Dynamical systems' preliminaries

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Population dynamics
Mathematical Methods in Biology, MAMME
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1. Flows and bifurcation of fixed points in continuous 1D models

See [S.H.Strogatz, Nonlinear Dynamics and Chaos, Part I.]

Representation and local analysis

$$\dot{x} = f(x), \qquad x \in \mathbb{R}$$

Fixed points satisfy $f(x) = 0$.

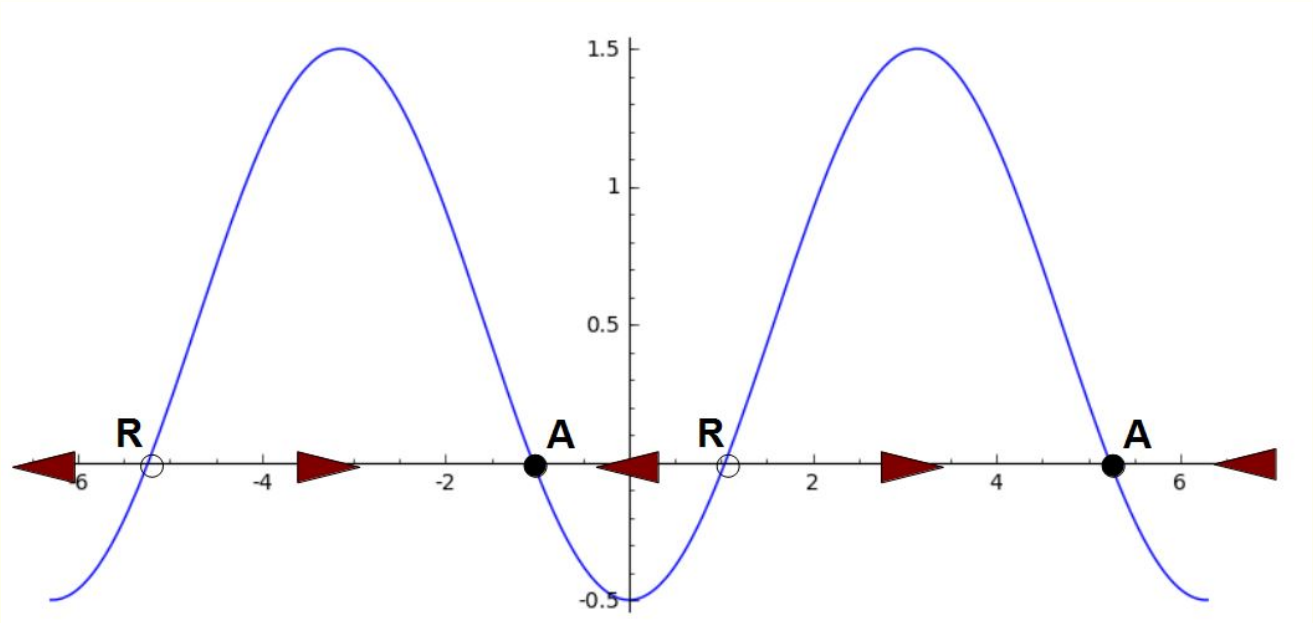


Figure: Dynamics of $\dot{x} = 0.5 - \cos x$ on the line.

Remind **exact and numerical methods for ODEs,** see also `PopDyn020-DifferentialEquations.sagesws`.

Representation and local analysis

The sign of the derivative at a fixed point determines its stability.

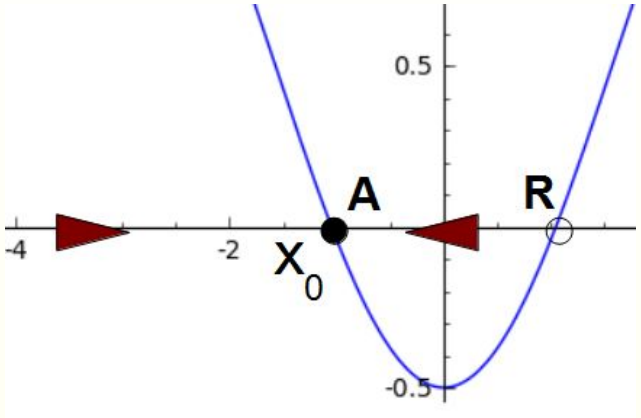


Figure: Dynamics around x_0, where $f(x_0) = 0$.

Linear stability analysis:

Consider $\eta = x - x_0$. Sufficiently close to x_0, if $f'(x_0) \neq 0$,

$$\dot{\eta} = \dot{x} = f(x) = f'(x_0)\eta + O(\eta^2)$$

and, then,

$$f'(x_0) < 0 (> 0) \Leftrightarrow x_0 \text{ in an Attractor (resp., Repellor)}$$

Generic co-dimension 1 bifurcations

Bifurcations arise when $f'(x_0) = 0$. They naturally appear in one-parameter (co-dimension 1 bifurcations) families of differential equations, see [Strogatz, pp. 44–61].

$$\dot{x} = f(x, \lambda), \qquad x \in \mathbb{R}, \qquad \lambda \in \mathbb{R}(^p),$$

Inside a family, $x_0 = x_0(\lambda)$, and so we search for λ_0 such that

$$D_x f(x, \lambda)|_{(x_0(\lambda_0), \lambda_0)} = 0.$$

The most common are:

- Transcritical bifurcation
- Saddle-node bifurcation
- Pitchfork bifurcation

Transcritical bifurcation

Normal form:

$$\dot{x} = r\,x - x^2$$

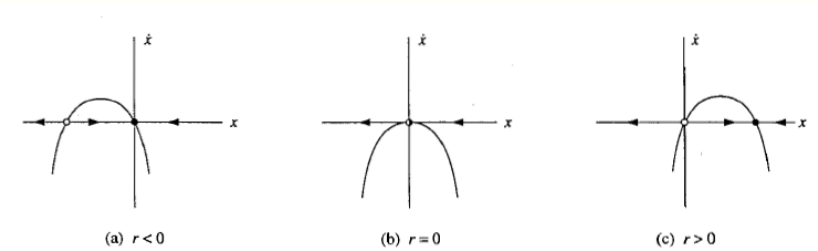


Figure 3.2.1

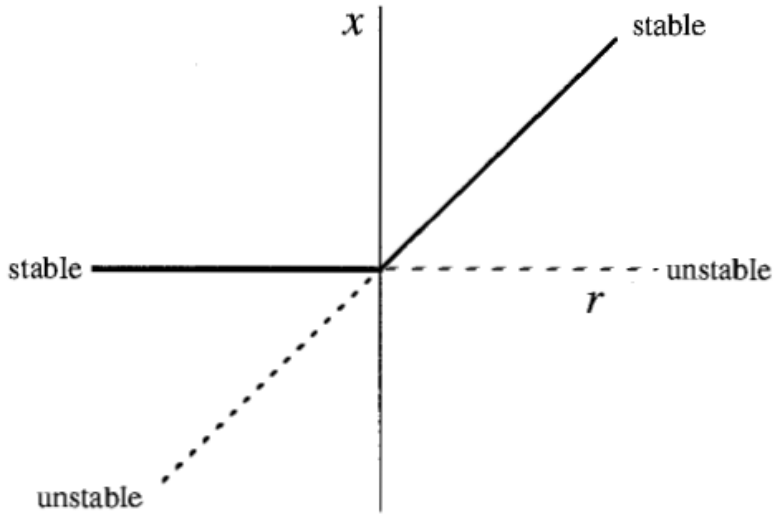


Figure 3.2.2

Figure: Different relative positions of $f(x)$ in the transcritical bifurcation.

Saddle-node bifurcation

Normal form:

$$\dot{x} = r + x^2$$

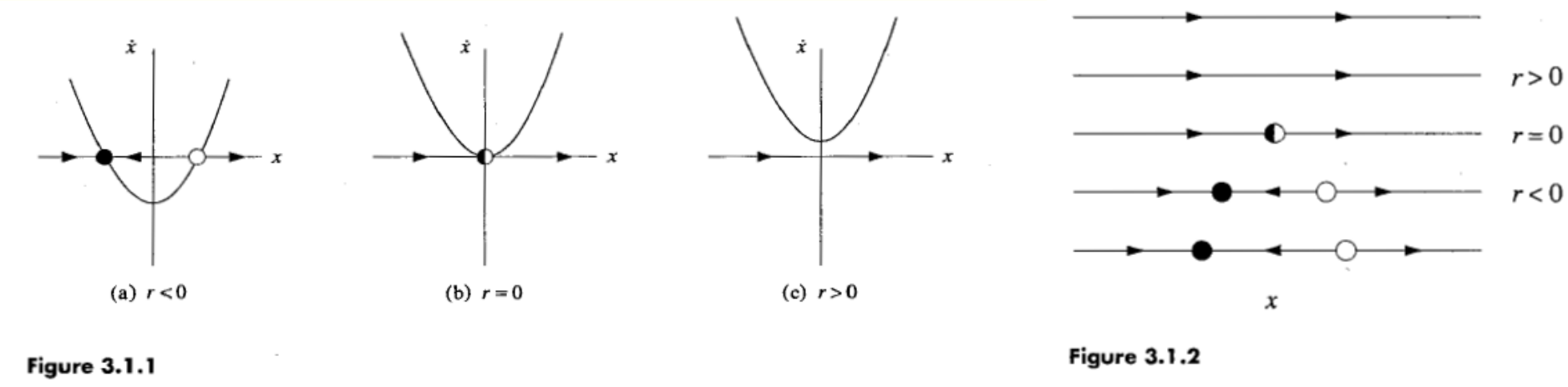


Figure 3.1.1

Figure 3.1.2

Figure: Different relative positions of $f(x)$ in the saddle-node bifurcation.

Note its genericity for **parabola-like** one-parameter families.

Saddle-node bifurcation

Normal form:

$$\dot{x} = r + x^2$$

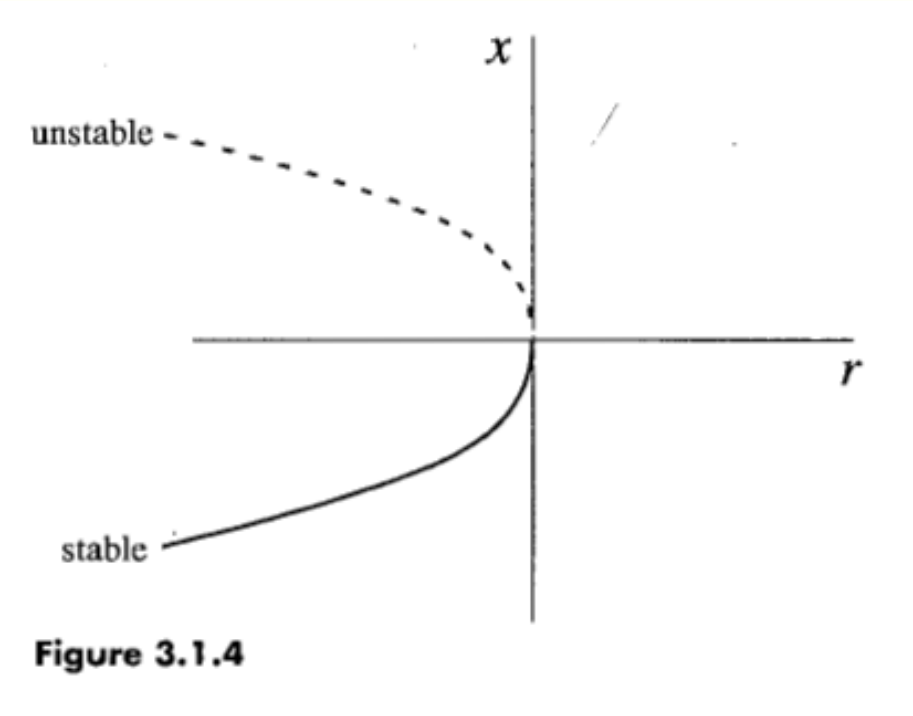


Figure: Different relative positions of $f(x)$ in the saddle-node bifurcation.

