Contents

Introduction

Mathematical modeling has become an essential element of any study and research in the field of ecology. This course is intended for Master's level students wishing to acquire mathematical modeling techniques in ecology. It presents the foundations of mathematical modeling concerning deterministic dynamic systems, particularly ordinary differential equations and discrete-time models.

Chapter 1

Continuous Systems and Population Dynamics

1.1 Single Population Dynamics Models

In the case of an isolated population, the state variable is the population size $x(t)$ at a time t, but sometimes the variable used is the density of individuals, which represents the number of individuals per unit area.

The general form of the population growth law is as follows:

$$\begin{cases} \frac{dx}{dt} = f(x) \\ x(t_0) = x_0 \end{cases}$$

with the initial condition $x(t_0) = x_0$.

1.1.1 Linear Growth Model (Malthus)

The simplest case is that of a linear model initially proposed by Malthus in 1798, which serves to describe the evolution of a certain quantity over time based on the following assumption: the change (or increase) of this quantity is proportional to itself, and the proportionality coefficient is constant over time. Let n be the birth rate per unit time and per individual, and m be the mortality rate.

n and m are assumed to be constant, leading to the following linear model:

$$\frac{dx}{dt} = nr - mx = rx \tag{1.1}$$

with: $r = n - m$ being the population growth rate.

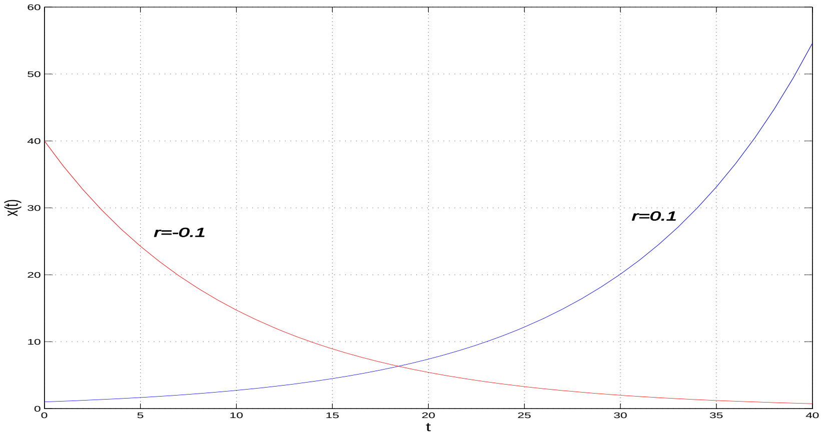
The solution to this equation is as follows:

$x(t) = x_0 \exp(rt)$

The sign of r determines whether the population is growing $(r > 0)$ or going extinct $(r < 0)$

The case $(r = 0)$ corresponds to a population whose size remains almost constant and equal to its initial value.

This model has the advantage of being simple and thus can be solved very easily. Moreover, it models well the beginning of bacterial growth, for example. However, it only describes two types of behaviors: extinction or explosion of populations. In cases where the growth rate is positive, this model proves inadequate for predicting long-term evolution since it assumes infinite resources and that no regulatory phenomenon intervenes.



Indeed, one of the limitations of this model is that it assumes infinite resources, since the population, if viable and not constant, grows infinitely without any regulatory phenomenon intervening. It is to address this divergence problem that Verhulst proposed his logistic model.

1.1.2 Logistic Growth Model (Verhulst)

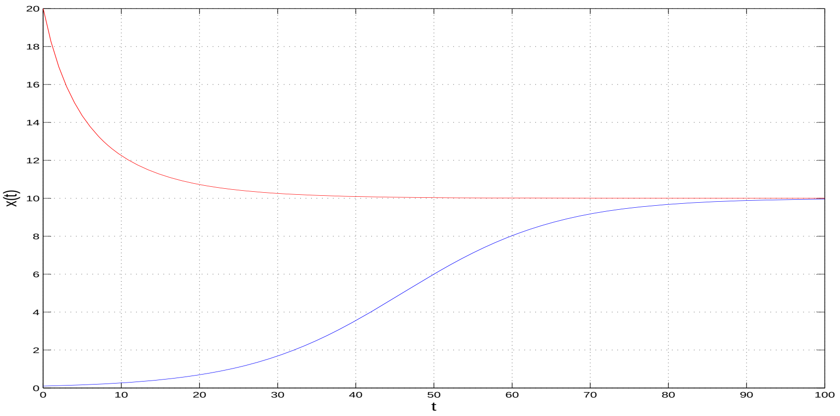
The Verhulst model proposed in 1838, known as the logistic model, is an alternative model to that of Malthus, particularly to introduce a self-regulating process of the observed quantity. In this model, it is assumed that the birth rate is not constant but decreases with the population size.
In the simplest case, we choose for the birth rate a decreasing linear function of the population size: $n(x) = \alpha - \beta x$ where: α and β are positive constants. Similarly, it is reasonable to assume that the mortality rate will, conversely, increase with the population size. For example: $m(x) = \gamma + \delta x$ where: γ and δ are positive constants.
Substituting the birth and mortality rates depending on the population size into the equation leads to the following growth equation:

$$\frac{dx}{dt} = rx(1 - \frac{x}{K}) = f(x) \tag{1.2}$$

with $r = \alpha - \gamma$ being the intrinsic growth rate of the population.
We assume that $\alpha > \gamma$ (i.e., $r > 0$).
K is called the carrying capacity of the environment given by: $K = \frac{\alpha-\gamma}{\beta+\delta} = \frac{r}{\beta+\delta} > 0$ as long as $r > 0$.
This continuous Verhulst model admits a unique maximal solution defined over an interval I,

$$x(t) = K\frac{1}{1 + (\frac{K}{x_0} - 1)e^{-rt}}$$

On the other hand, the equation admits two equilibrium points, the origin and K. We have $f'(x) = r - 2r\frac{x}{K}$. Thus, $f'(0) = r$ and $f'(K) = -r$. Therefore, the origin is unstable and K is a stable equilibrium. For any positive initial condition, we have: $\lim\limits_{t\to+\infty} x(t) = K$.



There is thus self-regulation of the population, whose size (or density or biomass) cannot exceed the carrying capacity of the environment.
When $K \to +\infty$, we recover the Malthus model.

1.2 Two Populations in Interaction

Consider the case of two interacting populations, with sizes $x(t)$ and $y(t)$, the corresponding dynamic model is a system of two coupled ordinary differential equations.
It is customary to decompose each equation into a sum of two terms: the first term corresponds to the growth of the isolated population, and the second term represents the interactions between the two populations:

$$\begin{cases} \dot{x} = f(x) + h(x, y) \\ \dot{y} = g(y) + k(x, y) \end{cases}$$

Where the functions $f(x)$ and $g(y)$ represent the growth of the isolated populations and depend only on the size of that population, while the terms $h(x, y)$ and $k(x, y)$ correspond to the interactions between the two populations.
The signs of the functions $h(x, y)$ and $k(x, y)$ reflect the favorable or unfavorable characters to growth; several cases are possible:

- $(-, -)$: Each population exerts a negative effect on the growth of the other, this is the case of interspecific competition.

- $(+, -)$ or $(-, +)$: One population has a positive effect on the growth of the other, with an inverse effect in the other direction.
  It is, for example, the case of prey-predator relationships or host-parasite.

- $(+, +)$: this is the case of mutualism or symbiosis.

- $(0,+),(+,0),(0,-)$ and $(-,0)$: called commensalism and amensalism.

In the case of predation, it is customary to consider that prey and predators move by exploring their environment at random.
This leads to a "mass action" type interaction where the average number of encounters between two populations is proportional to the product of the sizes.

1.2.1 Conservative Systems, First Integral

Definition 1 (**First Integral**). .
A function $H(x,y)$ *is said to be a first integral of a dynamic system of the form:*

$$\begin{cases} \dot{x} & = & f(x,y) \\ \dot{y} & = & g(x,y) \end{cases}$$

over a domain $\boldsymbol{D}$ *of the plane if* $H(x(t),y(t))$ *is constant for any solution* $(x(t),y(t))$.
When a first integral exists, it is not unique; in fact, $aH+b$ *is also a first integral for any* $a \neq 0$ *and* $b \in \mathbb{R}$. *The fact that the function* $H(x,y)$ *is a first integral implies that it verifies the following relation:*

$$\begin{aligned} \frac{dH(x,y)}{dt} &= \frac{\partial H(x,y)}{\partial x}\frac{dx}{dt} + \frac{\partial H(x,y)}{\partial y}\frac{dy}{dt} &= 0 \\ &= \frac{\partial H(x,y)}{\partial x} f(x,y) + \frac{\partial H(x,y)}{\partial y} g(x,y) &= 0 \end{aligned}$$

Definition 2 (**Conservative System**). .
A dynamic system that admits a first integral is said to be conservative.

Example
Hamiltonian systems defined by:

$$\begin{cases} \dot{x} = \dfrac{\partial H(x,p)}{\partial p} \\ \dot{p} = -\dfrac{\partial H(x,p)}{\partial x} \end{cases}$$

where x is the position of a particle, p its momentum, and $H(x,p)$ its total energy, are conservative systems.
The first integrals are very useful, particularly to demonstrate the existence of centers (as centers are not structurally stable, the Hartman-Grobman theorem does not apply).
To demonstrate the existence of closed trajectories around the equilibrium, one can try to show that the dynamic system is conservative and that its first integral has a local minimum at the equilibrium point; in this case, the trajectories of the system, which are the level curves of the first integral, are obtained by fixing the value of the first integral.

Heres the complete enhanced section for the **Lotka-Volterra Model** formatted in LaTeX:

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1.2.2 Lotka-Volterra Model

This model was proposed independently by the chemist and statistician Lotka in 1920, and the mathematician Volterra in 1926 to explain oscillatory phenomena, such as chemical reactions and fishery dynamics. In this section, we present the derivation of the equations that bear their name, followed by a qualitative study.

The model assumes that:

- The prey population has unlimited food at all times.
- In the absence of predators, the prey population x would increase proportionally to its size:
  $$\dot{x} = f(x) = rx,$$
  where r is the intrinsic growth rate of the prey.
- In the absence of prey, the predator population y would decrease proportionally to its size:
  $$\dot{y} = g(y) = -my,$$
  where m is the mortality rate of the predators.

