

Lecture Notes for the course Biomathematics 1
First Part: Population Dynamics
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This file is the first version of my lecture notes in English. They are based on material appeared in the book “Modelli Matematici in Biologia” (Springer) and on supplementary notes distributed to students. Both of these older sources were in Italian, and correspondingly several of the figures appearing here are still with some Italian words appearing in them.

Needless to say, the file is surely plagued with errors and mistakes; I will be grateful to those willing to point these out to me.

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Chapter 1

Introduction.

A trivial model for AIDS, and its use

Developing mathematical models for biological phenomena is surely going to require a substantial effort. Before embarking in such a task, one should thus ask “is this really worth it?”. From the point of view of a biologist, the question is actually “is it going to give some really new understanding?”.

Unfortunately, apart from some general abstract considerations and hand-waving, the only way to answer such a question is to actually analyzing some models and attempting to extract from these some *useful* information; here “useful” is referred to the biologist, not just to the mathematician seeking a new application for his/her theorems.

I would thus start by providing an example of a mathematical model which on the one hand is extremely simple (maybe it would be more appropriate to describe it as trivial); but on the other hand played a substantial role in the development of an effective treatment of AIDS. In fact, it helped to understand that the ideas generally accepted at the time about the development of the disease were wrong, and not just marginally.

I hope this model can motivate the reader, and in particular the student, about the potential usefulness of mathematical models; if not in full, at least enough to follow the pages of the book (and, for the student, the hours of the teaching course) in the hope it will contain something useful also for the understanding of Biology.

1.1 A short description of AIDS mechanism

The HIV (*Human Immunodeficiency Virus*) virus leads to AIDS (*Acquired ImmunoDeficiency Syndrome*, also known as “SIDA” in French and Spanish speak-

ing countries). Actually, HIV is a retrovirus, and hence replicates only in cells which are undergoing the subdivision process; we will however speak of “virus” for ease of language.

The growth rate of HIV *in vivo* is quite high, but despite this the immune system can, in favorable cases, halt virus progression for a very long period, even 5 or 10 years – but it cannot eliminate the infection. In other cases death can occur a few months after infection.¹

The HIV virus targets mainly a class of lymphocytes, i.e. the CD4 T-cells, which are essential for the functioning of the immune system; it also affects other types of cells. When the concentration of CD4 cells, which is in normal conditions around $1000/\mu\ell$, decreases to $200/\mu\ell$, the subject is considered as an AIDS case.²

The mechanism leading to the rarefaction of CD4 cells is not understood in detail and surely was not understood at all in the early times of the AIDS epidemic. The lymphocytes renewal is usually quite fast; thus *a priori* the infection could act on this either by affecting the source of these cells, either reducing the average cell life (or both).

When AIDS appeared, in a first time one thought that the long time between infection by HIV and the actual appearance of AIDS was a virus latency time; moreover, as the infection developed in such a slow way, one thought that all the intervening mechanisms were equally slow.

Actually things are not this way. Nowadays we know that in the growth of HIV infection there is a huge variety of time scales (i.e. the mechanisms at work have different time scales): minutes, hours, days, months, years, decades.

Understanding the infection *dynamics* has radically changed the way patients are treated, and led to much longer survival times.

We will now briefly discuss what has been the first step in the understanding of the infection dynamics.

1.2 The Ho model

It is rather clear that understanding what exactly is going on in the pseudo-latency period is essential in order to design an effective treatment for HIV infection.

In this period, the concentration of both viral particles (virions) and antibodies is essentially constant, while there is a slow diminishing of the concentration of CD4 cells

¹In 1997, the peak year, according to the UN AIDS program (UNAIDS; see <https://www.unaids.org/en>), in Africa half of the new born babies were infected with AIDS; in Zimbabwe 20% of adults were AIDS positive, and this fraction reached 35% in Botswana. As for 2020 data, the estimate is there are about 38 million people living with AIDS (25 millions of these in Africa), with about 27 million having access to antiretroviral therapy; the new infections and deaths in the year have been 1.5 million and 700,000 respectively.

²We are of course oversimplifying a quite more involved matter: one uses of course also other concurrent indicators and criteria.

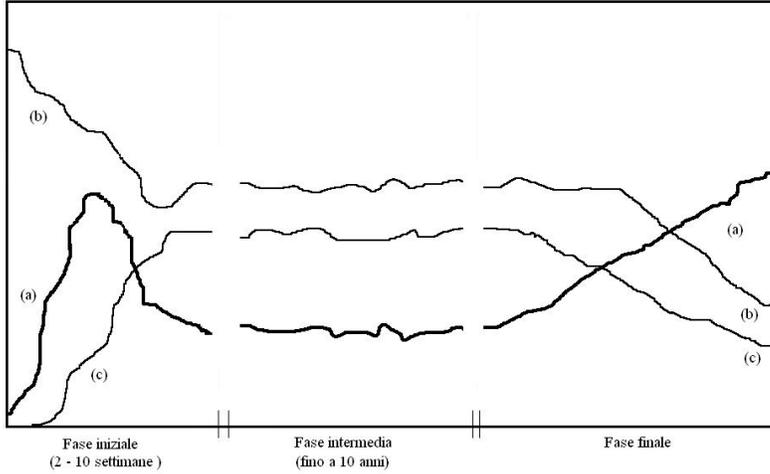


Figure 1.1: Typical behavior in time for the concentration of virions (a), of T-CD4 lymphocytes (b), and HIV antibodies (c) in an affected individual. The figure depicts, with widely different time scales, the initial phase, in which there are rapid changes ignited by the invasion of the virus in the body; the intermediate phase in which there is a substantial equilibrium; and the final phase where the immune systems collapses and the virus has free way.

Thus the first – and maybe most natural – idea was that the virus would be in a latency state; this is what happens e.g. for the *herpes* virus in between two acute phases. Treatment was thus designed under this assumption, to deal with a hiding enemy.

A way to understand if the virus is active or not active is to perturb its activity in a period in which no symptoms of the infection are active. In 1994, the group of D. Ho at Columbia University in New York experimented with administering an anti-viral (*ritonavir*) to 20 patients, obtaining excellent results; in Figure 1 the data for two of these patients are reproduced.³

In order to analyze the experimental data, and above all understand what they tell us about the virus replication speed, it is necessary to have a model. Ho and his collaborators used a very simple one.

Let us call $V(t)$ the virus population (the number of virions) at time t . Its change in time originates from a source of viral peptides, and contrasted by various elimination processes: these include the action of the immune system, but also the death of infected cells. We denote by P the number of virions produced per unit of time, and by c the virus elimination rate, i.e. the fraction

³The data are reproduced from the original paper: D. Ho et al., *Nature* **373** (1995), 123-126. The mathematical and modelling treatment of this experiment, as well as other AIDS models, are discussed in A.S. Perelson and P.W. Nelson, “Mathematical models of HIV-1 dynamics in vivo”, *SIAM Rev.* **41** (1999), 3-44. By the way, this work is based on the Ph.D. thesis of P.W. Nelson (defended in Seattle in 1998).