Supercritical pitchfork bifurcation

Normal form:

$$\dot{x} = r\,x - x^3$$

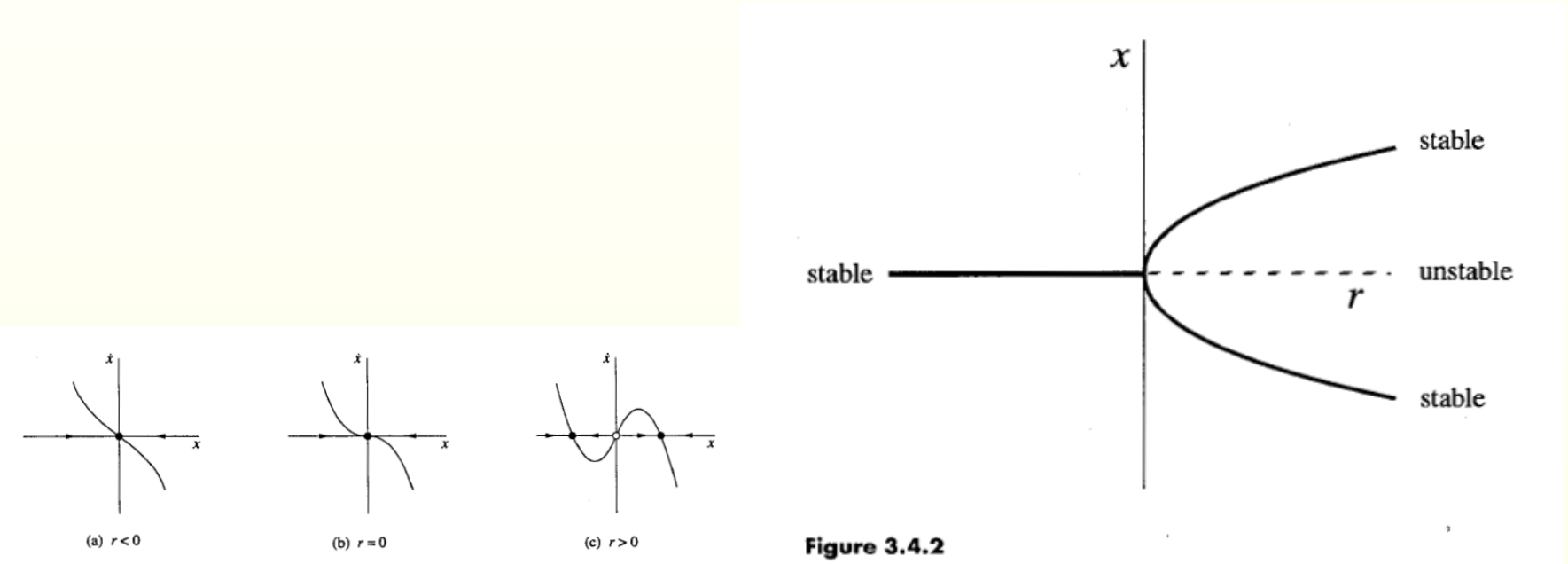


Figure: Different relative positions of $f(x)$ in the saddle-node bifurcation.

Subcritical pitchfork bifurcation

Normal form:

$$\dot{x} = r\,x + x^3$$

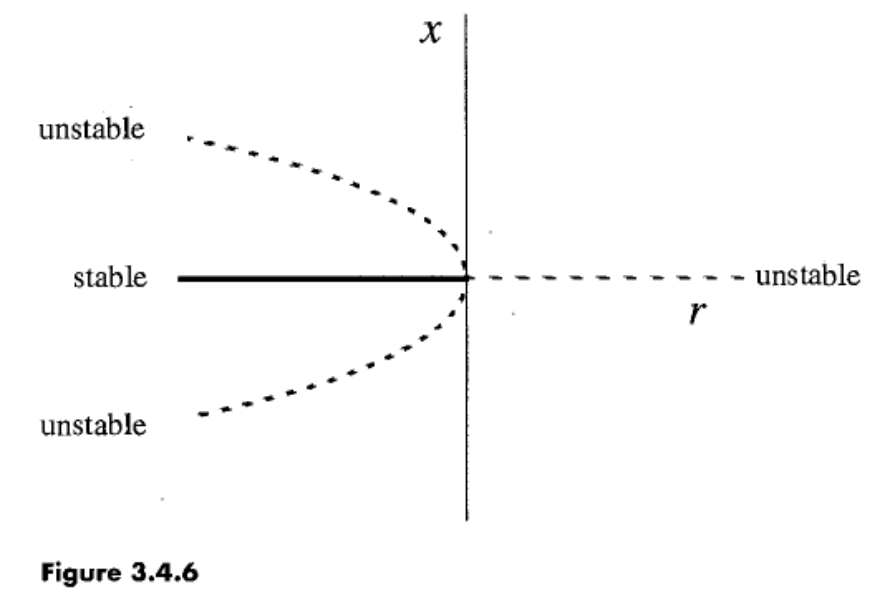


Figure: Different relative positions of $f(x)$ in the saddle-node bifurcation.

Co-dimension 2 bifurcations

Consider $\dot{x} = h + r\,x - x^3$.
If $h = 0$, it presents the classical *pitchfork bifurcation*. Parameter h breaks symmetry and gives rise to more interesting bifurcation diagrams, where two branches of *pitchfork bifurcation* merges at a cusp point.

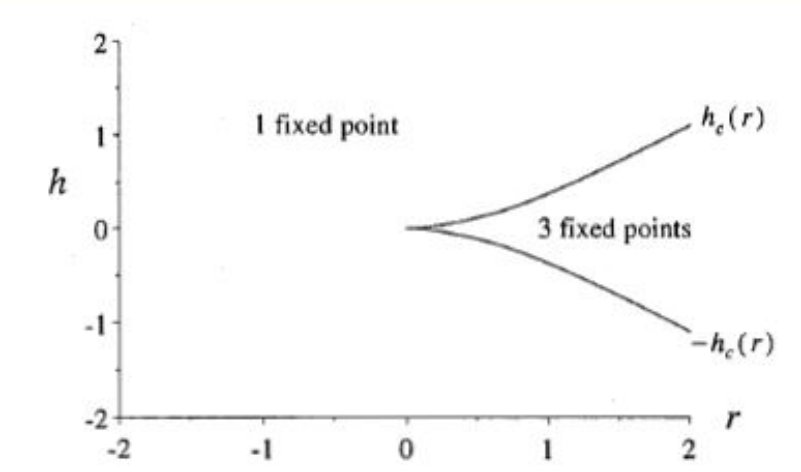


Figure: Biparametric bifurcation diagram, see [Strogatz, Fig. 3.6.2.].

2. Dynamics and bifurcations of fixed points in discrete 1D models

See [S.H.Strogatz, Nonlinear Dynamics and Chaos, Chapter 10.]

Representation and local analysis

$$x_{n+1} = f(x_n), \qquad x \in \mathbb{R}$$

We call them either 1D maps or 1D discrete dynamical systems.
Here, fixed points satisfy $f(x) = x$.
Linear stability analysis:
Sufficiently close to x^*,

$$x_{n+1} = f(x_n) = f(x^*) + f'(x^*)(x_n - x^*) + O((x_n - x^*)^2).$$

Consider $\eta_n = x_n - x^*$. Since $f(x^*) = x^*$:

$$\eta_{n+1} = f'(x^*)\eta_n + O(\eta_n^2)$$

and, then,

$$|f'(x^*)| < 1 (> 1) \Leftrightarrow x^* \text{ in an Attractor (resp., Repellor)}$$

Representation: time series, cobwebs and bifurcation diagrams, see `PopDyn030-1D-DiscreteDynamics-TheLogisticMap.sagesws`.

Generic co-dimension 1 bifurcations

Bifurcations arise when $|f'(x^*)| = 1$. They naturally appear in one-parameter (co-dimension 1 bifurcations) families of maps.

$$x_{n+1} = f(x_n, \lambda), \qquad x_n \in \mathbb{R}, \qquad \lambda \in \mathbb{R}(^p),$$

Inside a family, $x^* = x^*(\lambda)$, and so we search for λ^* such that

$$\left| D_x f(x, \lambda)|_{(x^*(\lambda^*), \lambda^*)} \right| = 1.$$

The most common are:

- Saddle-node (also called, *fold*), transcritical or pitchfork bifurcation, when $D_x f(x, \lambda)|_{(x^*(\lambda^*), \lambda^*)} = 1$. Just consider $x_{n+1} = x_n + r + x_n^2$, $x_{n+1} = x_n + r\, x_n - x_n^2$, and $x_{n+1} = x_n + r\, x_n - x_n^2$, respectively.
- Period-doubling or flip bifurcation, when $D_x f(x, \lambda)|_{(x^*(\lambda^*), \lambda^*)} = -1$
- Hopf bifurcation, when $D_x f(x, \lambda)|_{(x^*(\lambda^*), \lambda^*)} = a + b\, i$ with $a^2 + b^2 = 1$ and $b \neq 0$. It needs higher dimensions, but will often appear in this course.

3. Flows in higher dimensions: qualitative theory of dynamical systems

See [S.H.Strogatz, Nonlinear Dynamics and Chaos, Part II.]

Qualitative theory of dynamical systems

- Hartman-Grobman theorem: local hyperbolic dynamics.
- Stability of fixed points.
- Poincaré maps.
- Invariant manifolds of fixed points.
- Basic bifurcations.

Local analysis of fixed points

Consider a regular map (at least C^1),

$$f : \mathbb{R}^n \to \mathbb{R}^n$$

with a **hyperbolic** fixed point p:

$$f(p) = p; \qquad A = Df(p) \textbf{ hyperbolic linear map}.$$

(all eigenvalues with modulus different from 1).

Hartman-Grobman theorem: local hyperbolic dynamics

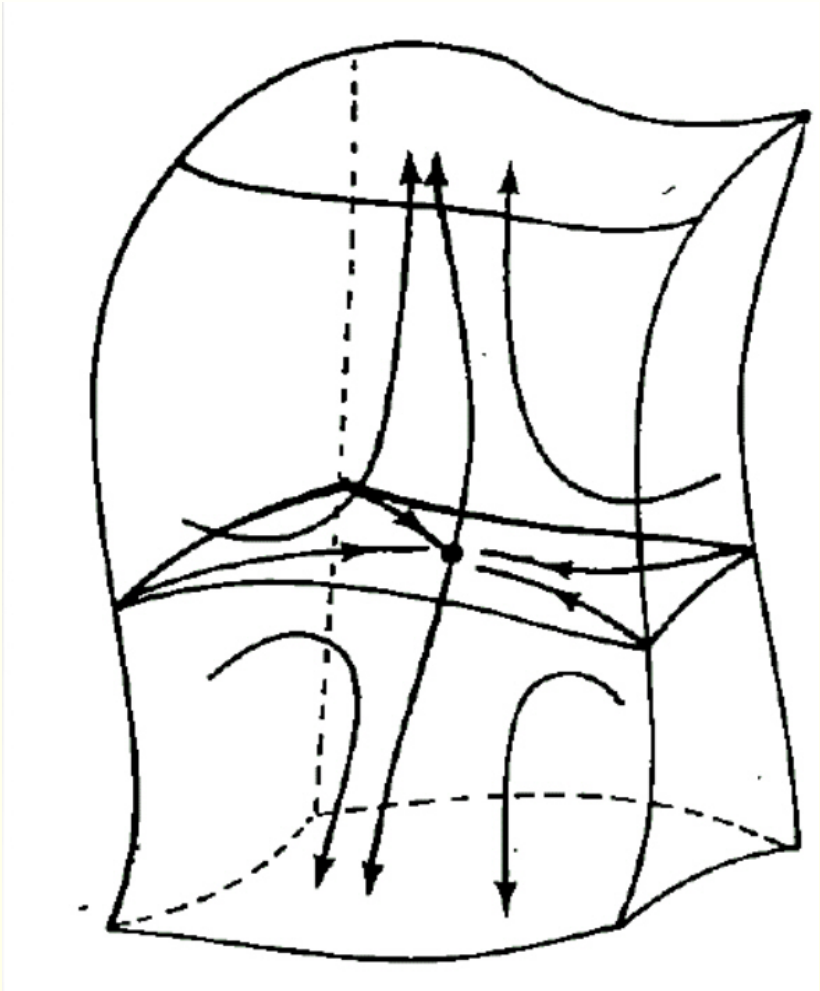
Theorem (Hartman-Grobman)

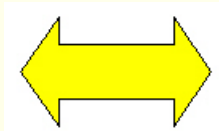
p hyperbolic fixed point of $f : \mathbb{R}^n \to \mathbb{R}^n$, regular. There exists a neighbourhood $U \subset \mathbb{R}^n$ of p and a homeomorphism $h : U \to \mathbb{R}^n$ such that

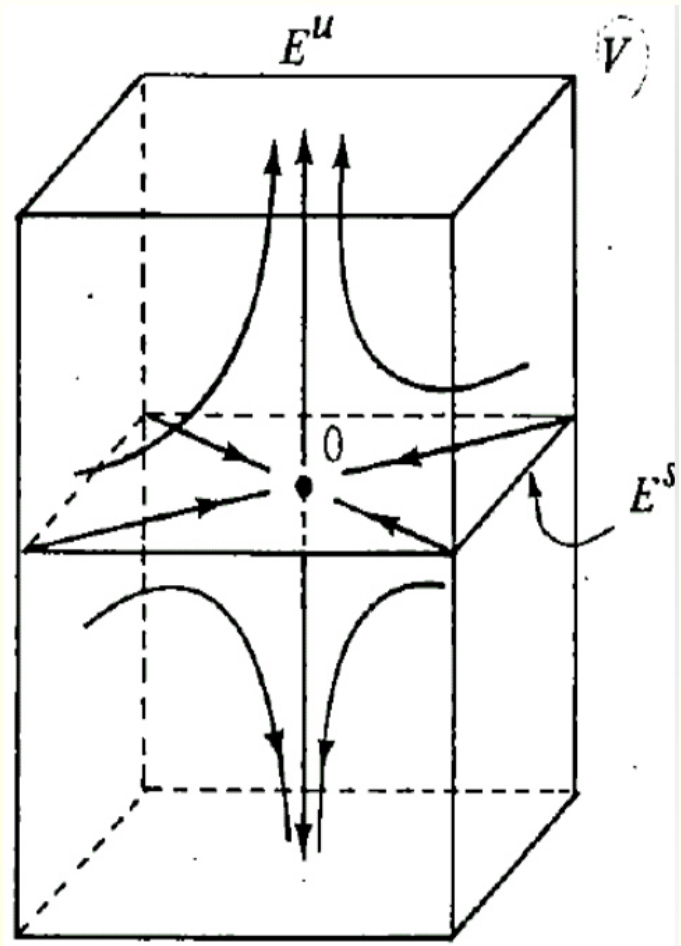
$$f_U = h^{-1} \circ A \circ h;$$

that is, f and its linearization around p are **topologically equivalent**.