The coupling of the two populations by predation terms can have stabilizing effects on the dynamics of both populations. In this model, it is assumed that the interaction term is of the classic Lotka-Volterra type. The model is written as follows:

$$\begin{cases} \dot{x} = rx - axy, \\ \dot{y} = -my + eaxy, \end{cases}$$

where a is the predation rate coefficient and e is the efficiency of converting prey into predator offspring.

We can rewrite it by factoring:

$$\begin{cases} \dot{x} = x(r - ay), \\ \dot{y} = y(-m + bx), \end{cases}$$

where $b = ea$.

We note that the axes $x = 0$ and $y = 0$ are zero isoclines (nullclines) of the system. Consequently, no trajectory can cross either the x-axis or the y-axis, thus any trajectory originating from an initial condition in the positive quadrant remains for $t \geq 0$ within this quadrant. We say that the positive quadrant is positively invariant.

The zero isoclines are as follows:

$$\begin{cases} \dot{x} = 0 \Rightarrow x = 0 \text{ or } y = \frac{r}{a}, \\ \dot{y} = 0 \Rightarrow y = 0 \text{ or } x = \frac{m}{b}. \end{cases}$$

It is easy to see that on the axis $x = 0$ for $y > 0$:

$$\dot{y} = -my < 0,$$

indicating that the predator population decreases when there are no prey. On the axis $y = 0$ for $x > 0$:

$$\dot{x} = rx > 0,$$

indicating that the prey population increases when there are no predators.

Similarly, we determine the direction of the vector field on the zero isoclines $x = \frac{m}{b}$ and $y = \frac{r}{a}$ as shown in the figure below.

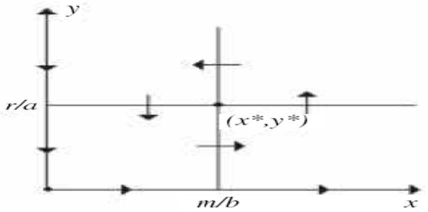


Figure 1.1: Direction field for the Lotka-Volterra model

The Lotka-Volterra model has two equilibrium points: the origin $(0, 0)$ and $(x^\star, y^\star)$ with:

$$\begin{cases} x^\star = \frac{m}{b}, \\ y^\star = \frac{r}{a}. \end{cases}$$

The Jacobian matrix is given by:

$$\mathcal{A}(x, y) = \begin{pmatrix} r - ay & -ax \\ by & -m + bx \end{pmatrix}.$$

Thus, at the equilibrium point $(0, 0)$:

$$\mathcal{A}(0, 0) = \begin{pmatrix} r & 0 \\ 0 & -m \end{pmatrix},$$

indicating that the origin is a saddle point.

At the equilibrium point $(x^\star, y^\star)$:

$$\mathcal{A}(x^\star, y^\star) = \begin{pmatrix} 0 & \frac{-ma}{b} \\ \frac{rb}{a} & 0 \end{pmatrix}.$$

Thus, the trace is zero and the determinant is:

$$\det(\mathcal{A}) = mr > 0.$$

The eigenvalues are $\lambda = \pm i\sqrt{mr}$, indicating that it is a center for the linearized system. However, the Hartman-Grobman theorem does not apply because $(x^\star, y^\star)$ is not hyperbolic.

Next, we seek a first integral that has a local extremum at the point $(x^\star, y^\star)$:

$$\frac{\partial H}{\partial y} = \frac{rx - axy}{-my + bx}.$$

This leads to a separable variable differential equation:

$$-my\,dx + bx\,dx = rx\,dy - axy\,dy.$$

For $xy \neq 0$, we find:

$$\frac{-m}{x}\,dx + b\,dx = \frac{r}{y}\,dy - a\,dy.$$

After integration, we obtain:

$$-m\log x + bx = r\log y - ay + c.$$

Thus, we can express the first integral as:

$$H(x, y) = -r\log y - m\log x + bx + ay,$$

which is a first integral because: $\frac{dH(x,y)}{dt} = 0$.

We have:

$$\frac{dH(x, y)}{dx} = \frac{-m}{x} + b \Rightarrow \frac{dH(x^*, y^*)}{dx} = -b + b = 0,$$

and

$$\frac{dH(x, y)}{dy} = \frac{-r}{y} + a \Rightarrow \frac{dH(x^*, y^*)}{dy} = -a + a = 0.$$

Finally, we find the second partial derivatives:

$$\frac{d^2H(x, y)}{dx^2} = \frac{m}{x^2} > 0 \Rightarrow \frac{d^2H(x^*, y^*)}{dx^2} = \frac{b^2}{m}.$$

This indicates that the first integral $H(x, y)$ has a local minimum at the equilibrium point $(x^\star, y^\star)$, confirming the stability of this equilibrium in the Lotka-Volterra model.