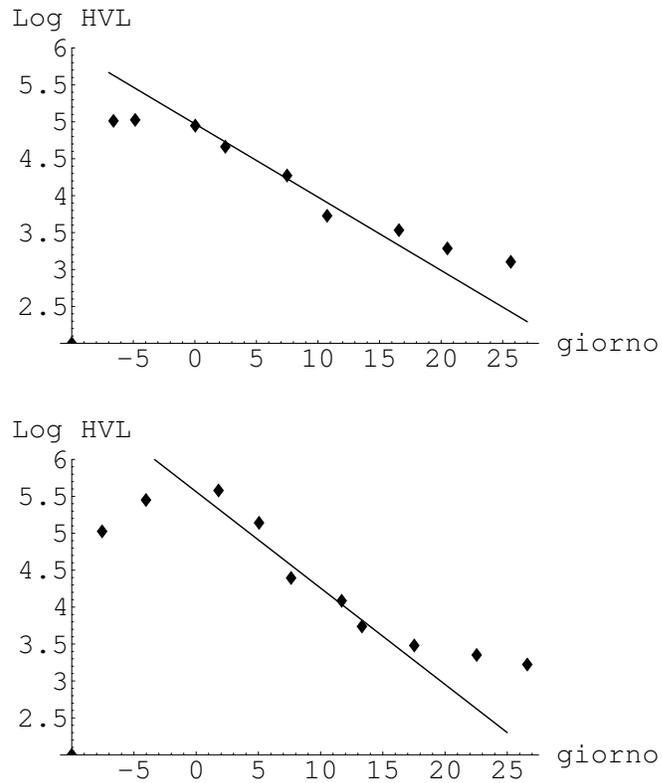


Figure 1.2: Variation of the concentration of virions $V(t)$ with treatment by protease inhibitor (the graph is in semi-logarithmic scale, so it shows $\log[V(t)]$ against t) for two different patients in the Ho experiment; time is measured in days from the beginning of treatment. The solid straight line represents a fit by exponential decrease.

of existing virions which are eliminated again in a unit of time.

Note that in this description we are implicitly assuming that this rate c is independent of the level of the total viral population $V(t)$. Actually, when one looks better at things, this assumption is not unreasonable: in the pseudo-latency period the viral population is essentially constant, and as our model will consider this situation, it suffices that c is constant for V nearly constant, i.e. that c varies slowly with V .

Similarly, the number P of new virions per unit of time will of course depend on $V(t)$; but with the same reasoning we expect we do not need to worry about this dependency and its detailed form.

We will thus write

$$\frac{dV}{dt} = P - cV . \quad (1.1)$$

This is a first order ODE, and we know its general solution is just

$$V(t) = (P/c) + e^{-c(t-t_0)} V_0 ,$$

where of course V_0 is the population at the initial time $t = t_0$; but actually we do not even need to solve the equation.

We have seen that the pseudo-latency phase corresponds to an equilibrium, and at equilibrium by definition $dV/dt = 0$; thus, denoting by V_e the viral population at equilibrium,

$$V_e = P/c . \quad (1.2)$$

On the other hand, in the Ho experiment protease is inhibited. Let us assume that the inhibition is complete, i.e. the drug is 100% effective, and it is administered starting from time $t = 0$. Then, for $t > 0$ we will have $P = 0$. Thus the equation describing $V(t)$ *under treatment* is even simpler than (1): for $t > 0$ we just have

$$\frac{dV}{dt} = -cV ; \quad (1.3)$$

its solution is just

$$V(t) = V_e e^{-ct} . \quad (1.4)$$

Note that we have used the information that at the beginning of the treatment (thus at $t = 0$, see above) we are at equilibrium, hence $V(0) = V_e$.

As $V(t)$ is measured in the experiment, we can use (1.3) to translate the measurement of $V(t)$ into a measurement of the constant c ; if t_* is the halving time for $V(t)$, then $c = [\log(2)]/t_*$. The straight lines in the graph of Figure 1 are just fits of experimental data with function of type (1.4).

The value of c will depend on many factors, including activity of the immune system and life span of infected cells; thus we expect it to be different for different patients, and this is indeed the case – as shown indeed in the different graphs of Figure 1.1. We will thus have to consider the average for different patients, and the variance (or the standard deviation) of these values around the average.⁴

⁴We assume the reader to be familiar with these concepts, which are introduced in the Probability and/or Statistics courses.

In the Ho experiment , the data provided

$$t_* = 2.1 \pm 0.4 \text{ days ;}$$

this corresponds for c to the value

$$c = 0.33 \pm 0.06 \text{ (day)}^{-1} .$$

We can finally evaluate P in the pseudo-latency phase. Recalling that before the treatment $V = V_e$ and using (1.2), we get

$$P = c V_e . \tag{1.5}$$

Now, let us go back once again to Figure 1.1, and look at the vertical axis, corresponding to the number of virions per mL . We realize that V_e is of the order of $10^6 - 10^7$ per cm^3 ; this also entails that

$$P \simeq (1/3) \cdot (10^6 - 10^7) \text{ cells}/(\text{m}\ell \cdot \text{day}) .$$

That is, the virus is not at all at sleep, but it is reproducing at a rate of about one million cells per milliliter per day!

This discovery has completely changed our understanding of the mechanisms of HIV infection, and with it also the therapies used to increase the life expectancy of patients – which have indeed dramatically increased since.

I would like to stop here my discussion of AIDS and related models⁵, and present instead some considerations both on this specific model and on models in general.

1.3 Some considerations

The first thing which needs to be said is that the model we have discussed is not at all realistic. In fact, we made some assumptions which are definitely unreasonable , and actually rather absurd:

- We assumed that the protease inhibitor had a 100% efficacy, which is obviously impossible;
- We assumed that in the pseudo-latency phase the number of virions was exactly constant, which is both false and absurd;
- We also assumed, again in the pseudo-latency phase, a virus production exactly constant in time;
- We assumed that the virus removal was exactly proportional to the number of viral particles, moreover disregarding any dependence on or interaction with the environment in which the infected cells and the immune system evolve;

⁵The interested reader can consult for this the text by J.D. Murray, *Mathematical Biology. I: An Introduction*, Springer (Berlin) 2002; see in particular chapter X therein.

- More generally, we disregarded any interaction with the external world – which in this context means also the non-infected cells present in the host organism.

Had we considered these weaknesses of the model before using it to analyze the data, we would most probably have decided it was so far from any realistic, or even just sensible, description that it was completely useless to work on it.

On the other hand, as you surely understand, the strength of the model lies precisely in its utter simplicity: we want to evaluate P , and we have disregarded whatever inessential detail, including facts that it is difficult to consider as “details”.

This is exactly what a model should do: analyze a given phenomenon or process understanding what are the essential mechanisms for that phenomenon or process; the success of the model shows that we looked at the right objects, i.e. at the “true protagonists” of the process.

Needless to say, once we are (reasonably) sure to be on the right track, we can always (and often we must) attempt at a refinement of the model inserting other details and trying to have a better agreement with the experimental findings.

Another lesson to be taken from this example is that sometimes one should not be too picky: if you look once again at Figure 1, you see that in order to read in the sequence of raw experimental data an exponential behavior (a straight line in the semi-logarithmic scale used there) one needs a substantial dose of optimism – and maybe of detachment from reality.

Actually, when one considers all the brutal approximations we have introduced (those in the list above, and not only), one should be surprised if the model was describing the experimental data in an effective way!

The same remark about the need to be not too demanding applies to the measurement that the model allows to extract from experimental data: our relative error on t_* was around 20%, and that on P is even greater (this is affected also by the error on the value of V_e); but despite this the increment of our knowledge about P was huge: we passed from the naive estimate $P \simeq 0$ to $P \simeq 10^6$!

This is the fundamental criterion to decide if a model is valid and valuable; the model should account reasonably well ⁶ for the experimental data, but also and foremost teach us something about the mechanisms underlying the phenomenon.

A more detailed (and hence complex) model will in general – if it is correct – reproduce in more detail the experimental results; but sometimes this happens – in particular if the detailed model is used too early – at the expenses of our understanding: a simpler model, less effective from the “quantitative” point of view may be highly superior from the “qualitative” point of view, in the sense it allows to focus on a smaller numbers of sides of the problem, and if successful it allows to better discriminate between essential aspects and less important ones.

⁶What “reasonably well” means depends of course on what we are looking for, and also on what we know before using the model.