Hartman-Grobman theorem: local hyperbolic dynamics







Maps vs differential equations

Differential equations $\dot{x} = F(x)$ can be thought of as maps considering the time-1 flow map $f(x) = \Phi(1; x)$.

- $F(p) = 0 \Rightarrow f(p) = p$.
- $Df(p) = \frac{\partial \Phi}{\partial x}(1; p)$. Variational equations:

$$\frac{d}{dt}\frac{\partial \Phi}{\partial x}(1; p) = DF(\Phi(1; p))\frac{\partial \Phi}{\partial x}(1; p) = DF(p)\frac{\partial \Phi}{\partial x},$$

and so,

$$Df(p) = \frac{\partial \Phi}{\partial x}(1; p) = \exp(DF(p)),$$

and

$$\sigma(Df(p)) = \exp(\sigma(DF(p))).$$

Then, p is hyperbolic if $DF(p)$ has all the eigenvalues with real part different from zero.

Stability of fixed points

Definition

A fixed point p is **asymptotically stable** if there exists a neighbourhood U of p such that for any $q \in U$, $f^k(q) \in U$ for any $k \geq 0$, and moreover

$$\lim_{k \to \infty} f^k(q) \to p.$$

The **basin of attraction** of p is the maximal set U.

Stability of fixed points: theorems

Theorem (Stability)

If all the eigenvalues of $Df(p)$ have modulus smaller than 1, then p is asymptotically stable.

Theorem (Stability of fixed points of ODEs)

If p is a fixed point of the flow $x' = F(x)$ with $F \in C^1(U)$ and all the eigenvalues of $Df(p)$ have negative real part, then p is asymptotically stable.

Theorem

A periodic orbit is asymptotically stable if the associated Poincaré map is asymptotically stable.

Liapunov stability approach

Definition

Given $F(p) = 0$, a C^1 function defined on U, neighbourhood of p, is a **Liapunov function** if:

1. $V(x) > 0$ for all $x \in U \setminus \{p\}$ and $V(p) = 0$ (V called positively defined).
2. $\dot{V}(x) < 0$ for all $x \in U \setminus \{p\}$, where

$$\dot{V}(x) = \langle \nabla V(x), F(x) \rangle$$

Second Liapunov method

Theorem

If there exists a Liapunov function for a fixed point p, then p is asymptotically stable.

Observe that

$$\dot{V}(x(t)) = \frac{d}{dt} V(x(t)) = \langle \nabla V(x(t)), x'(t) \rangle = \langle \nabla V(x(t)), F(x(t)) \rangle$$

implies that V monotonically decrease on the orbits.

Invariant manifolds of fixed points

Given $f : X \to X$ homeo with p a fixed point, the **stable set** of p is

$$W^s(f,p) = \{q \in X : f^n(q) \to p \text{ when } n \to \infty\};$$

the **unstable set** of p is

$$W^u(f,p) = \{q \in X : f^{-n}(q) \to p \text{ when } n \to \infty\}.$$

If U is a neighbourhood of p, the local stable/unstable sets are:

$$W^s_{\text{loc}}(f,p,U) = \{q \in U : f^n(q) \in U \text{ for all } n \geq 0\}$$

and

$$W^u_{\text{loc}}(f,p,U) = \{q \in U : f^n(q) \in U \text{ for all } n \leq 0\}$$

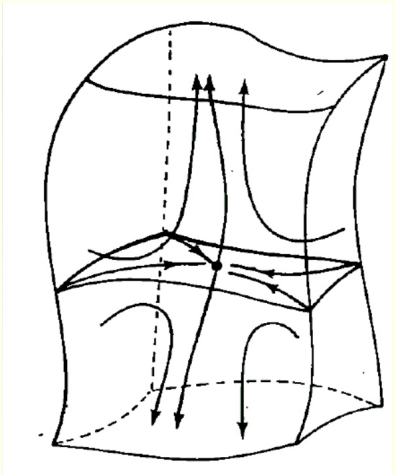
Stable manifold theorem

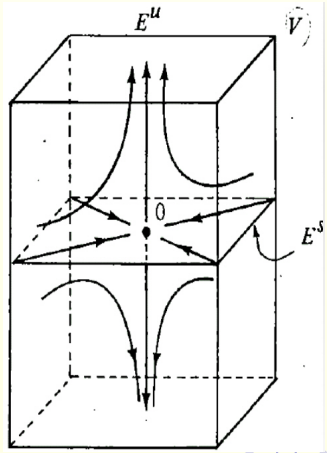
Theorem

$$f : U \subset \mathbb{R}^n \to \mathbb{R}^n$$

C^k map with a hyperbolic fixed point p. Then, there exists a neighbourhood U of p such that

1. $W^{s/u}(f, p) = W^{s/u}(f, p, U)$.
2. *$W^{s/u}(f, p)$ are C^k manifolds tangent, respectively, to $E^u(p)$ and $E^s(p)$, the stable and unstable space of the linearization of F on p.*
3. *The manifolds vary continuously in a neighbourhood of p.*





Stable manifold theorem: derived concepts and facts

- **Homoclinic** and **heteroclinic** orbits.
- **Homoclinic** and **heteroclinic** connections, or **separatrices**.
- Transversal intersections of stable and unstable manifolds lead to chaos.
- In 2D flows, separatrices “separate" the phase space, but this is no longer true for 2D maps.

Planar flows: Poincaré-Bendixson theorem

Given a flow $\varphi : \mathbb{R} \times X \to X$, a point x and an orbit γ containing x, a point y is called ω-**limit** of γ if there exists $(t_n)_{n\in\mathbb{N}}$ in $\mathbb{R}$ such that

$$\lim_{n\to\infty} t_n = \infty \qquad \lim_{n\to\infty} \varphi(t_n, x) = y.$$

(analogously, α-**limit**).

Theorem (Poincaré-Bendixson)

$$x' = f(x)$$

in an open simply connected planar set. If the ω-limit set of an orbit is compact and does not contain fixed points, then it is a periodic orbit.

Limit sets: points, periodic orbits, set points joined by separatrices.

Planar flows: Dulac criterion

Theorem

Let Ω be a simply connected planar region and we consider $x' = f(x)$, on $f \in C^1(\omega)$. Let $B \in C^1(\omega, \mathbb{R}^+)$ such that

$$\frac{\partial(Bf_1)}{\partial x_1} + \frac{\partial(Bf_2)}{\partial x_2}$$

neither vanishes nor changes sign in Ω. Then, there are no periodic orbits in Ω.

Basic (and most frequent) bifurcations

- **Saddle-node bifurcation:**
  http://www.scholarpedia.org/article/Saddle-node_bifurcation
- **Hopf bifurcation (sub/super):**
  http://www.scholarpedia.org/article/Andronov-Hopf_bifurcation
- **Saddle-node on an invariant curve (SNIC):**
  http://www.scholarpedia.org/article/Saddle-node_bifurcation
- **Saddle-node of limit cycles.**
- **Homoclinic bifurcation.**

Excellent overview of basic bifurcations at
http://www.math.uwaterloo.ca/šacampbe/mathbio/slides/bifnrev.pdf
(Sue Ann Campbell's web page).

Ecology

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4. One-dimensional models in ecology

4.1. Continuous models

4.1.1. Classical models with some historical notes

Malthusian growth I

Birth and death rates only (Malthus, 1798).

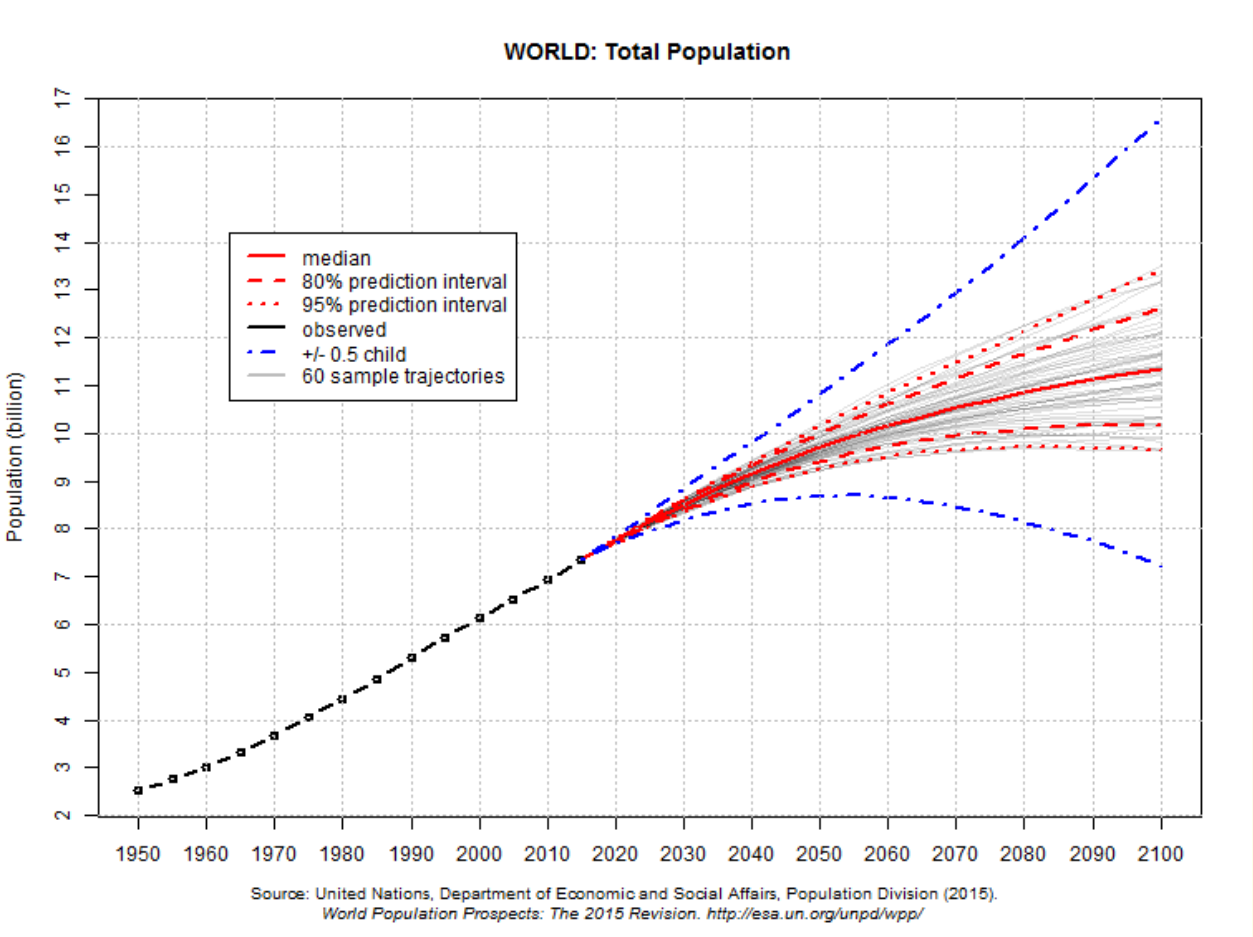
$$\frac{dN}{dt} = b\,N - d\,N \Rightarrow N(t) = N_0\,\exp((b-d)t).$$

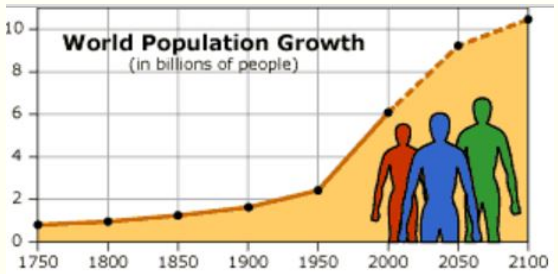
Malthus (1766-1834). Unrealistic approach...but the growth estimates for the total world population from the 17th to 21st centuries point towards an exponential growth...

