it may suggest that \( P. caudatum \) consumers more of the resource. However, when fitting the data where both species are competing we find unexpected estimates for the death rate and consumption of \( P. aurelia \). Apparently, there is too much freedom here, meaning that not all parameters are identifiable.

Question 12.4. Cryptic oscillations

a. Since bacteria readily evolve resistance to bacteriophages the stable \( E. coli \) population is most likely resistant to T4. If the resistant bacteria continue to revert to sensitive bacteria, one could postulate that a small subpopulation of sensitive \( E. coli \) maintains the predator-prey oscillations with the T4 phage.

b. This would mean that resistance evolves after about 200h of co-culture.

c. If you fail check Figure 4.6 in the book of Weitz [12] for an example.

Question 12.5. Lymphocyte migration

a. Because the total number of cells is not changing the number of cells in the blood can be
described with a conservation equation. The ODE would have been \( dB/dt = \epsilon_S S + \epsilon_L L - (i_S + ni_L)B \), and replacing the conservation equation with this ODE gives exactly the same model. Numerically, the version with the conservation equation is more stable because small numerical errors could make \( dB/dt + dS/dt + dL/dt \neq 0 \).

b. The steady state is \( \bar{S} \approx 22, \bar{D} \approx 1.9 \), and \( \bar{L} \approx 72.4 \) cells, and hence there will be \( B \approx 3.7 \) cells in the blood. Every lymph node is expected to contain \( 72.4/38 = 1.9 \) cells, which is also revealed by \( \bar{D} \approx 1.9 \).

c. The only term missing in the denominator is the \( \epsilon_L \epsilon_S \) term, and hence \( \bar{B} = \frac{\epsilon_L \epsilon_S}{\epsilon_L \epsilon_S + \epsilon_L i_S + \epsilon_S n i_L} \)
The recurrent pattern in the expression is that \( \bar{S} \) and \( \bar{L} \) increase with their own influx times the efflux of the other compartment. It makes sense that increasing the rate of efflux from the lymph nodes increases the number of cells in the spleen (and similarly in the blood).

d. Running the model for several days reveals that one needs 20 days of capturing cells to exceed \( D(t) = 50 \). Waiting for almost three weeks to recruit just 50% of the cognate naive T cells would be dangerously long.

e. Adding on a \( f_i = 9 \) fold increase in the influx to the draining lymph nodes reveals that it would take about 2.5 days to accumulate 50% of the cells. Note that this still requires that cognate cells do not egress from the draining lymph node: otherwise a new steady state is established where most of the cells reside in the other lymph nodes (because \( f_i < n - 1 \)).

f. To model infection with a gradual angiogenesis, one could replace the \( f_i L \) term by \( \frac{1}{\bar{D} t} \left( f_i - 1 \right) i_L \bar{B} + i_L \bar{B} \) to define that at \( t = 0 \) the influx is \( i_L \bar{B} \), at \( t = h \) the influx is \( \left( \frac{h - 1}{2} \right) i_L \bar{B} + i_L \bar{B} \), and that when \( t \to \infty \) the influx approaches the previous \( f_i i_L \bar{B} \).

**Question 12.6. Improving HIV therapy?**

a. To check the growth rate one could run the model starting from \( s \leftarrow c(T=1, I1=0, I2=0, V=1) \) for ten days and compute from \( V(10) = V(0) e^{10} = e^{10} \) that \( r = \log[V(10)/10] \approx 1.5 \text{ d}^{-1} \).

The dominant eigenvalue of the infected steady state is \( \lambda \approx 1.4 \) which is close to the desired growth rate of \( r = 1.5 \text{ d}^{-1} \). This is natural because this eigenvalue gives the growth rate along the only eigenvector pointing outwards.

b. Running \( \text{continue}(s,x="\text{beta"},y="\text{V"},ymin=-0.01) \), one finds that the uninfected steady state becomes stable at \( \beta \approx 2.2 \), which corresponds to \( R_0 = 1 \). This means that when \( (1 - \epsilon_\beta) \times 9.1 < 2.2 \) or \( \epsilon_\beta > 1 - 2.2/9.1 = 0.76 \) the virus should be eradicated in this model. This obviously does not happen in reality because there are latently infected cells.