On the other hand, once we got an understanding of the fundamental mechanisms, a detailed model will surely be more useful: in order to know which are the forces which make an airplane to fly, the model of a straight uniform wing – disregarding the fuselage and the engines – in an external air flow is wonderful; but in order to build a jumbo jet one needs some details more.⁷

1.4 Bibliography

The Ho model is introduced in the 1995 paper by Ho *et al.*; see also the 1989 paper by Ho, Mougdil and Alam⁸. This model is discussed in Murray, from which the present discussion is taken. A review of AIDS models is provided by Perelson and Nelson. An interesting discussion of how a too detailed model can actually produce *worse* agreement with experimental data even in a complex system (in this case, the dynamics of the atmosphere and weather forecast) is given in Cencini *et al.*

- D. Ho et al., “Rapid turnover of plasma virions and CD4 lymphocytes in HIV-1 infection”, *Nature* **373** (1995), 123-126
- D.D. Ho, T. Mougdil and M. Alam, “Quantitation of Human Immunodeficiency Virus Type 1 in the Blood of Infected Persons”, *New Engl. J. Med.* **321** (1989), 1621-1625
- J.D. Murray, *Mathematical Biology. I: An Introduction*, Springer (Berlin) 2002
- A.S. Perelson and P.W. Nelson, “Mathematical models of HIV-1 dynamics in vivo”, *SIAM Rev.* **41** (1999), 3-44
- M. Cencini, A. Puglisi, D. Vergni and A. Vulpiani, *A Random Walk in Physics*, Springer 2021

⁷The correspondence which can be attained with experimental data depends also on the nature of the system. For a complex system, like a biological one, a 10% error can be considered very satisfactory, while in elementary particle Physics it is customary to attain relative errors of order $10^{-7} - 10^{-8}$.

⁸The fact these are published in non-mathematical journals should not prevent the reader to have a glance at them; even more nowadays when subscription to electronic resources allows one to have access to all the periodicals available to the University, and not just to those of his/her specialized library.

Chapter 2

Population Dynamics

We want to consider the dynamics of a population. We denote by P the size of the population, i.e. the number of individuals - albeit in some cases we will not actually be able to count the individuals, but estimate this number e.g. in terms of a weight (e.g. for a bacterial colony, or for the number of wheat grains produced in a year).

There are two basic way to describe how P varies in time: that is, we can consider a *continuous* time variation, and then write $P = P(t)$ and describe the variation of P with t in terms of an ordinary differential equation – or a system thereof if we consider several interacting populations – i.e. by a (continuous time) *dynamical system*. Or we could consider how P varies from one generation to another and write P_k for the population in the k -th generation; in this case the evolution of P will be described by a map $P_k \rightarrow P_{k+1}$ – or a set of coupled maps if we consider interacting populations – i.e. by a discrete time dynamical system.

The latter description makes better sense when we have non-overlapping generations. This is e.g. the case for many plants and agricultural products (wheat, wine, ...) or for animals reproducing in a definite season (penguins, salmons,...). When we deal with species which can reproduce at any time (virus, bacteria, mammals, humans,...) it is natural to consider a continuous time description; one can also consider a discrete time description, but in this case this does not provide a generation-to-generation map, but just a discretization of the dynamics, e.g. giving the level of the population every day, or week, or month, rather than in continuous time.

2.1 Growth rate, and the Malthusian evolution

It is essential to familiarize with a couple of fundamental concepts. First of all, in ideal conditions (we will see in a moment what we mean by this) a given species in a given environment has a *growth rate*. In the continuous time description,

this means that – at least on a short time scale – we have

$$\frac{dP}{dt} = \alpha P ; \quad (2.1)$$

here the real constant $\alpha > 0$ is the growth rate (if $\alpha < 0$ we actually have a decrease rate).

Similarly, if we have a discrete time description then – at least on short time scales – we have

$$P_{k+1} = A P_k . \quad (2.2)$$

In this case the growth rate A is a real constant $A > 1$ (if $A < 1$ we have a decrease) which describes the ratio of the population at two successive times, i.e. P_{k+1}/P_k ; note that if we want to consider $P_{k+1} - P_k$, then this is described by $(A - 1) P_k$. In other words the “discretization” of

$$\alpha = \frac{dP/dt}{P} \simeq \frac{[P(+\delta t) - P(t)]/\delta t}{P(t)}$$

is then $(A - 1)$. Keeping in mind this trivial observation will avoid confusion in a number of occasions.

Now there is an obvious (at least to the mathematician) problem with eqs. (2.1) and (2.2): they lead to an *exponential growth*. In continuous time, we have

$$P(t) = e^{\alpha t} P(0) , \quad (2.3)$$

and in discrete time

$$P_k = A^k P_0 . \quad (2.4)$$

As well known, an exponential growth goes beyond any given limit within a *finite time*, whatever the initial (nonzero) level of the population.

That is, given any limit B , for the continuous time evolution we have¹

$$P(t) > B \quad \text{for } t > \frac{1}{\alpha} \log \left[\frac{B}{P(0)} \right] .$$

Similarly, for the discrete time evolution, we have

$$P_k > B \quad \text{for } k > \frac{1}{\log(A)} \log \left[\frac{B}{P_0} \right] .$$

The model of exponential growth of a population goes under the name of Th.R. Malthus (1766-1834); but actually Malthus was the first to realize that this is impossible, exactly for the simple reason mentioned above. In fact, a continued exponential growth is impossible not only for very long times, but also on timescales which are surprisingly small.

Exercise. The typical mass of a bacterium is $m_0 = 10^{-12}$ g. A bacterium provided with nutrients can reproduce, giving origin to two bacteria, in about

¹Here and below, $\log(x)$ will denote the *natural* logarithm of x .

20 minutes (or faster). Compute the time T needed for a single bacterium having unlimited nutrients to give rise to a bacterial colony of total mass M . Give explicit values for T_i in the following cases:

$$\begin{cases} M_1 = 70 \text{ kg} & \text{(mass of a human)} \\ M_2 = 200 \text{ tons} & \text{(mass of a blue whale)} \\ M_3 = 6 * 10^{24} \text{ kg} & \text{(mass of the Earth)} \\ M_4 = 2 * 10^{30} \text{ kg} & \text{(mass of the Sun)} \\ M_5 = 1.5 * 10^{53} \text{ kg} & \text{(mass of the Universe)} \end{cases}$$

It is convenient, in order to better appreciate the results, to express the T_i in days or years.

Solution. Let us work in the **cgs** system. We have to evaluate the growth rate α for the bacterial population; as we know that it doubles in 20 minutes, i.e. in $t_0 = 1200$ seconds, we must have $\exp[\alpha t_0] = 2$, i.e.

$$\alpha = \frac{1}{t_0} \log(2) \approx 5.78 * 10^{-4} \text{ s}^{-1} .$$

We can then use the formula obtained above, with $B = M_i/m_0$ and $P(0) = 1$:

$$T_i = \frac{1}{\alpha} \log \left[\frac{M_i}{m_0} \right] = \frac{t_0}{\log 2} \log \left[\frac{M_i}{m_0} \right] .$$

We leave to the reader to make the explicit computations.

Exercise. The numerical answers obtained in response to the previous exercise should be rather surprising. It is worth checking these by proceeding the other way round. Consider the bacterial colony generated by a single bacterium through replication every 20 minutes; compute the total mass $M(t_i)$ of the colony after times:

$$\begin{cases} t_1 = 1 \text{ hour} \\ t_2 = 1 \text{ day} \\ t_3 = 1 \text{ month} \\ t_4 = 1 \text{ year} \\ t_5 = 1 \text{ century} \end{cases}$$

Solution. The solution is simply

$$M(t) = e^{\alpha t} m_0 .$$

An even simpler answer – maybe needed in case of disbelief of the answer obtained for the previous Exercise – is provided by noting that the bacteria reproduce every 20 minutes, so there are 3 reproduction cycles in a hour, 72 reproduction cycles in a day, 2,160 cycles in a month, 26,280 cycles in a year, and 2,628,000 cycles in a century.