World population predictions

Date	Mid 17th century	Mid 19th century	1918–1927	1960	1974	1987	2000	2050	2100
Population in billions	0.5	1	2	3	4	5	6.3	10	11.2

United Nations median projections for the 21st century





Probabilistic projections obtained from http://esa.un.org/unpd/wpp/Graphs/Probabilistic/POP/TOT/.

Data fitting, statistical treatment,... see http://esa.un.org/unpd/wpp/ for a complete information.

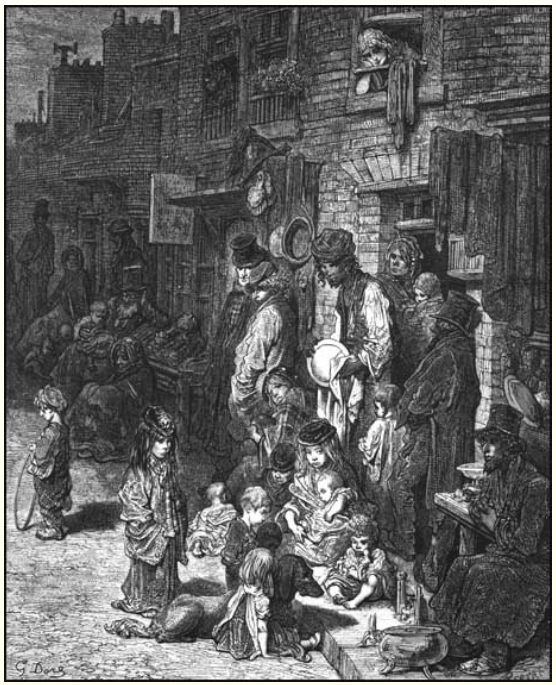
Compare, for instance, predictions for Africa (really exponential) and Europe (decreasing from now on!).

Malthusianism (population control)

"The great Malthusian dread was that indiscriminate charity would lead to exponential growth in the population in poverty, increased charges to the public purse to support this growing army of the dependent, and, eventually, the catastrophe of national bankruptcy. Though Malthusianism has since come to be identified with the issue of general over-population, the original Malthusian concern was more specifically with the fear of over-population by the dependent poor."

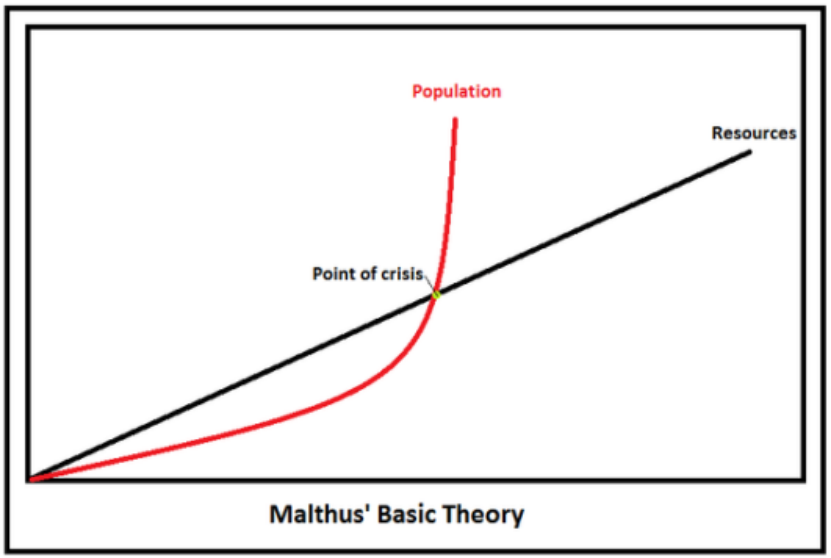
(Dan Ritschel,

http://history.umbc.edu/facultystaff/full-time/daniel-ritschel/)



Malthusianism (population control)

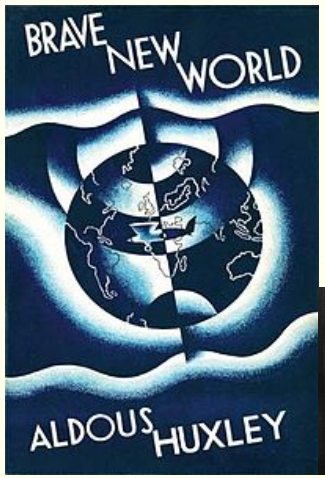
Exponential growth of population, linear growth of food. Moral restraints (abstinence, controlling marriages) or “positive checks" (diseases, starvation, war,...) resulting in Malthusian catastrophe.

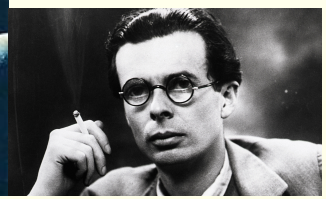


Population control: a permanent debate I

- calls for population control versus fear to have enough people for a “productive society".
- difficult to make long-term, or even relatively short-term, predictions unless we know sufficient facts.
- current consensus that ultimately, population growth on Earth is still too high, and will eventually lead to a serious crisis

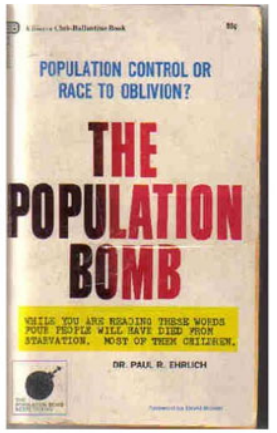
- Brave New World (Huxley,1931): Takes place in a dystopian state (inverse utopia). A benevolent dictatorship establishes a stable global society where the population is permanently limited.



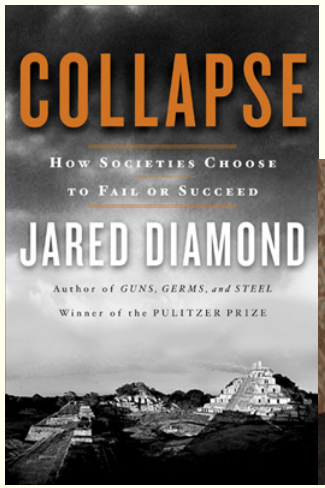


Population control: a permanent debate II

- The Population Bomb (Ehrlich,1968): warned about mass starvation of humans in the 1970s and 1980s due to overpopulation. Controversial, yes; alarmist?



- Collapse (Diamond, 2005): how societies choose to fail.



Population control: a permanent debate III

- Sustainability: see https://en.wikipedia.org/wiki/Sustainability, where high intersection with the course's topics can be found; United Nations Sustainable Development Summit 2015 http://www.unfpa.org/events/united-nations-sustainable-development-summit-2015;...

We go back to the historical review of models...

The logistic growth

$$\frac{dN}{dt} = r\,N\,(1 - x/K),$$

where

- r is the **growth rate**.
- K is the **carrying capacity**.

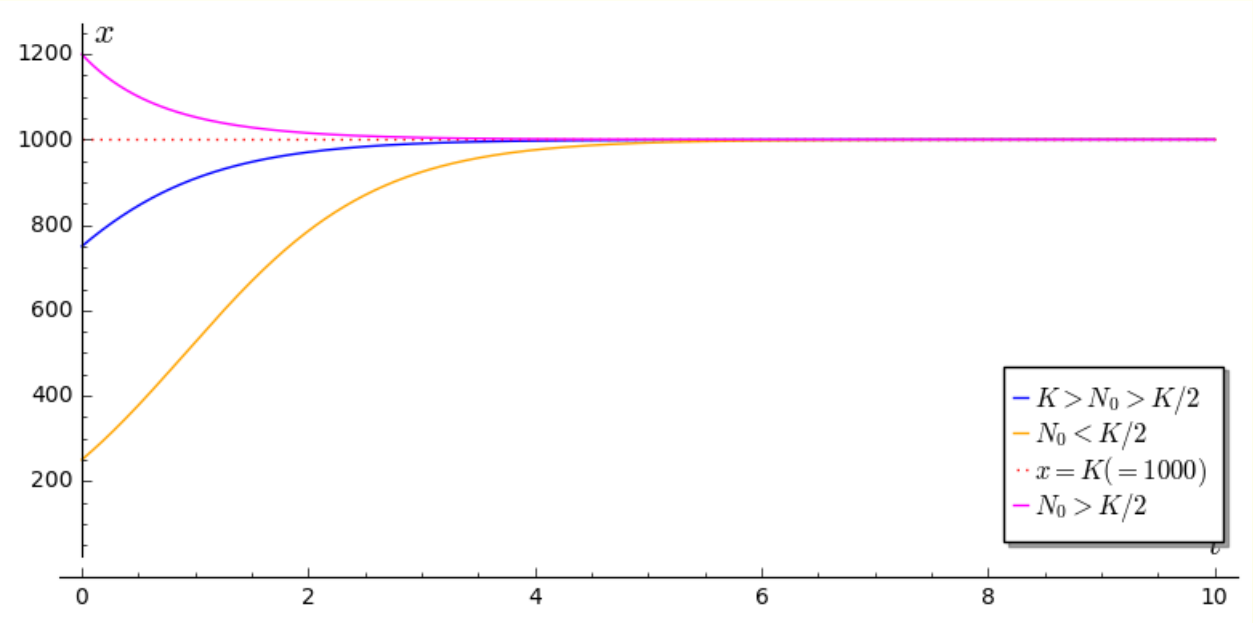
P. F. Verhulst
(1804–1849)

The continuous logistic growth: simple analysis

The points $N = 0$ and $N = K$ are the only stationary points (roots of $f(x)$). Since $f'(x) = r\left(1 - \frac{2N}{K}\right)$, $f'(0) = r > 0$ and $f'(K) = -r < 0$. Then $N = 0$ is a repellor and $N = K$ is an attractor.

In this case, the ODE can be solved explicitly and we see that all solutions tend to K as $t \to +\infty$:

$$N(t) = \frac{K\,N_0}{N_0 + (K - N_0)e^{-r\,t}}.$$

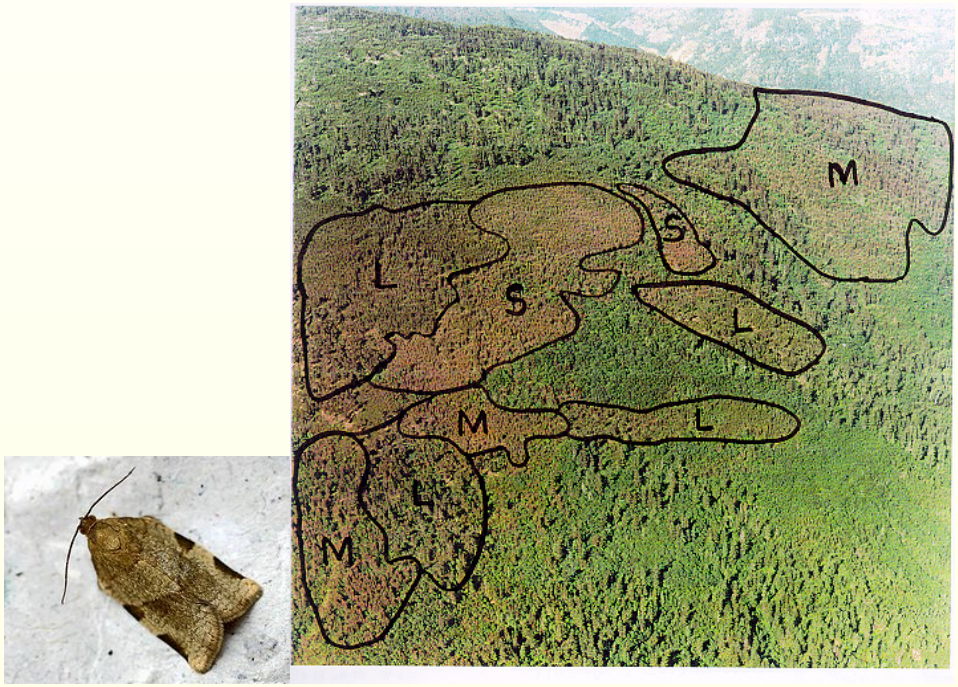


Observe the sigmoidal shape, ubiquitous in many mathematical models as a learning curve.