c. A therapy correspond to \( \epsilon_\beta = 0.9 \) does give a slope close to \( \delta = 1 \text{ d}^{-1} \). Because Gadhamsetty et al. [4] change \( d_1 \) and \( d_2 \) when they consider early and late killing, their parameterization is designed to deliver the desired \( \delta \approx 1 \) in both cases. Note that during perfect therapy the decline rate of the viral load ultimately approaches the slowest of the two infected cell populations.

d. In the early killing regime, adding on an efficacious therapy blocking \( \gamma \) steepens the initial downslope of the viral load, but slows down the late phase. In the end it takes much longer before the virus is “eradicated”. Adding an efficient therapy can therefore worsen the outcome [1], and the reason is that the slowest compartment, \( I_1 \), has become even slower, and will keep on producing \( I_2 \) cells over a much longer period of time. The same unexpected outcome does not happen in the early killing scenario. Since we do not know where the killing takes place, one should read the Cardozo et al. [1] before taking this drug.

e. Implementing the immune response used by Gadhamsetty et al. [4] reveals how the onset of the immune response reduces the set point viral load that is approach after the acute phase of the infection. Because it delivers similar killing rates at steady state the treatment results are hardly affected.

**Question 12.7. Stem cell renewal**

a. When on average half of the stem cell divisions deliver a new stem cell, their cell division is
not changing the density of stem cells, and on average delivers a single daughter cell into the population of differentiated cells:

\[
\frac{dS}{dt} = -d_S S \quad \text{and} \quad \frac{dD}{dt} = p_S S - d_D D ,
\]

where \(p_S\) is the fixed division rate of the stem cells, and the \(d\) parameters are death rates. This illustrates that the stem cell population will go extinct and that more than half of their divisions have to be asymmetric to compensate for their death rate (many models therefore set \(d_S = 0\)). Thus, if \(f\) is the fraction of asymmetric divisions, and one needs to solve

\[
\frac{dS}{dt} = -p_S S + 2fpS - d_S S = p_S(2f - 1)S - d_S S = 0 \quad \text{with} \quad \frac{dD}{dt} = 2p_S(1 - f)S - d_D D ,
\]

\(d\)

\[d\]

to derive that the stem cells will be at steady state when \(f = \frac{1 + d_S}{2p_S}\) (which indeed approaches \(f \to 1/2\) when \(d_S \ll p_S\)). Note that it is very unlikely that stem cells “know” this parameter expression for \(f\), which strongly suggests that the fraction of asymmetric divisions has to be regulated by the (local) environment.

b. The previous equation was already written with a free parameter, \(f\), for the fraction of asymmetric divisions, and we only need to rewrite that into a function, \(0 < f(D) \leq 1\), that should should decline with the density \(D\). A general choice would be a Hill function, e.g.,

\[
\frac{dS}{dt} = p_S[2f(D) - 1]S - d_S S = 0 \quad \text{and} \quad \frac{dD}{dt} = 2p_S[1 - f(D)]S - d_D D \quad \text{with} \quad f = \frac{1}{1 + D/h_f} .
\]

c. To allow for a density dependent division rate of the stem cells one multiplies the parameter \(p_S\) with another function, \(g(D)\) for growth rate, also declining as a function of \(D\):

\[
\frac{dS}{dt} = p_Sg(D)[2f(D) - 1]S - d_S S = 0 \quad \text{and} \quad \frac{dD}{dt} = 2p_Sg(D)[1 - f(D)]S - d_D D ,
\]

\(d\)

\[d\]

\(d\)

with \(f = \frac{1}{1 + D/h_f}\) and \(g = \frac{1}{1 + D/h_g}\). We have now arrived at the full, and quite complicated terms of the Lander et al. [7] model. Note that reading this equation is almost more difficult than deriving it.

d. If differentiated cells also divide we can add a similar growth term to \(dD/dt\):

\[
\frac{dS}{dt} = p_Sg(D)[2f(D) - 1]S - d_S S = 0 \quad \text{and} \quad \frac{dD}{dt} = 2p_S[1 - f(D)]g(D)S + p_D G(D) - d_D D ,
\]