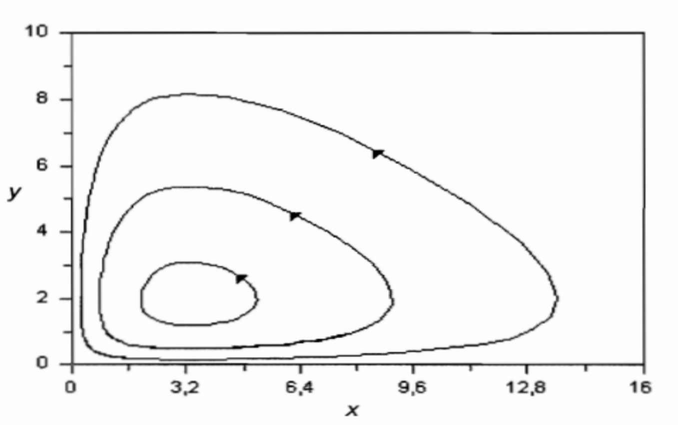


Figure 1.2: Phase portrait of the LotkaVolterra model exhibiting closed trajectories (centers) around the equilibrium point (x, y)

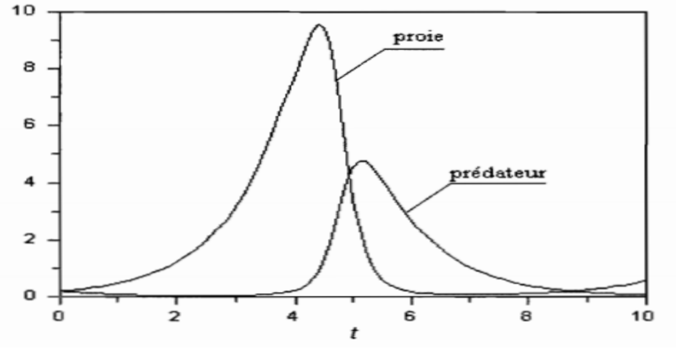
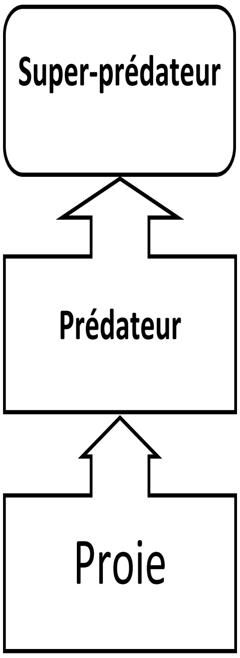


Figure 1.3: Time series of the LotkaVolterra model showing a peak in the prey population followed by a peak in the predator population.

1.3 Community Models

In ecology, a community is a set of organisms belonging to different species populations that form a network of relationships. In this section, we will study the case of a system involving more than two interacting populations. The simplest case involves a prey species, a predator, and a super-predator that feeds on the predator, which is referred to as a three-level trophic chain.

A simple model is based on the Lotka-Volterra equations with type I response functions, assuming that the super-predator follows a logistic growth law:



The system can be described by the following equations:

$$\begin{cases} \dot{x} = x(r - ay) \\ \dot{y} = y(-m + bx - cz) \\ \dot{z} = sz\left(1 - \frac{z}{k}\right) + dyz \end{cases}$$

where: - $x(t)$, $y(t)$, and $z(t)$ are the respective densities of the prey, predator, and super-predator at time t.

By letting $u = x$, $v = y$, $w = \frac{z}{k}$, and $\tau = st$, we obtain the system:

$$\begin{cases} \frac{du}{d\tau} = u(\rho - \alpha v) \\ \frac{dv}{d\tau} = v(-\mu + \beta u - \gamma w) \\ \frac{dw}{d\tau} = w(1-w) + \delta v w \end{cases}$$

with the following relationships: $\rho = \frac{r}{s}$, $\alpha = \frac{a}{s}$, $\mu = \frac{m}{s}$, $\beta = \frac{b}{s}$, $\gamma = \frac{ck}{s}$, and $\delta = \frac{d}{s}$.

The three planes $u = 0$, $v = 0$, and $w = 0$ are zero isoclines; therefore, no trajectory can cross any of these planes, which implies that the strictly positive quadrant is positively invariant under the flow.

The system has two equilibrium points: the origin $(0, 0, 0)$ and the point (u^*, v^*, w^*) with:

$$u^* = \frac{\mu}{\rho} + \frac{\gamma}{\beta}\left(1 + \frac{\delta\rho}{\alpha}\right) > 0,$$

$$v^* = \frac{\rho}{\alpha} > 0,$$

$$w^* = 1 + \frac{\delta\rho}{\alpha} > 0.$$

To study stability, we calculate the Jacobian matrix:

$$\mathcal{A} = \begin{pmatrix} \rho - \alpha v & -\alpha u & 0 \\ \beta v & -\mu + \beta u - \gamma w & -\gamma v \\ 0 & \delta w & 1 - 2w + \delta v \end{pmatrix}$$

and

$$\mathcal{A}(0,0,0) = \begin{pmatrix} \rho & 0 & 0 \\ 0 & -\mu & 0 \\ 0 & 0 & 1 \end{pmatrix}.$$

When $\rho > 0$ and $\mu > 0$, the origin is a saddle point.