After N reproduction cycles, the total mass will be

$$M = 2^N m_0 .$$

2.2 Carrying capacity

The point, as emphasized by Malthus, is that exponential growth can take place only in the presence of abundant resources. But resources are abundant (or scarce) in relation to the population which feeds on them, and whatever the level of available resources an unlimited growth – as it is the case for exponential growth – will make them scarce, and actually insufficient. The limitedness of resources leads necessarily to a *limit of growth*.²

For a given species in a given environment – which includes also the other living species – say providing a stable amount of renewable resources (e.g. food), there is a maximal number of individual which can be stably supported by the available resources. This number is known as the *carrying capacity*.³

If we insist, as customary in Biology, to describe the evolution of $P(t)$ in terms of a growth rate, we should thus admit that this is not a constant, but it depends – in a given environment – on the level reached by $P(t)$. In other words, when we write (2.1), we should see α as a function of P . Note that here we are implicitly assuming the environment is stationary, so that the equations are autonomous; a varying environment would lead to a non-autonomous equation, hence on α depending on time as well.

Needless to say, if $\alpha = \alpha(P)$, then (2.1) is not any more a linear one⁴; at this point, different choices for the function $\alpha(P)$ will lead to different time evolution, and one should discuss what are the features a realistic choice of α should have, and how different choices within this class will affect the dynamics.

2.3 The logistic model

We want thus to consider an equation of the form

$$\frac{dP}{dt} = [\alpha(P)] P . \quad (2.5)$$

We know that for small P resources are surely abundant; thus we are in the "ideal" situation mentioned above, and the growth rate will correspond to some constant depending essentially on the characteristics of the living species we are

²This very classical concept was made clear by Malthus at the end of XVIII century; apparently, the enthusiasm for economic growth led to oblivion of it, so that when it was "rediscovered" – or actually recalled, for it was surely not forgotten by scientists – in 1960 as the "limit to growth" in a famous report by a MIT working group led by A. Pececi, it came as a surprise, if not to scientists at least to politicians and media leaders, and actually sound unbelievable to them for many decades.

³Note that this is not a "physical" limit: the population could temporarily grow beyond this limit, but this means an overconsumption of resources, and in turn this means that renewable resources can actually not be renewed and replaced, causing a diminution of the carrying capacity for later generations.

⁴One could argue that in this case the very concept of "growth rate" is ill-defined; as already mentioned, we will use this in the same way as biologists and demographers do, i.e. just as a description of the instantaneous ratio $(dP/dt)/P$.

considering. In mathematical terms, this means

$$\lim_{P \rightarrow 0^+} \alpha(P) = \alpha_0 ;$$

here of course α_0 is a positive real constant. We will also write, with some sloppiness,

$$\alpha(0) = \alpha_0 ;$$

note that strictly speaking this is meaningless: if the population is exactly zero, there is nobody which can reproduce, and the growth rate is thus exactly zero. Thus the above is just a lazy notation to mean the limit, as we correctly wrote just before.

On the other hand, once the population reaches the carrying capacity P_* , it will not be able to grow further and we will have a stationary population. In view of (2.5) this means we should require

$$\alpha(P_*) = 0 .$$

We have thus established two constraints on the form of $a(P)$:

$$\alpha(0) = \alpha_0 , \quad \alpha(P_*) = 0 . \quad (2.6)$$

The simplest choice for the function $\alpha(P)$ satisfying these is of course a *linear* function $\alpha(P) = \alpha_0 - \beta_0 P$; the value of β_0 is readily obtained imposing $\alpha(P_*) = 0$, which yields $\beta_0 = \alpha_0/P_*$, and hence

$$\alpha(P) = \alpha_0 \left(1 - \frac{P}{P_*} \right) .$$

The equation for $P(t)$ is hence

$$\frac{dP}{dt} = \alpha_0 \left[1 - \frac{P}{P_*} \right] P . \quad (2.7)$$

This equation is known as the *logistic equation*, and correspondingly the model of growth with $\alpha(P)$ of this form is known as the *logistic model*. It is associated with the name of the Flemish mathematician P.F. Verhulst (1804-1849), who developed it in the first half of the XIX century. The equation – or rather the growth model – was then “rediscovered” a number of times, e.g. by McKendrick, Pearl, and Lotka.

It is rather clear looking at (2.7) that one would have some advantage in considering not the absolute value reached by P , but rather the fraction of P_* it represents. In fact, (2.7) contains two parameters, i.e. α_0 and P_* , while passing to measuring P in terms of P_* only α_0 would be left. Note also that even this parameter can be eliminated by changing the unit of time.

We thus introduce the ratio

$$x := P/P_* . \quad (2.8)$$

In other words, we write $P(t) = p_* x(t)$, and obviously have $dP/dt = P_*(dx/dt)$. The equation (??) becomes then

$$P_* dx/dt = \alpha_0 [1 - x(t)] P_* x ,$$

and eliminating $P_* \neq 0$ from both sides we reach the dimensionless form

$$\frac{dx}{dt} = \alpha_0 (1 - x) x . \quad (2.9)$$

As mentioned above, this can be further simplified simply by changing the unit of time, i.e. writing $t = \tau/\alpha_0$. Then we have

$$\frac{dx}{d\tau} = (1 - x) x . \quad (2.10)$$

Solving (2.10) or (2.9) is of course as difficult – or actually, as easy – as solving (2.7), but these formulations have a double advantage:

- If we want to study how well this model describes the growth of a given population, using (2.9) we have to fit a single parameter, i.e. α_0 , against experimental data.
- The “universal” form (2.10) shows that the growth of different species with – possibly, hugely – different time scales is described within this model by a universal behavior, and differ indeed just for the involved time scale.⁵

We will mainly work with the model in its form (2.9), i.e. retain the information about the timescale of the population growth.

Solving (2.9) is elementary: in fact, this is a separable equation, and writing it in separated form as

$$\frac{dx}{(1-x)x} = \alpha_0 dt$$

and integrating, we get

$$x(t) = \frac{e^{\alpha_0 t}}{e^{\alpha_0 t} - c} ,$$

with c a constant. Expressing this in terms of the initial condition $x(0) = x_0$, we get

$$x(t) = \frac{e^{\alpha_0 t} x_0}{1 - x_0 + e^{\alpha_0 t} x_0} . \quad (2.11)$$

This type of curve is known as a *sigmoid*. See fig.2.1 for a plot.

Exercise. Derive the explicit solution of the logistic equation.

Exercise. Predict the qualitative behavior of $x(t)$ based on the equation (2.9) alone, i.e. without explicitly solving it.

⁵The reader having some familiarity with Fluid Mechanics will specially appreciate this feature.

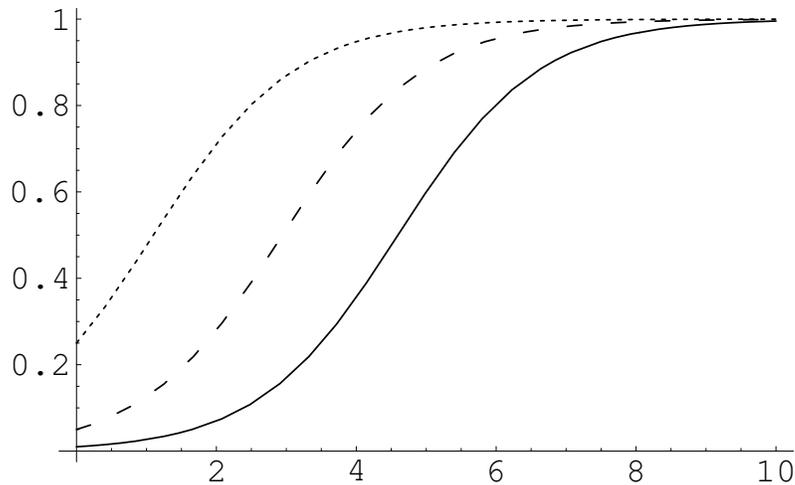


Figure 2.1: Plot of $x(t)$ solving the logistic equation with $\alpha_0 = 1$ and for different initial data, i.e. $x_0 = 0.01$ (solid), $x_0 = 0.05$ (dashed), $x_0 = 0.25$ (dotted).