4.1.2. A model for the Spruce Budworm

A model for Spruce Budworm

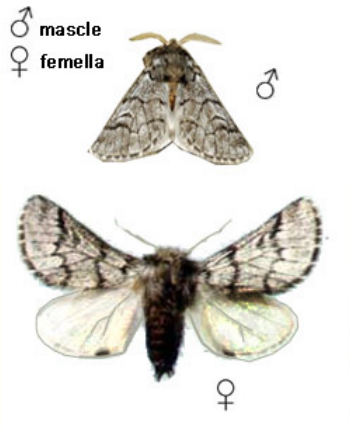
Spruce budworm (*Choristoneura fumiferana*) damage appears in May. Evidence of a spruce budworm infestation includes the destruction of buds, abnormal spreading of new twigs, defoliation of current-year shoots and, if an affected branch is disturbed, the presence of large numbers of larvae suspended from strands of silk.[1]

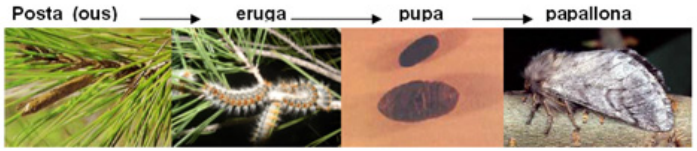


Pest damage: Examples of LIGHT, MODERATE and SEVERE mortality of lodgepole pine. See also http://www.ncrs.fs.fed.us/gla/natdist/mn_sbw.htm.

[1] See [Murray, Chap 1, pp. 7–13]

Examples in the Mediterranean area





Pine Processionary control

The model (Ludwig et al (1978))

$$\frac{dN}{dt} = r_B N\left(1 - \frac{N}{K_B}\right) - p(N), \quad p(N) = \frac{BN^2}{A^2 + N^2}.$$

- r_B is the worm growth rate and units are time^{-1}.
- K_B is the carrying capacity of the habitat without predation and, therefore, it has the same units as N.
- A is the population of worms when the rate of predation is half the maximum ($N_c = A$) and thus has the same dimensions as N.
- B is the maximum predation rate. B has the same units as the left hand side of the equation (rate growth per N) and their units are time^{-1}.
- $p(N)$, the predator's functional response tends to saturation for large values of N. Predation cannot grow unbounded. In between the behaviour is the following. For small values, predators tend to look for nutrients elsewhere. Around N_c, the predation is linear and for large values it saturates. Around $N = 0$ it grows less than linearly.

Equilibrium Points

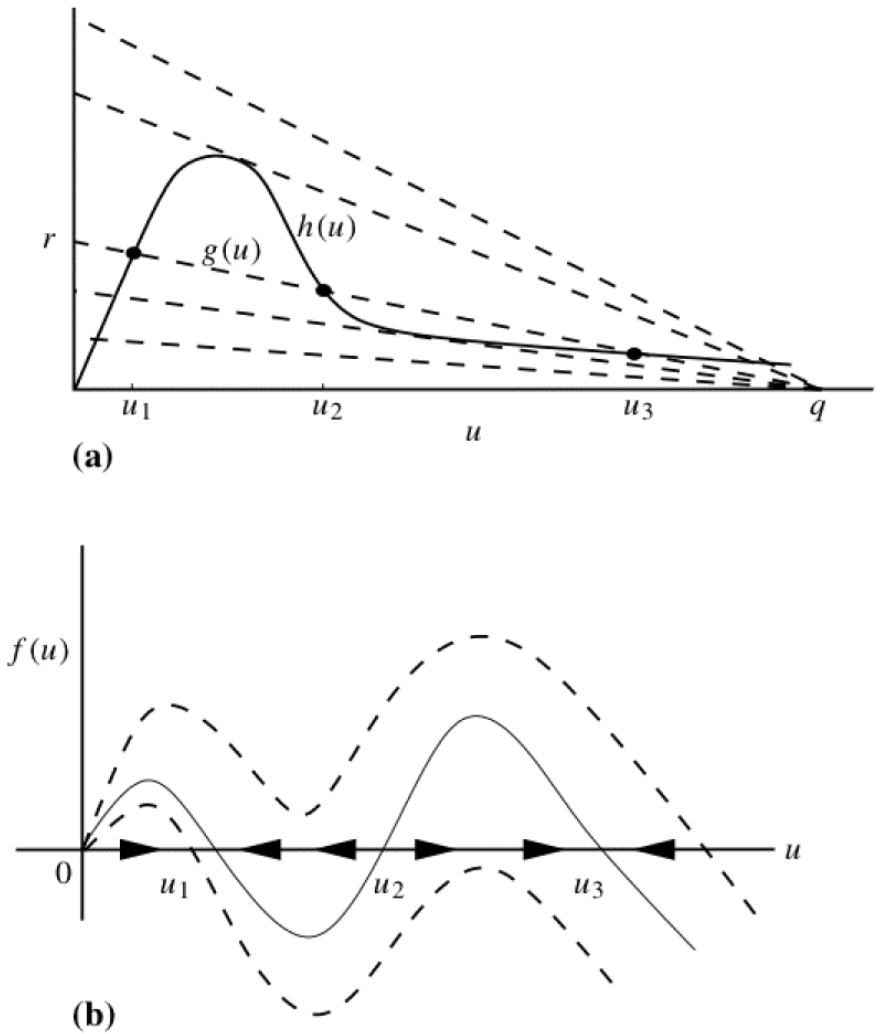


Figure 1.5. Equilibrium states for the spruce budworm population model (1.8). The positive equilibria are given by the intersections of the straight line $r(1 - u/q)$ and $u/(1 + u^2)$. With the middle straight line in **(a)** there are 3 steady states with $f(u; r, q)$ typically as in **(b)**.

Bifurcation Diagram

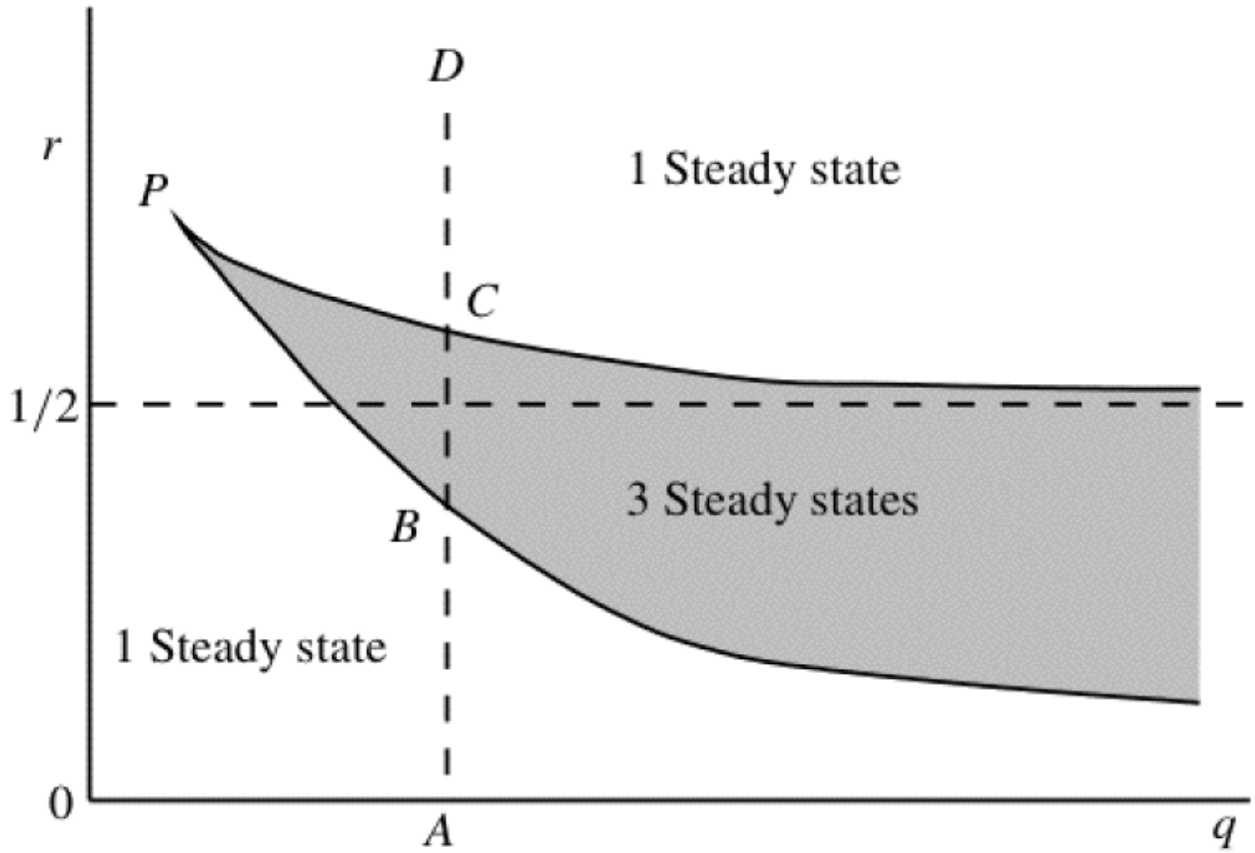


Figure 1.6. Parameter domain for the number of positive steady states for the budworm model (1.8). The boundary curves are given parametrically (see Exercise 1) by $r(a) = 2a^3/(a^2+1)^2, q(a) = 2a^3/(a^2-1)$ for $a \geq \sqrt{3}$, the value giving the cusp point P.

Hysteresis

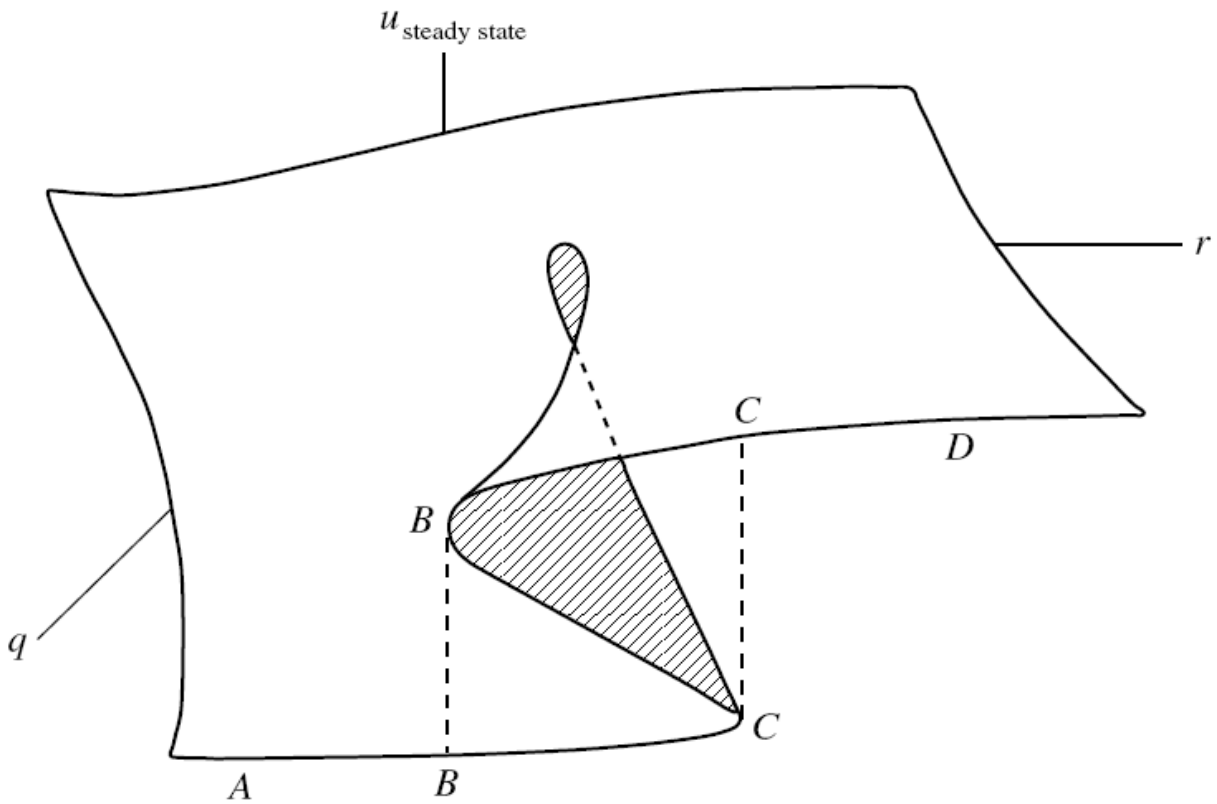


Figure 1.7. Cusp catastrophe for the equilibrium states in the $(u_{\text{steadystate}}, r, q)$ parameter space. As r increases from A, the path is $ABCCD$ while as r decreases from D, the path is $DCBBA$. The projection of this surface onto the r, q plane is given in Figure 1.5. Three equilibria exist where the fold is.