For the equilibrium point (u^*, v^*, w^*):

$$\mathcal{A}(u^*, v^*, w^*) = \begin{pmatrix} 0 & -\alpha u^* & 0 \\ \beta v^* & 0 & -\gamma v^* \\ 0 & \delta w^* & -w^* \end{pmatrix}.$$

The characteristic equation is given by:

$$\lambda^3 + w^*\lambda^2 + (\alpha\beta u^* v^* + \alpha\delta v^* w^*)\lambda + \alpha\beta u^* v^* w^* = 0.$$

To study the sign of the eigenvalues without calculating them, we apply the Routh-Hurwitz criterion.

Routh-Hurwitz Criterion

Consider the linear differential system $\dot{x}_i = \sum_{j=1}^{n} a_{ij} x_j; i = 1, n^-$. Let $\det A = [a_{ij}]$ be a square matrix with constant coefficients and $\det(A) \neq 0$. The characteristic equation can be written as:

$$\lambda^n + a_1\lambda^{n-1} + \cdots + a_{n-1}\lambda + a_n = 0.$$

The Hurwitz determinants are given by:

$$H_1 = a_1,$$

$$H_2 = \begin{vmatrix} a_1 & a_3 \\ 1 & a_2 \end{vmatrix},$$

$$H_3 = \begin{vmatrix} a_1 & a_3 & a_5 \\ 1 & a_2 & a_4 \\ 0 & a_1 & a_3 \end{vmatrix},$$

$$H_k = \begin{vmatrix} a_1 & a_3 & a_5 & \cdots & a_{k-1} \\ a_1 a_2 & a_4 & \cdots & a_k & \\ a_1 & a_3 & \cdots & a_{k-1} & \\ 0 & 1 & a_2 & \cdots & a_k \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & \cdots & \cdots & \cdots & a_k \end{vmatrix}.$$

In the case of dimension n, all a_j with $j > n$ are assumed to be equal to zero. The equilibrium point 0 is asymptotically stable if and only if: $\forall k = (1, n)^-; H_k > 0$ (the real parts of all eigenvalues are negative).

In our model, we have:

$$a_1 = w^* > 0,$$

$$a_2 = \alpha\beta u^* v^* + \alpha\delta v^* w^* > 0,$$

$$a_3 = \alpha\beta u^* v^* w^* > 0.$$

Thus, we have:

$$H_1 = a_1 > 0,$$

$$H_2 = a_1 a_2 - a_3 = \alpha\delta v^* w^* > 0,$$

$$H_3 = a_3 H_2 > 0.$$

According to the Routh-Hurwitz criterion, (u^*, v^*, w^*) is asymptotically stable. “‘

Chapter 2

Discrete Systems and Population Dynamics

The general form of a discrete system is given by $x_{n+1} = f(x_n); x_n \in \mathbb{R}^n$.

2.1 Fixed Point

Definition 3. *A fixed point of a discrete system, denoted as x^*, satisfies the condition: $x^* = f(x^*)$.*

2.2 Local Stability of a Fixed Point

We define a local variable $u_n = x_n - x^*$ and proceed with linearization (in the case $n = 1$). By performing a Taylor expansion of the function f around x^*, the linearized local model is expressed as:

$$u_{n+1} = \lambda u_n$$

where $\lambda = \frac{df(x^*)}{dx_n}$. The fixed point for this system is the origin. The solution of the linearized system can be written as:

$$u_n = \lambda^n u_0$$

where u_0 is the initial condition.

Several cases are possible:

1. If $\lambda < -1$:
$$u_n = (-1)^n |\lambda|^n u_0$$
The solution diverges from the fixed point, taking on alternating signs.

2. If $\lambda = -1$:
$$u_n = (-1)^n u_0$$
Thus, the solution alternates between u_0 and $-u_0$.

3. If $-1 < \lambda < 0$:
$$u_n = (-1)^n |\lambda|^n u_0$$
The solution alternates in sign and approaches the fixed point.

4. If $\lambda = 0$: From the first iteration, we move to the fixed point 0.

5. If $0 < \lambda < 1$:
$$u_n = \lambda u_0$$
The solution converges to 0.
6. If $\lambda = 1$:
$$u_n = u_0.$$
7. If $\lambda > 1$:
$$u_n = \lambda u_0.$$

2.3 Application to Population Dynamics

2.3.1 Dynamics of a Single Population

a) **Verhulst Model:**

The Verhulst equation in discrete time is expressed as:

$$x_{n+1} = \rho \frac{x_n}{x_{n+k}} = f(x_n)$$

where ρ is the growth rate of the population, k is a positive parameter, and x_n is the population size at iteration n.

The fixed points of this equation are solutions to the equation:

$$f(x) = x \Rightarrow \rho x = x(x+k) \Rightarrow x = 0 \text{ or } x = \rho - k.$$

To analyze the stability of the fixed points, we compute:

$$f'(x) = \frac{\rho(x+k) - \rho x}{(x+k)^2} = \frac{\rho k}{(x+k)^2}.$$

Thus:

$$f'(0) = \frac{\rho}{k}.$$

1) If $\frac{|\rho|}{k} < 1 \Rightarrow |\rho| < k$, then $x = 0$ is asymptotically stable.
2) If $|\rho| > k$, then $x = 0$ is unstable.