2.4 Examples and counterexamples of applications of the logistic model

The logistic model is our “standard model” for population growth. This status is not a consequence of its simplicity, but of its success in matching experimental results.

In Fig.?? we report data for the growth of a population of *Drosophila*; this was a historical experiment (indeed our figure is a graphical elaboration on data given by A. Lotka in his cornerstone book). As the reader can see, the logistic equation succeeds in describing rather well the population dynamics in this case.

On the other hand, one should be careful about other applications of the logistic growth. In his wonderful book, Murray, reports the claims that the logistic model also described the growth of human population, which was made by some authors, based on demographical data for France and USA. Needless to say, these data cover quite different stage of the countries’ life (as will be clear to anybody with a vague idea of their history); in particular, the data for USA fall in the initial (growing) part of the sigmoid, while those for France fall in the later (slowing down growth) part.

These data do not mean anything, only witness that there is some competition for resources. Fitting a small part of the curve is not a test but just tells that near a fixed point the dynamics is governed by a linearized equation. We refer to Murray for a detailed discussion.

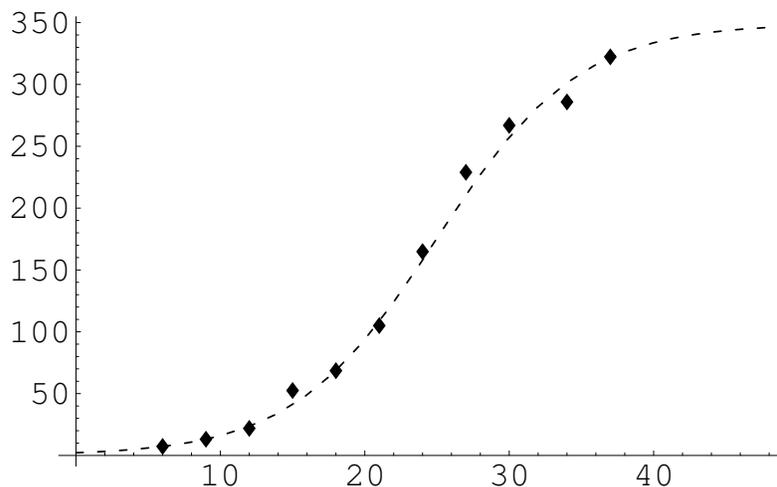


Figure 2.2: Growth of a *Drosophila* population; graphical elaboration from A.J. Lotka, *Elements of Mathematical Biology*, Dover 1956. The solid dots represents experimental data, the dashed line is a fit with the logistic growth $p(t) = \alpha/(1 - \beta e^{-\lambda t})$. In this case the parameters yielding the best fit (shown here) are $\alpha \simeq 349.33$, $\beta \simeq -158.73$, $\lambda \simeq 0.203$.

2.5 Generalized logistic growth

We have so far considered the *simplest* choice for the function $\alpha(P)$ in (2.5); this allowed for an explicit integration of the model.

Needless to say, one could choose different $\alpha(P)$, provided the limit conditions in $P = 0$ and $P = P_*$ are satisfied. Actually, it would be rather natural to suppose that the effect of limited resources does not really show up until scarcity of these can be felt, i.e. until we are near enough to P_* . The simplest way to take this into account is by assuming

$$\alpha(P) = (1 - P/P_*)^\gamma$$

for some real exponent $\gamma > 0$. We will denote the equation

$$\frac{dx}{dt} = n\alpha_0 (1 - x)^\gamma x \quad (2.12)$$

as the *generalized logistic equation*.

In view of the good success of the logistic model at describing population growth, we expect the biologically interesting range of γ is for $\gamma \approx 1$.

Exercise. Plot $\alpha(x)$ of this form for different choices of γ , e.g. for $0.5 < \gamma < 2$ choosing a few different values of γ in this range.

Solve then numerically the corresponding generalized logistic equations for the same initial datum $x(0) = x_0 \ll 1$, and compare the dynamics.

Exercise. Consider the following (fake) experimental data for the growth of a population (these are actually generated by a standard logistic growth with fluctuations), given in the form $(t, x(t))$:

$$\begin{aligned} &(1, 0.0096), (2, 0.0204), (3, 0.0002), (4, 0.0681), (5, 0.0942), \\ &(6, 0.0627), (7, 0.1540), (8, 0.2587), (9, 0.2793), (10, 0.4158), \\ &(11, 0.6213), (12, 0.4921), (13, 0.6795), (14, 0.8597), (15, 0.8810), \\ &(16, 0.9423), (17, 0.9402), (18, 0.9549), (19, 0.9623), (20, 0.9669) \end{aligned}$$

Try to fit these by your generalized logistic growth for several (say, four) different choices of γ , and give the best fit and the χ^2 value for this. Note that for the fit you give γ for granted – albeit you should try with different values – but you do not know neither α_0 nor x_0 .

2.6 The Allee effect

We have supposed that the best situation for the replication of an individual is to be in a rarefied population, so that there is plenty of resources for each one. However, life can arrange things differently.

For example, social insects (like ants or bees) live in structured colonies, and these cannot work properly below a certain size. Actually, they cannot survive, even less reproduce, if the colony is too small.

Similarly, consider mammals which live alone but need to meet to mate: if their spatial density is too low, meeting can become exceedingly difficult or rare, leading to a diminished growth or even a negative one.

The fact that the growth rate can be negative for $P < P_0$ is known as the *Allee effect*, after W.C. Allee (1885-1955; his works on the mechanism which later on took his name go back to around 1930).

In this case we have (at least) two zeros for $\alpha(P)$:

$$\alpha(P_0) = 0, \quad \alpha(P_*) = 0;$$

generically, we have $\alpha'(P_0) > 0$ and $\alpha'(P_*) < 0$.

The simplest choice satisfying these constraints is

$$\alpha(P) = \tilde{\alpha}_0 \left(\frac{P}{P_0} - 1 \right) \left(1 - \frac{P}{P_*} \right). \quad (2.13)$$

In terms of x this reads

$$\alpha(x) = \alpha_0 (x - \delta) (1 - x), \quad (2.14)$$

having introduced the new constants

$$\alpha_0 := \tilde{\alpha}_0 \frac{P_*}{P}, \quad \delta := \frac{P_0}{P_*}.$$

Note that $0 < \delta < 1$.

The corresponding growth equation is of course

$$\frac{dx}{dt} = \alpha_0 (x - \delta) (1 - x) x . \quad (2.15)$$

This is still a separable equation, but its solution can not be expressed by elementary functions. On the other hand, we have trivially that the dynamics admits an unstable fixed point at $x_u = \delta$ and two stable ones at $x = 0$ and $x = 1$; the solution $x(t)$ will be asymptotic to either one of these depending on $x(0) = x_0$ being lower or higher than x_u .

2.7 The logistic model in a different context. Chemical kinetics

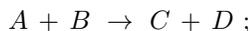
The main mathematical feature of the logistic model is that its nonlinearity is *quadratic*. From the physical point of view, this corresponds to having a *two-body interaction*. In our population dynamics context, this is reflected in the (implicit) assumption that each individual is competing for resources with any other one (the case in which the competition is not among individuals but among small groups, e.g. wolf herds, only changes by a little factor the involved scale).