\(d\)

\[d\]

\(d\)

where \(G = \frac{1}{1 + D/h_G}\). There will be two dynamical regimes because the differentiated cells only strictly depend on the stem cells when \(p_D < d_D\), i.e., if their maximal self-renewal rate cannot fully compensate for their death rate. Note that Lander et al. [7] also allow for asymmetric division in the early stages of the differentiated cells.

e. Having \(K\) sites where stem cells can bind, the number free sites is given by \(F = K - S\). Assuming that one daughter re-occupies the spot of the mother cell, the probability that the other daughter finds a spot adjacent to the mother cell would be \(f = F/(K - 1) \simeq F/K\), when \(K\) is large. This would give something like

\[
\frac{dS}{dt} = p_S fS - d_S S \quad \text{with} \quad \frac{dD}{dt} = (1 - f)p_S S - d_D D \quad \text{where} \quad f = \frac{K - S}{K - 1} \simeq 1 - \frac{S}{K} ,
\]

where the fraction of asymmetric divisions depends almost linearly on the stem cell density.

**Question 12.8. Early warning signals**

a. Using `continue(state=s,x="c",y="X",xmin=0.1,xmax=3,ymin=10)` one obtains the classical picture with two saddle-node bifurcations.
b. Drawing normally distributed disturbances of the population size (with 10% standard deviation) one could run something like the following R-script, where the call to plot() depicts $X_{t+1}$ as a function of $X_t$, and the call to cor() computes the correlation. One should do this for various values of $c$ to test if the variation and the auto-correlation increases when the saddle-node bifurcation is approached:

```
after <- "state[1]<-abs(state[1]*rnorm(1,1,0.1))"
p["c"] <- 2; s <- newton(run())   # start at steady state
data <- run(750,after=after,table=TRUE)
plot(data$X[1:nrow(data)-1],data$X[2:nrow(data)],type="p")
cor(data$X[1:nrow(data)-1],data$X[2:nrow(data)])
```

c. Use `after <- "parms[1]<-abs(parms[1]*rnorm(1,1,0.1))"`.

d. Using the parameters in the model defines by warning.R we basically get no early warning signal.

**Question 12.9. Linear models**

The steady state is $x = y = 0$ and the Jacobian, $J = \begin{pmatrix} a & b \\ c & d \end{pmatrix}$, is the same as the interaction matrix. Use Fig. 13.8 to create an interaction matrix with the eigenvalues corresponding to the different types of steady states.

**Question 12.10. Noise and $r$ and $K$-selected species**

$r$-selected species recover more quickly from disturbances of the population density, but can also fluctuate more than $K$-selected species by tracing the variation in parameter values.

**Question 12.11. Sexual reproduction**

Figure made with the previous version of Grind:

A model with density dependent death rates would be something like

\[
\frac{dN_1}{dt} = N_1 \left[ \frac{b_1N_1}{h + N_1} - d_1(1 + e_1N_1 + c_1N_2) \right] \quad \text{and} \quad \frac{dN_2}{dt} = N_2 \left[ \frac{b_2N_2}{h + N_2} - d_2(1 + e_2N_2 + c_2N_1) \right]
\]

This model is available as the file sexual.R. Note that one has to separate birth from death because the sexual reproduction should only affect reproduction, and not the death. Assuming that the chance to find a mate approaches one when the population is close to its carrying capacity, i.e., assuming $h \ll K$, the carrying capacity is approximately $K_i \simeq (R_{0i} - 1)/e_i$. In the absence of sexual reproduction, i.e., when $h \rightarrow 0$, the nullclines are solved from $b_i - d_i(1 + e_iN_i + c_iN_j) = 0$ delivering the normal straight lines

\[
N_2 = \frac{R_{01} - 1}{c_1} - \frac{e_1}{c_1}N_1 \quad \text{and} \quad N_2 = \frac{R_{02} - 1}{e_2} - \frac{e_2}{e_2}N_1
\]
which may or may not intersect, intersect in a stable state when there is resource competition, and intersect in an unstable steady state when there is interference competition. From these three situations one can sketch the three Panels depicted above. For instance, the \( \frac{dN_1}{dt} = 0 \) nullcline is given by