For the fixed point $x = \rho - k$:

$$f'(\rho - k) = \frac{\rho k}{(\rho - k + k)^2} = \frac{k}{\rho}.$$

1) If $\frac{k}{|\rho|} < 1$, we have $k < |\rho|$, thus $x = \rho - k$ is asymptotically stable.
2) If $k > |\rho|$, then $x = \rho - k$ is unstable.

Chapter 3

Disease Transmission Models

One of the main concerns of epidemiology is to understand the dynamics governing the spread of infectious diseases in order to establish prevention and intervention strategies that can reduce their impact on public health.

3.1 First Mathematical Model in Epidemiology (Daniel Bernoulli)

On April 16, 1760, the Royal Academy of Sciences in Paris presented a public reading of a work by Daniel Bernoulli, in which he provided a new analysis of mortality caused by smallpox and justified inoculation as a preventive measure. His model was the first mathematical model in epidemiology, giving rise to what is now called "biomathematics."

The following hypotheses were adopted:

- An individual infected for the first time by smallpox has a probability p of dying and a probability $1 - p$ of surviving, independently of their age;
- An individual has a probability q of being infected in a year, also independently of their age (i.e., the probability that an individual is infected during the small time interval dt between age t and age $t + dt$ is $q \cdot dt$);
- When an individual survives after being infected by smallpox, they are immunized for the rest of their life.

Let $m(t)$ denote the natural mortality at age t. The probability that an individual dies in a small time interval dt between age t and age $t + dt$ is $m(t) \cdot dt$. Considering a group of P_0 individuals born in the same year, let us define:

- $S(t)$: the number of individuals still alive at age t who have not been infected (and are therefore still susceptible);
- $R(t)$: the number of individuals still alive at age t who are immunized;
- $P(t) = S(t) + R(t)$: the total number of individuals still alive at age t.

Between age t and age $t + dt$ (with dt infinitesimally small), each individual who has never been infected has a probability $q \cdot dt$ of contracting smallpox and a probability $m(t) \cdot dt$ of dying from another cause. Thus, the change in the number of uninfected individuals is given by:

$$dS(t) = -qS(t) \cdot dt - m(t)S(t) \cdot dt,$$

leading to the differential equation:

$$\frac{dS(t)}{dt} = -qS(t) - m(t)S(t). \tag{3.1}$$

During the same small time interval, the number of individuals dying from smallpox is $pqS(t) \cdot dt$, and the number of individuals who survive and become immunized is $q(1-p)S(t) \cdot dt$. Additionally, $m(t)R(t)$ individuals who are already immunized die naturally, leading to a second differential equation:

$$\frac{dR}{dt} = q(1-p)S(t) - m(t)R(t). \tag{3.2}$$

By summing the two equations, we obtain:

$$\frac{dP}{dt} = -pqS - m(t)P(t). \tag{3.3}$$

From equations (3.1) and (3.3), it can be shown (as an exercise) that the fraction of individuals who are still susceptible to contracting smallpox at age t is:

$$\frac{S(t)}{P(t)} = \frac{1}{(1-p)e^{qt} + p}. \tag{3.4}$$

To illustrate his theory, Bernoulli used Halley's mortality table (1656-1742), constructed for the city of Breslau (1693). This table lists the number of survivors at the beginning of year t ($t = 1, 2, \ldots$) from a cohort of 1300 individuals born in year 0. The parameter values are:

- Probability of dying from smallpox: $p = \frac{1}{8}$;
- Annual probability of contracting smallpox: $q = \frac{1}{8}$.

This probability is estimated based on observations made in several cities across Europe. Using formula (3.4) and the values of $P(t)$ provided in the table, one can calculate the number of individuals $S(t)$ still alive at age t who have not been infected. One then determines the number of individuals still alive at age t who have had smallpox and survived: $R(t) = P(t) - S(t)$. Finally, for each age t, the number of deaths due to smallpox between age t and age $t+1$ is determined, given by the integral approximated by the trapezoidal rule:

$$pq \int_t^{t+1} S(t)\, dt \approx pq \frac{(S(t) + S(t+1))}{2}.$$

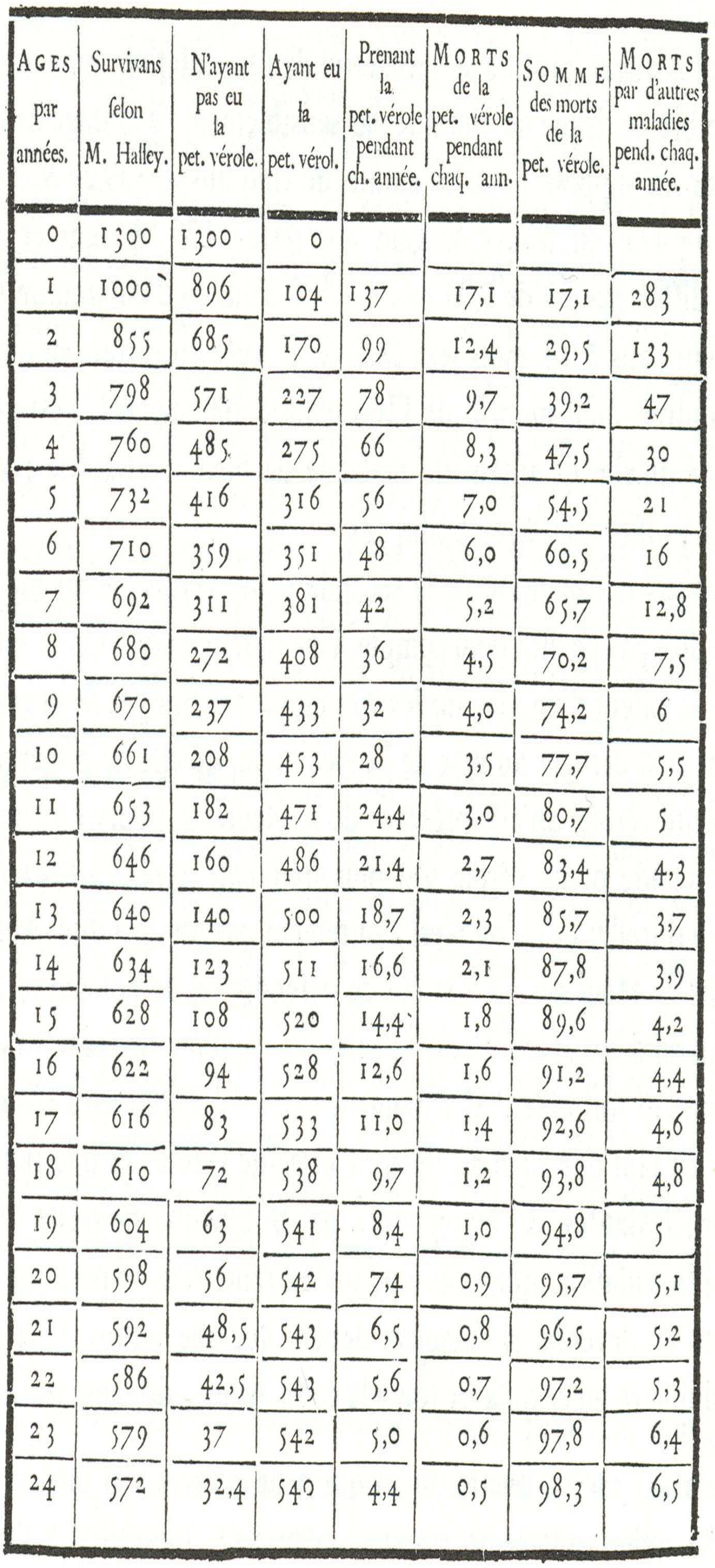
Bernoulli deduced that out of the 1300 newborns, 101 were destined to die from smallpox, which is nearly the expected fraction of $\frac{1}{13}$.

He then considered the situation where smallpox would be inoculated to the entire population at birth and would cause no deaths. Smallpox would be eradicated, and the question arose about estimating the increase in life expectancy. Starting with the same number P_0 at birth and letting $P^*(t)$ denote the number of individuals aged t when smallpox has disappeared, we have:

$$\frac{dP^*}{dt} = -m(t)P^*.$$

Using equation (3.3), we find:

$$P^*(t) = \frac{P(t)}{1 - p + pe^{-qt}}.$$



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4	760	485	275	66	8,3	47,5	30
5	732	416	316	56	7,0	54,5	21
6	710	359	351	48	6,0	60,5	16
7	692	311	381	42	5,2	65,7	12,8
8	680	272	408	36	4,5	70,2	7,5
9	670	237	433	32	4,0	74,2	6
10	661	208	453	28	3,5	77,7	5,5
11	653	182	471	24,4	3,0	80,7	5
12	646	160	486	21,4	2,7	83,4	4,3
13	640	140	500	18,7	2,3	85,7	3,7
14	634	123	511	16,6	2,1	87,8	3,9
15	628	108	520	14,4	1,8	89,6	4,2
16	622	94	528	12,6	1,6	91,2	4,4
17	616	83	533	11,0	1,4	92,6	4,6
18	610	72	538	9,7	1,2	93,8	4,8
19	604	63	541	8,4	1,0	94,8	5
20	598	56	542	7,4	0,9	95,7	5,1
21	592	48,5	543	6,5	0,8	96,5	5,2
22	586	42,5	543	5,6	0,7	97,2	5,3
23	579	37	542	5,0	0,6	97,8	6,4
24	572	32,4	540	4,4	0,5	98,3	6,5

From this relationship, we deduce that the ratio $\frac{P(t)}{P^*(t)}$ tends towards $1-p$ as t becomes sufficiently large. To compare $P(t)$ and $P^*(t)$, we estimate the life expectancy at birth with smallpox:

$$E(P) = \frac{1}{P_0}\int_0^{\infty} P(t)\,dt,$$

and without smallpox, we have a similar expression by replacing P with P^*. Bernoulli again used the trapezoidal rule to approximate the integral:

$$E(P) \approx \frac{(P_0/2 + P(1) + P(2) + \ldots)}{P_0}.$$

Continuing the table beyond 24 years up to 84 years, he ultimately obtained a life expectancy E with smallpox equal to:

$$\frac{1300/2 + 1000 + \ldots + 20}{1300} \approx 26.57 \text{ years, or 26 years and 7 months.}$$

Without smallpox, he obtained a life expectancy E^* equal to:

$$\frac{1300/2 + 1015 + \ldots + 23}{1300} \approx 29.65 \text{ years, or 29 years and 8 months.}$$

Of course, inoculating a less virulent strain of smallpox is not completely safe. If p' is the probability of dying from smallpox immediately after inoculation ($p' < p$), then the life expectancy would be $(1-p')E^*$ if everyone underwent inoculation at birth. This life expectancy remains higher than the natural life expectancy E if $p' < 1 - \frac{E}{E^*}$, or approximately 11. Data regarding p' was difficult to obtain at the time. However, Bernoulli estimated that the risk p' was less than 1. For him, there was no doubt: inoculation should be promoted by the state.