Needless to say, two-body interactions are very common – actually, the most common case – and it is not surprising that the same type of equations is met in many rather different physical contexts.⁶

Here we will to briefly consider the case of *Chemical kinetics*. We will consider “ordinary” reactions as well as auto-catalytic ones (the reason for considering these will be apparent in the following).

2.7.1 Ordinary reactions

Let us consider a chemical reaction



we will denote by λ and μ the initial concentrations of the reagents A and B , and assume (just to dispose of some inessential constants) that C and D are initially absent. Moreover, we assume that throughout the time in which the reaction is observed, the reagents are well stirred, i.e. their concentration is spatially uniform. With a slight abuse of notation, we will denote by $A(t)$ the concentration of A at time t , and so on; thus we have $A(0) = \lambda$, $B(0) = \mu$.

Actually, as C and D are initially absent and they are produced in the same reaction and in equal number, we will always have $C(t) = D(t)$; we will denote

⁶After all, the real strength of (applied) Mathematics is that the same abstract equations can be studied once and describe different phenomena.

these quantities by $y(t)$. Similarly, the production of C and D is exactly equal to the consumption of A and B ; thus we have

$$A(t) = A(0) - y(t) = \lambda - y(t), \quad B(t) = B(0) - y(t) = \mu - y(t).$$

We have so far used some “conservation laws” to reduce the initial four degrees of freedom to just one. But we have not yet described the kinetics.

The reaction will develop at a characteristic speed, depending on the chemical affinity α of A and B and on the concentration of the reagents: that is, we have

$$\frac{dy}{dt} = \alpha A(t) B(t) = \alpha (\lambda - y) (\mu - y). \quad (2.16)$$

Note that the reaction automatically stops when $x(t)$ reaches either λ or μ , as this condition means it runs out of fuel. We will suppose, for definiteness, that $\lambda > \mu > 0$, with $\lambda - \mu = \delta$.⁷

The equation (2.16) is again a separable equation, and is readily integrated in terms of elementary functions. The resulting expression is somewhat simplified by our initial condition $y(0) = 0$, and with this we get

$$y(t) = \lambda + \frac{\delta}{(\mu/\lambda) \exp[-\alpha\delta t] - 1}. \quad (2.17)$$

What has this to do with the logistic model? It suffices to operate the change of variables

$$x := y - \mu$$

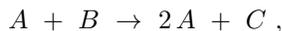
to transform the equation (2.16) into

$$\frac{dx}{dt} = -\alpha (\lambda - \mu - x) x = -\alpha (\delta - x) x, \quad (2.18)$$

i.e. to recover the logistic equation.

2.7.2 Autocatalytic reactions

The correspondence is even more direct when we consider an autocatalytic reaction, i.e. a chemical reaction of the form



i.e. a “bare reaction” $B \rightarrow A + C$ in which one of the products of the reaction acts as a catalyzer for the reaction itself. Proceeding as above, and assuming again $C(0) = 0$ – and writing $C(t) = y(t)$ for ease of notation – we have

$$\frac{dx}{dt} = \alpha A(t) B(t);$$

⁷The reader is invited to consider, as an exercise, the degenerate case $\lambda = \mu > 0$.

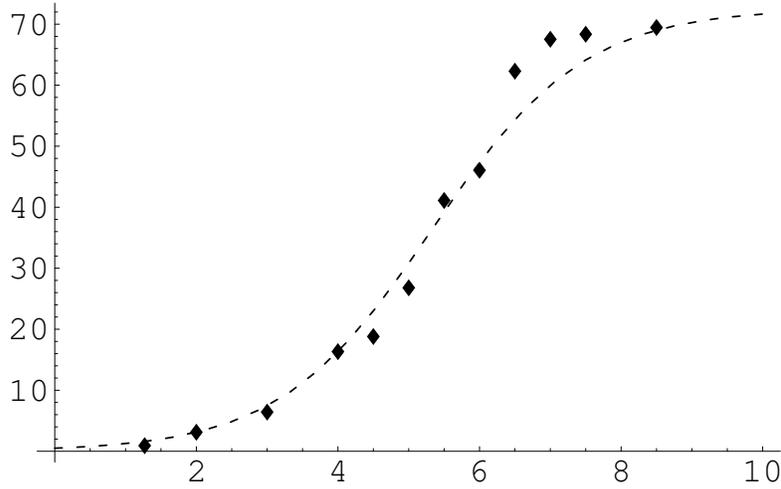


Figure 2.3: Auto-catalytic activation of crystalline tripsinogen; graphical elaboration from J.H. Northrop, M. Kunitz and R.M. Herriot, *Crystalline enzymes*, Cambridge UP 1948. Solid dots represent experimental measurements, while the dashed curve is a fit by a logistic curve $y = \alpha/(1 - \beta e^{-\lambda t})$; here time is measured in hours, and $\alpha \simeq 72.56$, $\beta \simeq -193.94$, and $\lambda = 0.933$.

on the other hand, as each elementary reaction dispose of a C and produces an A , we have

$$B(t) = B(0) - y(t), \quad A(t) = A(0) + y(t);$$

thus, writing again $A(0) = \lambda$, $B(0) = \mu$, the dynamical equation reads

$$\frac{dy}{dt} = \alpha (\lambda + y) (\mu - y); \quad (2.19)$$

finally we either solve this in the same way as in the previous subsection, either operate the change of variables

$$x = y - \mu$$

which yields

$$\frac{dx}{dt} = \alpha (\lambda - \mu + x) x. \quad (2.20)$$

The theoretical curve, obtained as solution to this equation, is compared with experimental results in Fig.??.

2.8 Exercises

Exercise 1. Consider the population dynamics described by

$$dP/dt = A(P) P, \quad A(x) := k e^{-x}.$$

Determine the equilibria, and their stability, as the real positive parameter k is varied.

Exercise 2. Consider the population dynamics described by

$$dP/dt = A(P) P, \quad A(x) := k (x - e^{-x}) .$$

Determine the equilibria, and their stability, as the real positive parameter k is varied.

Exercise 3. Consider the population dynamics described by

$$dP/dt = A(P) P, \quad A(x) := \alpha (\beta^2 - x^2) .$$

Determine the equilibria, and their stability, as the real positive parameters α and β are varied.

Exercise 4. Consider the population dynamics described by

$$dP/dt = A(P) P, \quad A(x) := k (1 - x^3) .$$

Determine the equilibria, and their stability, as the real positive parameter k is varied.

Exercise 5. Consider the population dynamics described by

$$dP/dt = A(P) P, \quad A(x) := \alpha \left(\frac{1 - \beta^2 x^2}{1 + \beta x} \right) .$$

Determine the equilibria, and their stability, as the real positive parameters α and β are varied.

2.9 Bibliography

Population models are discussed in virtually any text in Mathematical Biology; we will thus just quote our generally favorite ones, i.e. those by Murray, Britton, and Edelshtein.

For historical information – not only about these matters, but in general for the development of Mathematical Biology – one can refer to the very nice book by N. Bacaer.

The treaty by T.R. Malthus, *An Essay on the Principle of Population*, was first published anonymously in 1798; the reader will find an account of its history and contents in the dedicated Wikipedia article. This article also contains a link to a full version of Malthus treaty.