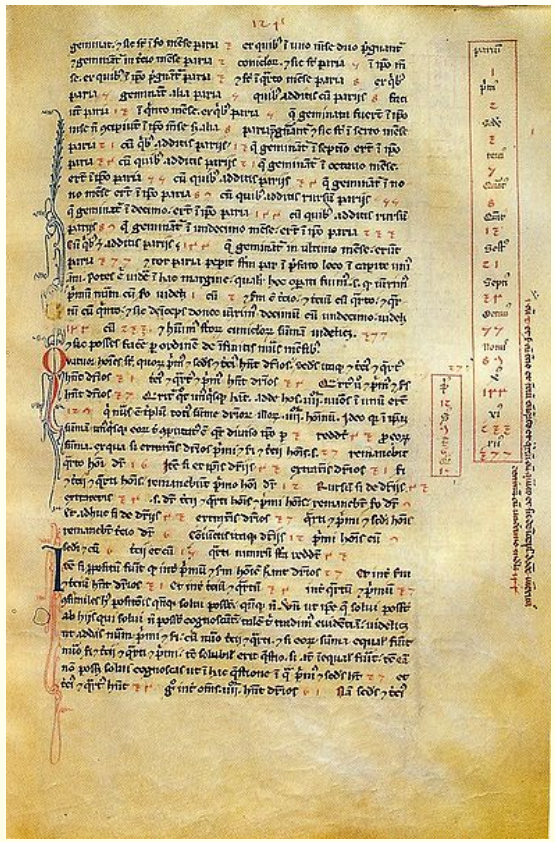
$\longrightarrow$ **Let's go to Sage!**

4.2. Discrete models

Historical examples

India (ca. 200 BC). Related to oral Sanskrit tradition . How long (L) syllables mix with the short (S), counting the different patterns of L and S. The number of patterns that are m short syllables long is the Fibonacci number F_{m+1}.

Leonardo of Pisa (Fibonacci), Liber abaci 1202.
Growth of an idealized (biologically unrealistic) **rabbit population**.



Fibonacci sequence

Idealized (biologically unrealistic) rabbit population:

- a newly born pair of rabbits, one male, one female, are put in a field;
- rabbits are able to mate at the age of one month (at the end of its second month a female can produce another pair of rabbits);
- rabbits never die and a mating pair always produces one new pair (one male, one female) every month from the second month on.

How many pairs will there be after one year?

$$F_m = F_{m-1} + F_{m-2}, \text{ with } F_0 = F_1 = 1,$$

0,1,1,2,3,5,8,13,21,...

Current modeling of discrete population dynamics

$$N_{t+1} = N_t F(N_t) = f(N_t), N_0 > 0.$$

Examples[2]:

- $F(N_t) = r => N_t = r^t N_0$ (discrete Malthusian).
- $f(N_t) = r N_t^{1-b}$ (survival to breeding).
- $F(N_t) = r(1 - N_t/K)$ (discrete Verhulst, logistic).
- $F(N_t) = \exp(r(1 - N_t/K))$ (severe death when overcrowded).

Time steps can be thought of as delays, so should we expect oscillatory dynamics as well?

[2][Murray, Mathematical Biology, Chapter 2]

Discrete population dynamics: the logistic map

$$N_{t+1} = r\,N_t(1 - N_t/K), \quad N_0 > 0. \text{ (discrete Verhulst)}$$

“Perhaps we would all be better off, not only in research and teaching, but also in everyday political and economical life, if more people would take into consideration that simple dynamical systems do not necessarily lead to simple dynamical behaviour." (Robert May, 1976)

Now, you will see why he said so...

Transcritical bifurcation in the logistic map

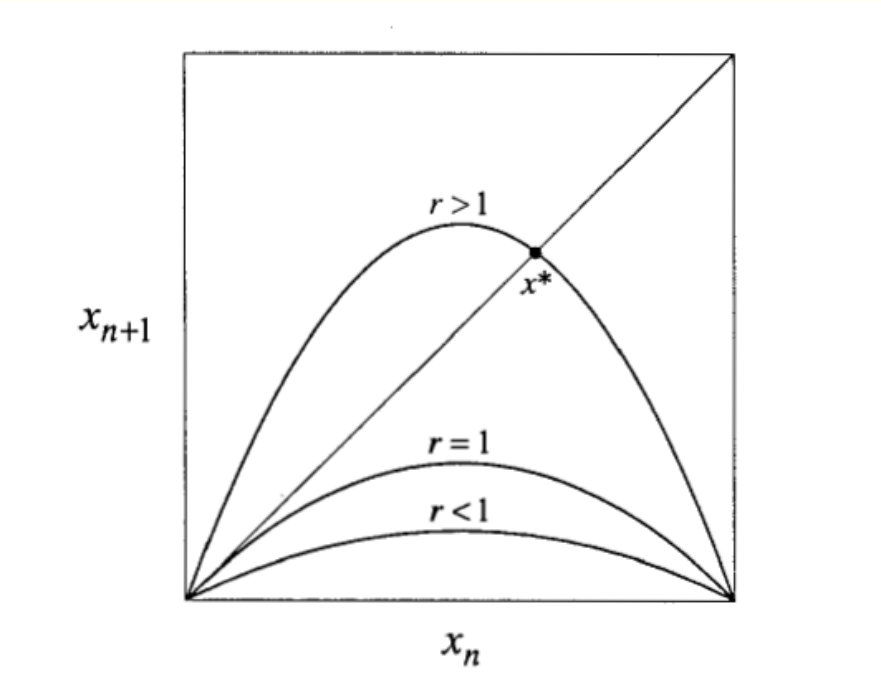
Important features:

- If $t = n$ and $x_n = N_t/k$ for all $t, n \in \mathbb{N} \cup \{0\}$, then $f(x) = r\,x\,(1-x)$.
- Fixed points of f:

$$f(x) = x \Leftrightarrow r\,x(1-x) = x \Leftrightarrow x = 0 \text{ or } x = 1 - 1/r =: x_r^*$$

- Transcritical bifurcation at $x = 0$

$$f'(0) = r = 1 \text{ when } r = 1.$$



Period doubling bifurcation

- Stability loss of $x^*(r)$ at $r = 3$:

$$f'(x^*(r)) = r(2/r - 1) = -1 \text{ when } r = 3.$$

- A stable 2-periodic orbit arises at $r = 3$ as $x^*(r)$ becomes unstable.

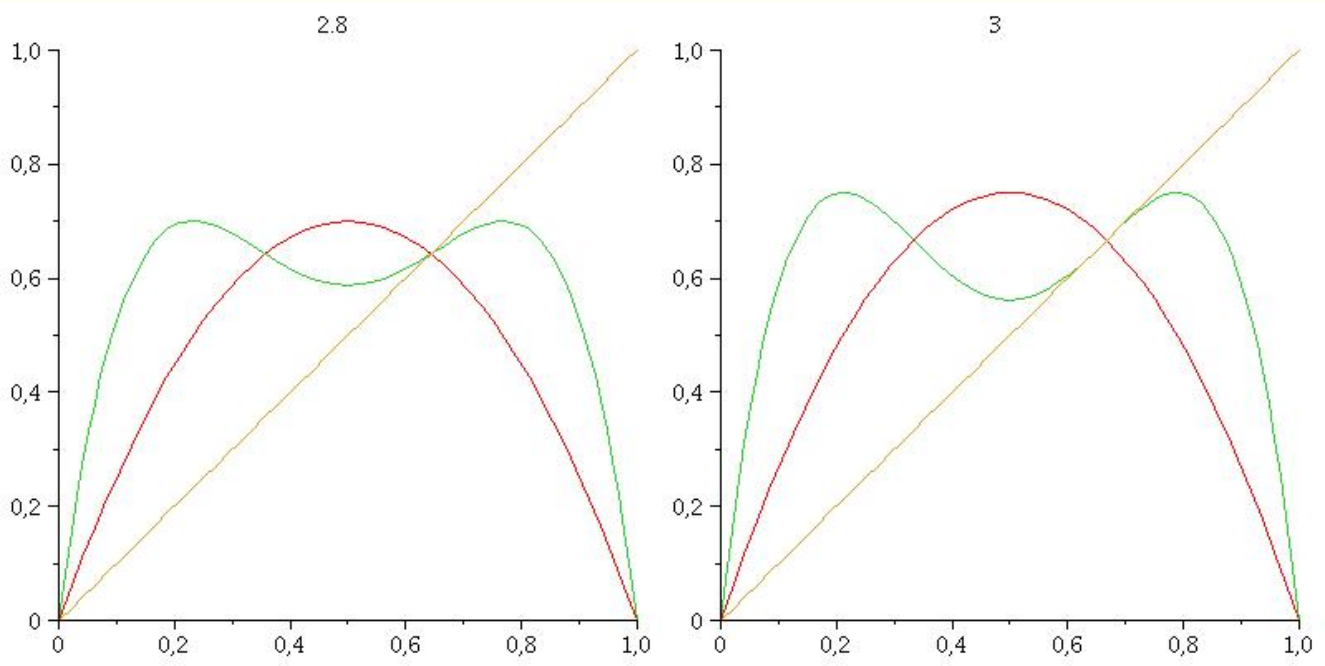
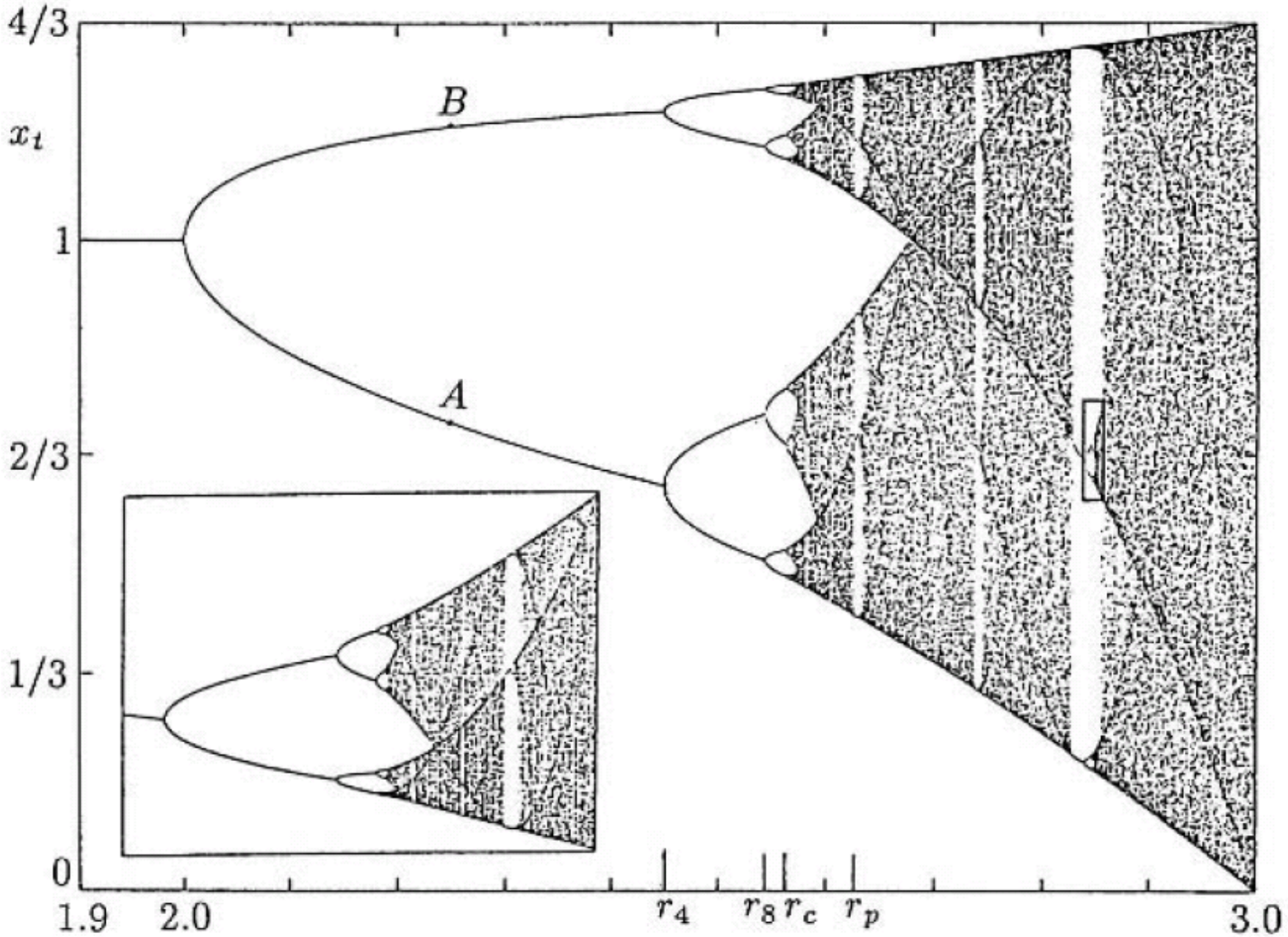


Figure: Graphs of $f(x)$ and $f^2(x) = f(f(x))$.