\[
N_2 = \frac{1}{c_1} \left[ R_0 \frac{N_1}{h + N_1} - 1 \right] - \frac{e_1}{c_1} N_1,
\]

which resembles the straight line with slope \(-e_1/c_1\) for \( N_1 \gg h \), and which gives \( N_2 = -1/c_1 \) when \( N_1 = 0 \). Panel (a) would correspond to non-intersecting nullclines, Panel (b) to resource competition (i.e., \( c_i < e_i \)), and Panel (c) to resource competition (i.e., \( c_i > e_i \)). Note that sexual reproduction implies an Allee effect, and that \((0,0)\), and the two carrying capacities are always stable (stable states are marked by closed boxes, unstable states by open boxes).

**Answers to Chapter 13**

**Question 13.1. Sketch a few functions**

Figures made with the previous version of Grind:

(a) \( y = \frac{h}{h+x} \) is a straight line with slope 1, and which gives \( y = 1 \) when \( x = 0 \). For \( x \to \infty, y \to 0 \). For \( x \to -\infty, y \to 0 \). There is an vertical asymptote at \( x = -h \). See Panel (a).

(b) \( y = \frac{x}{h+x} = 0 \) when \( x = 0 \). For \( x \to \infty, y \to 1 \). For \( x \to -\infty, y \to 1 \). There is an vertical asymptote at \( x = -h \). See Panel (b).

(c) \( L = \frac{aA}{c+bd} \) with horizontal asymptote \( L = a/b \) or \( A = \frac{cL}{a-bL} \) with vertical asymptote \( L = a/b \). See Panel (c).
d. Write \( Y = 0 \) and \( X = (a/b)(1 - Y)(c + Y) \). See Panel (d).

e. Intersection with \( x \)-axis: \( x = \frac{ak - dq - dk}{a - d} \),
intersection with \( y \)-axis: \( y = \frac{ak}{a + k} - d \)
Horizontal asymptote: \( y = a - d \), and vertical asymptote: \( x = q + k \). See Panel (e), where
the dashed lines denote the two asymptotes.

**Question 13.2. Linearization**

a. \( \partial_x x^2 = 2x \)

b. For \( x = 3 \) one obtains \( x^2 = 9 \)

c. \( y = 9 + 0.1 \times 2 \times 3 = 9.6 \). The true value is \( 3.1^2 = 9.61 \).

**Question 13.3. Scaling**
The Lotka Volterra equations are

\[
\frac{dR}{dt} = [r(1 - R/K) - aN]R \quad \text{and} \quad \frac{dN}{dt} = [caR - d]N
\]

a. Defining \( x = R/K \) and dividing all rates by \( r \) one obtains

\[
\frac{dKx}{dt} = [(1 - Kx/K) - aN/r]Kx \quad \text{and} \quad \frac{dN}{dt} = \left[\frac{ca}{r}Kx - d/r\right]N
\]

and by defining \( \alpha = a/r \) this simplifies into

\[
\frac{dx}{dt} = [(1 - x) - \alpha N]x \quad \text{and} \quad \frac{dN}{dt} = \left[\frac{ca}{r}Kx - d/r\right]N
\]

with only one parameter in the resource equation. Defining \( y = \alpha N \), i.e., \( N = y/\alpha \), we
remove that parameter from \( dx/dt \)

\[
\frac{dx}{dt} = [(1 - x) - y]x \quad \text{and} \quad \frac{1}{\alpha} \frac{dy}{dt} = \left[\frac{ca}{r}Kx - \frac{d}{r}\right] \frac{y}{\alpha}
\]

where \( dy/dt \) can be simplified by lumping the parameters

\[
\frac{dy}{dt} = [\gamma x - \delta]y ,
\]

where \( \gamma = \alpha K = cK\alpha/r \) and \( \delta = d/r \).

b. We went from five to two parameters for which we even know that is a scaled fitness \( R_0 = \gamma/\delta \),
and that \( \gamma/\delta > 1 \) is required for co-existence.
Bibliography