3.2 Simple Deterministic Model by Hamer (SI Model)

W. H. Hamer proposed a simple epidemic model in 1906, in which he assumed that the considered population N is divided into two categories:

- Susceptible individuals (S);
- Infected individuals (I).

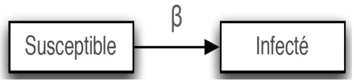
Infection spreads through direct contact between a susceptible S and an infected I individual. Thus, the number of new cases increases with the number of susceptible S and infected I individuals, with a proportionality factor α (also called the infection rate). An individual, once infected, becomes infectious and remains so for the rest of their life. This assumption is reasonable for many diseases in the early stages of infection. We assume that the population is closed, i.e., for all $t \in \mathbb{R}^+$:

$$S + I = N,$$

where N is constant and corresponds to the total population size.

The Hamer model is then described by the following differential system:

$$\begin{cases} \dot{S} = -f(S, I) \\ \dot{I} = f(S, I) \end{cases}$$



where f is an increasing function of S and I that expresses the rate at which infection occurs. The simplest case is when:

$$f(S, I) = \beta S I,$$

in which β expresses that not all possible contacts necessarily occur, and those that do not always result in a new case (a contact does not necessarily lead to contamination). The following diagram illustrates the disease transmission model.

Thus, the model can be written as:

$$\begin{cases} \dot{S} = -\beta S I \\ \dot{I} = \beta S I \end{cases}$$

And since $S + I = N$, we obtain:

$$\begin{cases} \dot{S} = -\beta S(N - S) \\ \dot{I} = \beta I(N - I) \end{cases}$$

These are logistic-type differential equations, initially introduced by Verhulst to describe a population growth model. By integrating, we obtain:

$$I = \frac{N}{1 + (N - 1)e^{-N\beta t}}, \quad I(0) = 1.$$

This model is overly simplistic and does not accurately represent reality in epidemiology.

3.3 General Deterministic Model by Kermack and McKendrick (SIR Model)

The SIR model by Kermack-McKendrick (1927) describes the evolution of an epidemic in a population divided into three categories:

The susceptible individuals S, infected individuals I, and individuals who can no longer transmit the disease or are resistant (due to recovery, immunity, or death), denoted as R. The simple system can be expressed as:

$$\begin{cases} \dot{S} = -\beta S I \\ \dot{I} = \beta S I - \gamma I \\ \dot{R} = \gamma I \end{cases}$$

The parameter γ can be zero, reducing the case to the simple Hamer model. It can also be equal to 1, in which case all infected individuals exit the process during the time interval dt. Generally, the exit rate γ takes an intermediate value. Studying this model allows us to establish the "threshold theorem." The system is equipped with the initial condition:

$$S(0) = S_0, \quad I(0) = I_0, \quad R(0) = 0,$$

with $I_0 \approx 0$ and $S_0 \approx N$. This corresponds to considering a population of susceptibles into which a small number of infectious individuals are introduced. It can be shown that the positive quadrant is positively invariant.

Since $N = S + I + R =$ constant, the system reduces to:

$$\begin{cases} \dot{S} = -\beta S I \\ \dot{I} = \beta S I - \gamma I \end{cases}$$

and $R = N - S - I$.

By dividing the second equation of the system by γI, we obtain:

$$\frac{I'(t)}{\gamma I} = \frac{\beta S(t)}{\gamma} - 1.$$

Thus, for $\frac{\beta S(t)}{\gamma} > 1$, each infected individual will contaminate more than one susceptible individual, and the disease will spread to an ever-increasing number of individuals. This will continue until the number of susceptibles $S(t)$ is such that $\frac{\beta S(t)}{\gamma} > 1$. The ratio $\frac{\beta}{\gamma}$ can then be interpreted as the number of contacts capable of transmitting the disease by infected individuals throughout their contagious period. Therefore, by multiplying by the fraction of susceptible individuals at any moment, we obtain the number of new infections caused by a single contagious individual.

The last equation highlights the importance of initial conditions in such models because if $\frac{\beta S_0}{\gamma} > 1$, there will necessarily be an epidemic, whereas if $\frac{\beta S_0}{\gamma} < 1$, only a few individuals will be infected before the disease propagation stops on its own.

Let $R_0 = \frac{\beta N}{\gamma}$. This threshold R_0 is called the basic reproduction number and corresponds to the number of secondary cases produced by an average infectious individual during their infectious period in a population entirely composed of susceptibles. In the case where $R_0 > 1$, although the system cannot be solved explicitly, some properties can be demonstrated.