Period doubling bifurcation cascade

Value of r	New period
$r_1 = 3$	2^1
$r_2 = 3.449\ldots$	2^2
$r_3 = 3.54409\ldots$	2^3
$r_4 = 3.5644\ldots$	2^4
$r_5 = 3.568759\ldots$	2^5
$\vdots$	$\vdots$
$r_\infty = 3.569946\ldots$	∞

Period doubling bifurcation cascade



Detection of chaos (Lyapunov exponents) I

We want to determine the rate of divergence between two close trajectories.
Let x_0 and $x_0 + \delta_0$ be two close initial conditions ($|\delta_0| \ll 1$), and $\delta_n := f^{(n)}(x_0 + \delta_0) - f^{(n)}(x_0)$ the difference between the n-th iterates of the respective orbits ($x_i := f^{(i)}(x_0)$ for $i \geq 0$).
Assume that $|\delta_n| = |\delta_0| \exp(n\lambda)$. Then,

$$\begin{aligned}\lambda &= \frac{1}{n} \ln \left| \frac{\delta_n}{\delta_0} \right| = \frac{1}{n} \ln \left| \frac{f^{(n)}(x_0+\delta_0) - f^{(n)}(x_0)}{\delta_0} \right| \\ &\approx \frac{1}{n} \ln \left| f^{(n)\prime}(x_0) \right| = \frac{1}{n} \ln \left| \prod_{i=0}^{n-1} f'(x_i) \right| = \frac{1}{n} \sum_{i=0}^{n-1} \ln |f'(x_i)| .\end{aligned}$$

The Lyapunov exponent is defined as

$$\lambda := \lim_{n \to +\infty} \left\{ \frac{1}{n} \sum_{i=0}^{n-1} \ln \left| f'(x_i) \right| \right\},$$

Detection of chaos (Lyapunov exponents) II

assuming that this limit exists. Although not strictly correct, $\lambda > 0$ is taken as an indication of chaos[3].
In higher dimensions, since it depends on the vector δ_0, there arises an spectrum of Lyapunov exponents.

[3] Although the definition of chaos itself is another controversial issue.

5. Dynamics of Interacting Populations

5.1. Populations interacting through symbiosis, competition, predation,...

See 5.1 at 5-1-DynamicsOfInteractingPopulations-20160331.pdf

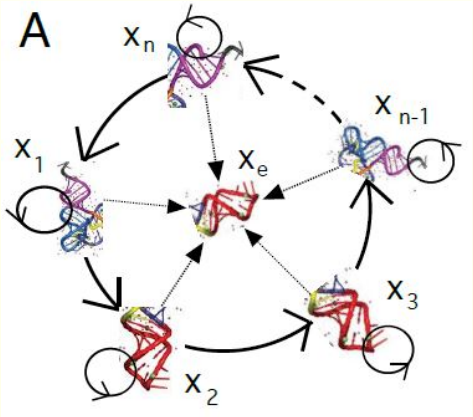
5.2. Oscillating hypercycles at the origin of life: an example of cooperative dynamics

Eigen's paradox and the origin of life

- One of the most solid theories to explain **how did complexity arise at the origin of life**?
- **Quasispecies:** large group of related genotypes that replicate under high mutation rates.
- Since the size of replicator molecules is limited by the accuracy of replication, large sequences (like RNA) would not be possible because of loss of information across generations $\longrightarrow$ **Eigen's paradox (1971):** No possible mechanisms of error correction without large information contents and no possible large information contents without error correction mechanisms.

Hypercycles and the origin of life

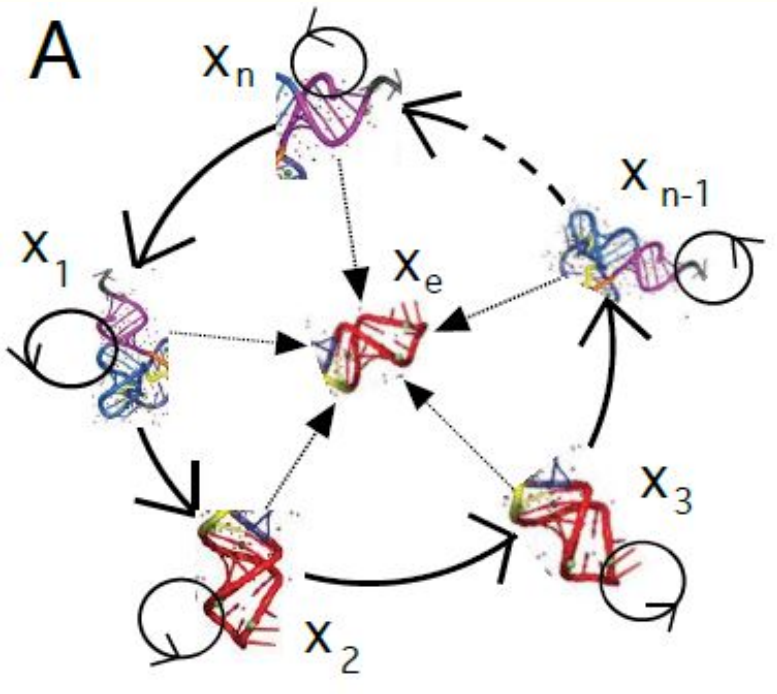
- The **hypercycle**, a possible solution to the Eigen's paradox: would allow that many low-size replicators could co-exist and store a high quantities of information and allow for more complex biological functionality.
- Catalyitc interactions ensure an **all-species coexistence** and the information content can be larger than the one found in a quasispecies (where only competition processes occur)



Hypercycles: the model

$$\dot{x}_i = x_i(A_i Q + \mathbf{K_i} x_{i-1} Q - \phi), \quad 1 \leq i \leq n, \; x_{n+1} = x_1; \qquad (1)$$

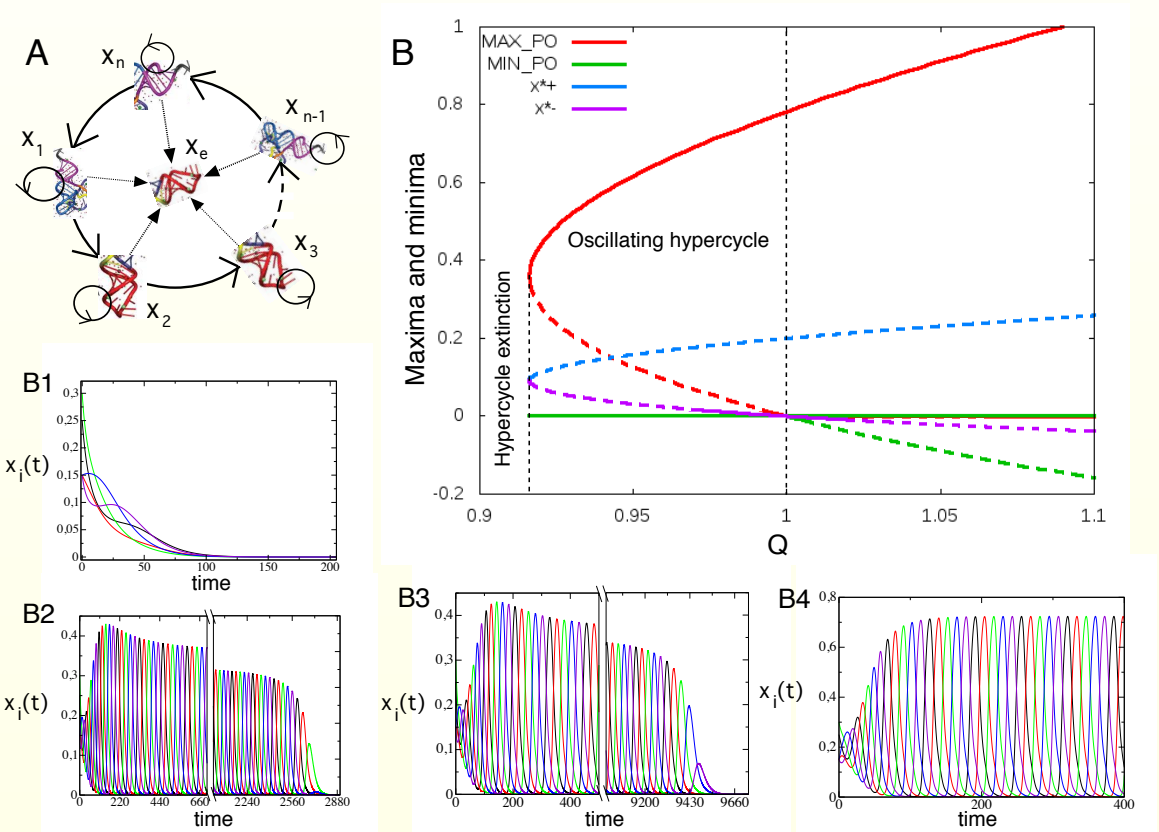
- x_i : population of the functional hypercycle template i;
- A_i: Malthusian (non-catalytic) replication rate of species x_i;
- Q: average copying fidelity during replication ($1 - Q$, average mutation rate);
- K_i: carrying capacity of species x_i;



- $\phi = \sum_{i=1}^{n} x_i(A_i + K_i x_{i-1}) + A_e(1 - \sum_{i=1}^{n} x_i)$ is a dilution outflow, competition between all the hypercycle members and the pool of mutants.

Strong fluctuations in large enough hypercycles: recent results

$$A_i = a,\ K_i = k, \text{ for all } i.$$



6. Extended models in ecology

Population models: general scheme

Endogenous factors:

- Birth and death rates, migrations
- Competition for resources
- Evolution
- Population structure (age, size, sex,...)

Exogenous factors:

- Human action: environmental, species preservation, hunting, sterilisation...
- Animal action: symbiosis, predation,...
- Ecological renewal
- Climate

Stochastic factors

Timing issues: Are the effects instantaneous?

Delay equations

Logistic equation with one delay

$$\frac{dN}{dt} = r\,N(t)\left(1 - \frac{N(t-\tau)}{K}\right)$$

Logistic equation with distributed delays

$$\frac{dN}{dt} = r\,N(t)\left(1 - \frac{1}{K}\int_{-\infty}^{t} w(t-s)N(s)\,ds\right)$$

1D delay equations can have oscillatory solutions

$$\frac{dN}{dt} = \frac{-\pi}{2\,\tau}\,N(t-\tau) \Longrightarrow N(t) = A\cos\left(\frac{\pi\,t}{2\,\tau}\right)$$

Epidemiology

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Population dynamics
Mathematical Methods in Biology, MAMME
March 29th, 2016

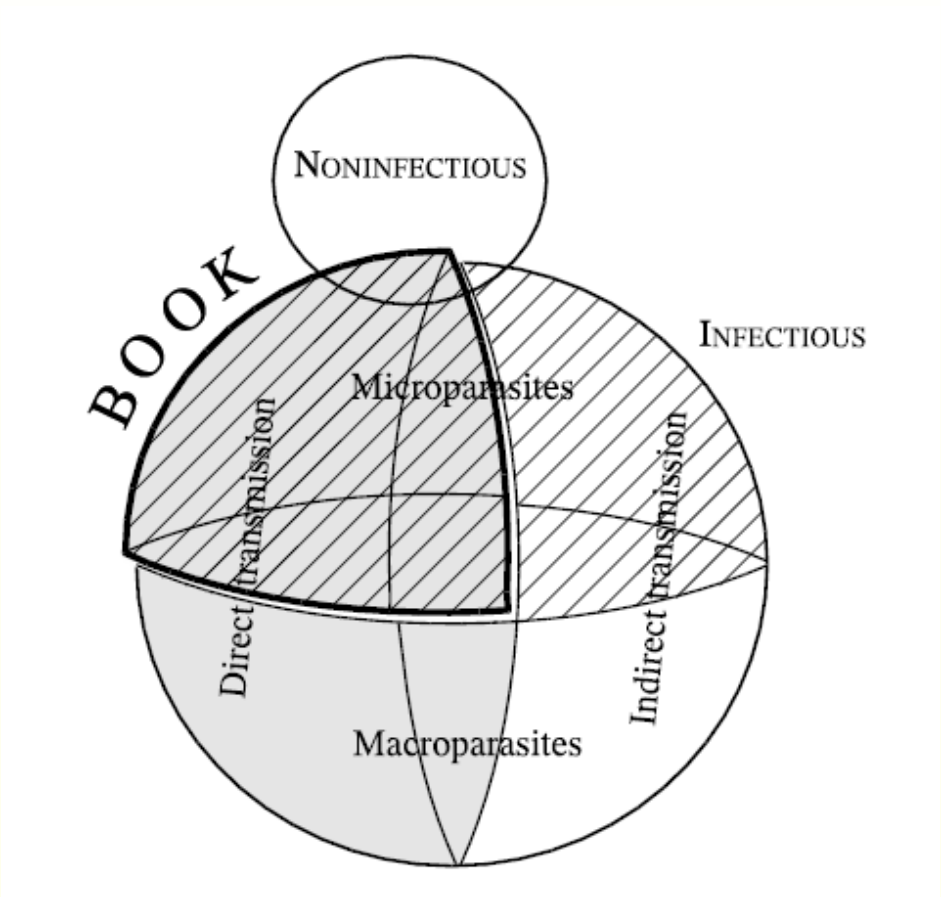
7. Mathematical models of infectious diseases

7.1. Infectious diseases: classification, characterization and control

Classifications of diseases I

Criteria: infection's pathophysiology, clinical symptoms (medical doctors), etiological agents like virus, bacterium, fungus,... and their environment (microbiologists), features that determine patterns of disease and its transmission (epidemiologists).
Infecting pathogens: micro-parasites (generally, single-cell organisms like viruses, bacteria, protoza, prions,...) and macroparasites (with a complex life cycle).
Transmission: Direct (generally microparasites) or indirect (generally macroparasites).
Choice arguments: (1) Microparasites evolve rapidly, thus one can focus on the host's infection status. (2) There are extensive long-term data and a good mechanistic understanding of transmission. (3) 53% of pathogens are microparasites (77% of the most prominent). 61% of microparasites can transmit from animals to humans.