- N. Bacaer, *A Short History of Mathematical Population Dynamics*, Springer 2011
- T. Britton, *Essential Mathematical Biology*, Springer 2005
- L. Edelstein-Keshet, *Mathematical Models in Biology*, SIAM 2005
- J.D. Murray, *Mathematical Biology. I: An Introduction*, Springer 2002

Chapter 3

Population dynamics with delay

Introduction

In our basic equation for population dynamics,

$$\frac{dP}{dt} = \alpha(P) P, \quad (3.1)$$

it is assumed that the population at time P reproduces at a rate $\alpha(P)$ which depends on the level reached by the population itself. As it often happens, there is a hidden assumption here, i.e. that the *whole* population is reproducing. If we think of a human population, we immediately realize this is not realistic: only individuals which are in their fertile age can reproduce. Similarly, if we think of any kind of living being, no offspring can be delivered before reaching reproductive age¹. Thus, to make an example, in our example of bacterial growth considered in a previous Chapter, we should not consider the whole population as possibly reproducing, but only the bacteria which were in existence at least 20 minutes earlier.

That is, the population taking part in reproduction at time t is not $P(t)$, but $P(-\delta)$, with δ a *delay* which depends on the species at hand². Note that, on the other hand, the whole population $P(t)$ competes for the available resources at time t .

Thus our equation (3.1) should more realistically be replaced by

$$\frac{dP(t)}{dt} = \alpha[P(t)] P(t - \delta), \quad (3.2)$$

with $\delta > 0$ a real constant; i.e. by a *differential equation with delay*.

¹Or its equivalent: e.g. for cells the criterion is to have reached a sufficient size.

²And possibly on environmental conditions: e.g., for humans the lack of fat in the body delays fertility, and in turn lack of fat often depends on lack of sufficient nutrition.

Note that here we are assuming that the whole population having reached a sufficient age is able to reproduce; in other words, we are disregarding the fraction of population which is too old to reproduce. This is uncorrect in the case of humans, but correct for most beings.

In this regard, it should maybe be emphasized that albeit we always refer to α as the growth rate, it is the net result of a reproduction rate and a mortality rate. While only individuals in reproductive age can reproduce, all of them may die. Thus we should more precisely write an equation of the form

$$\frac{dP(t)}{dt} = \beta[P(t)] P(t - \delta) - \mu P(t) , \quad (3.3)$$

where β represents a birth rate, and μ a mortality rate. We will for the time being discuss the simpler equation (3.2), deferring discussion of (3.3) after we will have learned how to deal with delay equations.

3.1 Dynamical systems with delay

When we study nonlinear dynamical systems, we are aware that except in very lucky circumstances we are unable to exactly solve the system, and base our understanding on qualitative analysis. The first step in this is to study the equilibria of the system and their stability.

We will adopt the same approach for systems with delay. We will define a general handy notation, i.e. if $x(t)$ is a function of t , and the delay δ is given, we define

$$\hat{x}(t) := x(t - \delta) . \quad (3.4)$$

We will use this notation without further notice in the following.

3.1.1 Equilibria and dynamics near equilibria

We consider a generic autonomous system

$$\frac{dx}{dt} = f(x, \hat{x}) . \quad (3.5)$$

Our discussion is restricted to one-dimensional systems, as suggested by the notation above, but it would extend with no conceptual difficulties – albeit with a more involved notation, and more careful setting of necessary and/or sufficient conditions – to higher dimensions; we believe the essential point is to grasp how to deal with the delay, and no unneeded complications should be considered at this stage.

First of all we look for equilibria; these correspond to $x(t)$ being a constant. The condition means of course that $x(t) = x_0$ for all t , and this in turn implies that (at equilibria)

$$\hat{x}(t) = x(t) .$$

Thus equilibria are characterized as solutions to

$$F(x) := f(x, x) = 0 . \quad (3.6)$$

In qualitative terms, equilibria are obtained considering the system with no delay ($\delta = 0$).

Let us now suppose we have determined one or more equilibria, and denote it as x_0 . We should then discuss if this is stable or unstable. The discussion is standard in the case of no delay, and the answer is just given by the sign of the derivative of F computed at x_0 , i.e. by

$$F'(x_0) .$$

We thus are able to know the stability of the system with no delay. But the delay could create an instability for a stable equilibrium, or even stabilize an unstable one; and we have to understand how to study this question.

As for any stability discussion, we can pass to consider the *linearization* of our equation (3.5) about the equilibrium point x_0 . This is given by

$$\frac{dx}{dt} = \left(\frac{\partial f}{\partial x} \right)_{(x_0, x_0)} (x - x_0) + \left(\frac{\partial f}{\partial \hat{x}} \right)_{(x_0, x_0)} (\hat{x} - x_0) + \text{h.o.t.} . \quad (3.7)$$

In the following we will omit to stress that there are higher order terms, and just work at first order in $(x - x_0)$ and $(\hat{x} - x_0)$.

As emphasized by our notation, the partial derivatives of f w.r.t. x and \hat{x} should be computed at the equilibrium, i.e. for $x = x_0$, $\hat{x} = x_0$. Thus they are just numbers, and we can rewrite the linear equation (3.7) as

$$\frac{dx}{dt} = A (x - x_0) + B (\hat{x} - x_0) . \quad (3.8)$$

Moreover, it is obviously convenient to use different coordinates, centered in $x = x_0$; we thus define

$$y(t) := x(t) - x_0 ;$$

note that with our general notation we also have

$$\hat{y}(t) = y(t - \delta) := x(t - \delta) - x_0 = \hat{x}(t) - x_0 .$$

The equation (3.8) reads then

$$\frac{dy}{dt} = A y + B \hat{y} . \quad (3.9)$$

Needless to say, this is the most general linear equation with (simple) delay, and we could have known *a priori* that in the end we had to analyze this kind of equations in order to study stability.

3.1.2 Stability

We will make an *ansatz* (a very reasonable one) for the solutions to the linear equation (3.9); that is, we look for solutions in the form

$$y(t) = e^{\lambda t} y_0 ; \quad (3.10)$$

we can assume $y_0 \neq 0$ (or we would just stay at the equilibrium, which is not interesting), but as we are morally in two dimensions we will not assume λ real. In general, we consider $\lambda \in \mathbf{C}$. Obviously, $y = 0$ will be stable if $\text{Re}(\lambda) < 0$, unstable if $\text{Re}(\lambda) > 0$. It is convenient to write

$$\lambda = \mu + i \nu , \quad (3.11)$$

with μ and ν real; thus stability is controlled by the sign of μ .

With (3.10) we have also

$$dy/dt = \lambda e^{\lambda t} y_0 = \lambda y , \quad \hat{y}(t) = y(t - \delta) = e^{\lambda(t-\delta)} y_0 = e^{-\lambda \delta} y(t) .$$

Inserting these into eq.(??), we get

$$\lambda y = A y + B e^{-\lambda \delta} y . \quad (3.12)$$

As we assumed $y_0 \neq 0$, we also have $y(t) \neq 0$ (for all finite t) and we can eliminate y from the previous equation. We are thus left with an equation for λ in terms of A, B, δ , i.e.

$$\lambda = A + B e^{-\lambda \delta} . \quad (3.13)$$

Unfortunately, this is in general (i.e. for $B \neq 0$; but for $B = 0$ we have a no-delay system from the beginning, and nothing to discuss) a transcendent equation, and we know it cannot be solved exactly.

On the other hand, the solution to the equation (3.13) would provide λ as a function of δ , and as remarked above we are actually primarily interested in μ (rather than in the whole λ), and more precisely in the sign of $\mu = \mu(\delta)$ as a function of δ . But we know the sign of $\mu_0 = \mu(0)$, since we know how to study stability for zero delay. Thus we are actually interested in determining if and when – i.e. for which values of δ – there is a *change of sign* in $\mu(\delta)$. For the sign to change, $\mu(\delta)$ must go through zero.