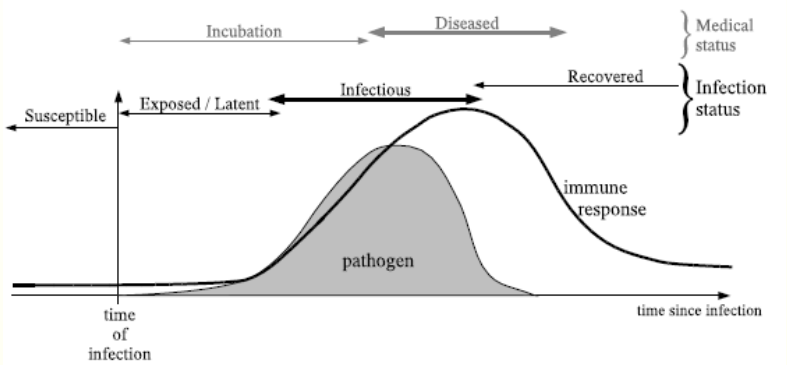
Classifications of diseases II



We will be devoted to **infectious direct transmission microparasitic diseases**.[4]

[4] See mostly [Keeling-Rohani, 2008]

Characterization of diseases



A caricature of the time-line of infection, showing the dynamics of the pathogen (gray area) and the host immune response (black line) as well as labeling the various infection classes: Susceptible, Exposed, Infectious, and Recovered. Note that the diseased period, when symptoms are experienced, is not necessarily correlated with any particular infection class.

Variants of the model: SIR when exposed class is quick enough, SI mostly in plants, that usually remain infectious until death, SIS in sexually transmitted diseases because of vast antigenic variation,...

Control of diseases and the role of mathematical modeling I

- Common **methods**: vaccination (reducing the number of susceptible), Quarantining (reducing the number of infected) and culling (reducing both).
- Models allow us to predict population-level epidemic dynamics from an individual-level knowledge of epidemic factors.
- The sort of models depends on the available data and the required precision.
- They can be more quantitative (specific control policies) or qualitative (understanding dynamics).
- Models help predicting and understanding; in epidemiology, they also help optimizing the use of resources or to shape control measures efficiently.
- Accuracy, transparency and flecibility.

Control of diseases and the role of mathematical modeling II

No model is perfect and no model can accurately predict the detailed outcome of an infection process. But

- a model should be suited to its purpose –that is, it should be as simple as possible, but no simpler– having an appropriate balance of accuracy, transparency, and flexibility. Models designed to help us understand the behavior of an infectious disease versus models built for accurate prediction
- the model should be parameterizable (where necessary) from available data, although parameterization and availability of data are less important in mechanistic models.

Therefore, it is clear that what constitutes a good model is context dependent.

7.1.1. Simple epidemic models: SIR models

Simple epidemic models: SIR models

- Infections that are strongly immunizing or that do not give rise to immunity share that individuals are either Susceptible to infection, currently Infectious, or Recovered (previously infected and consequently immune).
- Typical cases: acute (fast) infections like influenza (*grip*), distemper (*borm, moquillo*), rabies, chickenpox (*varicel·la*), rubella,... with long immunity periods (R) after pathogen infection (I).
- Other factors ignored: heterogeneity in levels of susceptibility, transmissibility or immunological response, contact networks,...

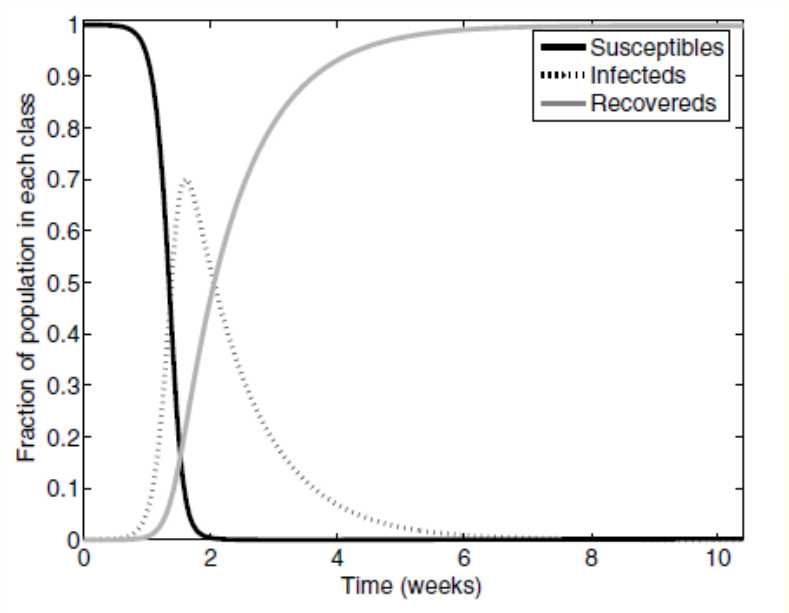
SIR model without demography (births, deaths, migrations, . . .)

- Infection is so quick that demographic changes do not influence.
- I$\rightarrow$ R: infectious period has a distribution with mean $< \tau_{individual} >=: 1/\gamma$, where γ is the **recovery rate**.
- S$\rightarrow$ I: we model the prevalence of infecteds, population contacts and probability of transmission.
  - λ is the force of infection and λX is the rate of production of new infected (X is the cardinal of population S, Y is the cardinal of I).
  - $\lambda = \beta\, Y/N$ (frequency-dependent, more natural in humans) or $\lambda = \beta\, Y$ (density dependent, animals and plants mostly).
  - Defining $S = X/N$ and $I = Y/N$ (proportions), then $\beta\, S\, I$ is the **rate of newly infected** or the **transmission term**.
- We assume a closed population.

SIR model without demography

$$\begin{cases} dS/dt &= -\beta\, S\, I, \\ dI/dt &= \beta\, S\, I - \gamma\, I, \\ dR/dt &= \gamma\, I, \end{cases}$$

with $S + I + R = 1$.

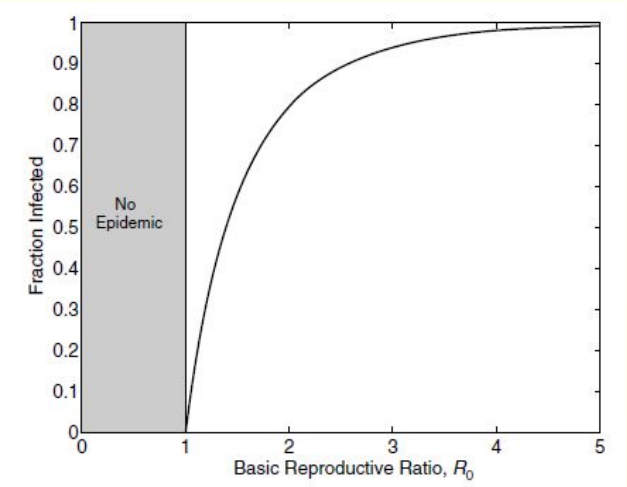


Time-evolution of model, with $S(0) = 1$; $\beta = 520$ per year and $1/\gamma = 7$ days, giving $R_0 = 10$.

SIR model without demography: two interesting phenomena

- The thresholding phenomenon, illustrating the role of the **basic reproductive number,** R_0 (average number of secondary cases arising from an average primary case in an entirely susceptible populations).
- The epidemic burnout: $S(t) = S(0)\exp(-R(t)R_0)$; when $I = 0$ and $t \to +\infty$:

$$1 - R(\infty) - S(0)\exp(-R(\infty)R_0).$$



Some Estimated Basic Reproductive Ratios

Infectious Disease	Host	Estimated R_0	Reference
FIV	Domestic Cats	1.1-1.5	Smith (2001)
Rabies	Dogs (Kenya)	2.44	Kitala et al. (2002)
Phocine Distemper	Seals	2-3	Swinton et al. (1998)
Tuberculosis	Cattle	2.6	Goodchild and Clifton-H. (2001)
Influenza	Humans	3-4	Murray (1989)
Foot-and-Mouth Dis.	Livestock f.(UK)	3.5-4.5	Ferguson et al. (2001b)
Smallpox	Humans	3.5-6	Gani and Leach (2001)
Rubella	Humans (UK)	6-7	Anderson and May (1991)
Chickenpox	Humans (UK)	10-12	Anderson and May (1991)
Measles	Humans (UK)	16-18	Anderson and May (1982)
Whooping Cough	Humans (UK)	16-18	Anderson and May (1982)

7.1.2. The logistic equation in epidemiology

Disease with unprotected hosts

There are several diseases, often caused by bacteria, that do not produce an immune response in the body, e.g., gonorrhea or syphilis.

These diseases, when given treatment leave the host unprotected, so the infected individuals return to the susceptible population.

Dynamics of transmission of gonorrhea: the hypotheses

Dynamics of transmission of gonorrhea in an active sexual population, Hethcote (1976).

- $S(t)$ susceptible population: non-infected sexually active individuals.
- $I(t)$ infected and infectious population.
- $N(t) = S(t) + I(t)$ total population.

- Net income of individuals (all non-infected): $\nu N(t)$, where ν is a growth rate.
- Constant population hypothesis: people leave the active population at a rate νN, and death rate only affects the infected population.
- Homogeneous mixing concerning contacts between individuals.

Dynamics of transmission of gonorrhea: the model

$$S'(t) = -\beta\, S(t)\, \frac{I(t)}{N(t)} + \nu\, I(t) \tag{2}$$

$$I'(t) = \beta\, S(t)\, \frac{I(t)}{N(t)} - \nu\, I(t). \tag{3}$$

where β is the transmission rate and $\beta S \frac{I}{N}$ the number of sexual contacts between Susceptibles and Infected per time unit.
From the constant population hypothesis, we can assume:

$$I'(t) = \beta\, (N - I(t))\, \frac{I(t)}{N} - \nu\, I(t) \tag{4}$$

$$= (\beta - \nu)\, I \left(1 - \frac{\beta}{N(\beta - \nu)}\, I\right). \tag{5}$$

Observe the logistic equation with $r = \beta - \nu$ and $K = \dfrac{N(\beta - \nu)}{\beta}$.

Dynamics of transmission of gonorrhea: conclusions

Defining

$$R_0 = \frac{\beta}{\nu}, \quad K = N\,(1 - 1/R_0)\,, \qquad r = \nu\, R_0\,(1 - 1/R_0)\,.$$

- $\mathbf{R_0 > 1} \Rightarrow r, K > 0$ and $I \to K$ as $t \to +\infty$. $K < N$. Endemic.
- $\mathbf{R_0 < 1} \Rightarrow r < 0, K < 0$ and $I \to 0$ as $t \to +\infty$. Epidemic.

R_0, the basic reproduction number/ratio, is very important in epidemiology. It relates the contact rate, β, to the cure rate, ν. Alternatively, we see that R_0 represents the number of secondary infections caused by a single infected individual, β, during his/her infectious period, $1/\nu$.

$\mathbf{R_0 = 1}(r = 0)$ is a bifurcation value; in fact, it presents a **transcritical bifurcation**.

7.2. Extended epidemiological models

[Keeling-Rohani, 2008]

Variations from the SIR model

- Still simple but different...
  - No immunity, or different immunity (SIS, SIRS)
  - Latent Period (SEIR)
  - Discrete-Time Models
- Host Heterogeneities
  - Risk-structure (e.g., sexually transmitted infections)
  - Age-structure (e.g., childhood infections)
  - Dependence on time since infection
- Multi-pathogen/multi-host Models
- Temporally forced models (seasonality).
- Stochastic dynamics
- Spatial models (heterogeneity, interaction, isolation, localized extinction,...)
  - Metapopulations.
  - Continuous-space continuous-population models (PDEs)
  - Lattice-based models and individual-based models (IBMs)
  - Networks