We can now rewrite eq.(??) using (3.11), which gives a system of two real equations (corresponding to the real and imaginary parts of the two members), i.e.

$$\begin{cases} \mu = A + B e^{-\mu \delta} \cos(\nu \delta) \\ \nu = B e^{-\mu \delta} \sin(\nu \delta) \end{cases} . \quad (3.14)$$

For $\mu = 0$ these yield

$$\begin{cases} A + B \cos(\nu \delta) = 0 \\ B \sin(\nu \delta) = \nu \end{cases} . \quad (3.15)$$

The first equation requires

$$\cos(\nu \delta) = -(A/B) ; \quad (3.16)$$

this admits a solution provided

$$R := |A/B| \leq 1 . \quad (3.17)$$

Our first conclusion is therefore that if (3.17) is *not* satisfied, then the stability does not change, and this for any value of the delay δ .³

Let us study the case where (3.17) is satisfied. Under the condition (3.17), we have

$$\sin(\nu \delta) = \pm \sqrt{1 - A^2/B^2} = \pm \sqrt{\frac{B^2 - A^2}{B^2}} .$$

Thus at the critical value for δ , call it δ_* and correspondingly $\nu_* := \nu(\delta_*)$, we have

$$\nu_* = B \sin(\nu_* \delta_*) = \pm B \frac{\sqrt{B^2 - A^2}}{B} = \pm \sqrt{B^2 - A^2} . \quad (3.18)$$

Note that by definition $\mu(\delta_*) = 0$, so at $\delta = \delta_*$ the solutions (3.10) are oscillating ones; the parameter ν_* describes the frequency of these oscillating solutions, and more generally the frequency of bifurcating solutions at the critical point.

Going back to (3.16), this yields the critical delay as

$$\delta_* = \pm \frac{\arccos(-A/B)}{\sqrt{B^2 - A^2}} .$$

Finally we recall that by definition $\delta > 0$ (for $\delta < 0$ we would not have a delay, but an anticipation), so we can finally write

$$\delta_* = \frac{|\arccos(-A/B)|}{\sqrt{B^2 - A^2}} . \quad (3.19)$$

It should be noted that (3.19) does not uniquely determine δ_* . Actually, this is completely correct: we will have not only one change of stability, but multiple ones, taking place at all the delays δ_* satisfying (3.19).

3.2 Logistic equation with delay

We have understood how to study equilibria and stability for a (scalar) ODE with delay. We are now ready to apply what we have learned to the population dynamics (3.2). In this case we have simply

$$dP/dt = f(P, \hat{P}) := \alpha(P) \hat{P} ; \quad (3.20)$$

³It may be worth remarking that $|A/B| = |A|/|B| \leq 1$, hence $|B| \geq |A|$, means that at equilibrium the derivative of f w.r.t. the delayed variable \hat{x} must be not smaller – in absolute value – than the derivative w.r.t. the standard variable x .

therefore,

$$\partial f / \partial P = \alpha'(P) \hat{P}, \quad (\partial f / \partial \hat{P}) = \alpha(P).$$

At the equilibrium $P = P_e$, we have in terms of the notation used in our general discussion

$$A = P_e \alpha'(P_e), \quad B = \alpha(P_e).$$

Thus the parameter controlling the possibility of a change of stability is simply

$$R = \left| \frac{A}{B} \right| = \left| \frac{\alpha(P_e)}{P_e \alpha'(P_e)} \right|.$$

Let us focus on the standard logistic equation with delay, i.e.

$$\frac{dP}{dt} = \alpha_0 \left[1 - \frac{P}{P_*} \right] \hat{P}. \quad (3.21)$$

It is of course still convenient to operate the change of variables $P = P_* x$, and with this we get

$$\frac{dx}{dt} = \alpha_0 (1 - x) \hat{x} := f(x, \hat{x}). \quad (3.22)$$

There are two equilibria,

$$x = x_0 = 0, \quad x = x_* = 1.$$

As for the derivatives, we have

$$\frac{\partial f}{\partial x} = -\alpha_0 \hat{x}, \quad \frac{\partial f}{\partial \hat{x}} = \alpha_0 (1 - x).$$

Thus at the two equilibria we have (with a self-evident notation)

$$A_0 = 0, \quad B_0 = \alpha_0; \quad A_* = -\alpha_0, \quad B_* = 0.$$

It descends that

$$R_0 = \left| \frac{A_0}{B_0} \right| = 0, \quad R_* = \left| \frac{A_*}{B_*} \right| = \infty. \quad (3.23)$$

This shows, in view of our general discussion, that the equilibrium x_* corresponding to the carrying capacity does not change its stability, whatever the delay; on the other hand, the trivial equilibrium x_0 , which is unstable for zero delay, changes stability and hence becomes stable for

$$\delta = \delta_0 = \frac{\pi}{\alpha_0};$$

the critical frequency ν at this bifurcation is given by

$$\nu_0 = \pm \alpha_0.$$

Exercise. Analyze the stability of equilibria for the delay equation

$$\frac{dx}{dt} = \alpha_0 (1 - \hat{x}) x := f(x, \hat{x}). \quad (3.24)$$

Exercise. Analyze the stability of equilibria for the delay generalized logistic equation

$$\frac{dx}{dt} = \alpha_0 (1 - x)^k \hat{x} := f(x, \hat{x}). \quad (3.25)$$

3.3 Bibliography

Population dynamics with delay is discussed e.g. in Murray, which we followed here. A general discussion of equations with delay is provided e.g. in the books by Erneux or by Smith. The book by Gopalsamy deal specifically with these in Population Dynamics.

We mentioned that in order to study delay equations one should actually discuss functional differential equations. For these, see e.g. Hale & Lunel.

- J. Murray, *Mathematical Biology*, Springer 2001
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- H.L. Smith, *An introduction to delay differential equations with applications to the life sciences*, Springer 2011
- K. Gopalsamy, *Stability and oscillations in delay differential equations of population dynamics*, Springer 2013
- J.K. Hale & S.M.V. Lunel, *Introduction to functional differential equations*, Springer 2013

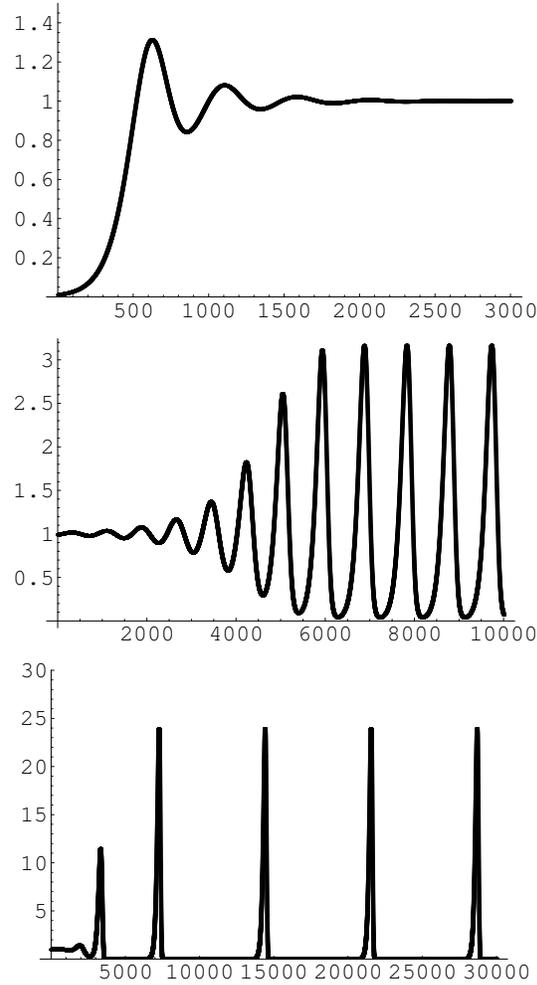


Figure 3.1: Numerical integration of equation (3.24), for $A = 1$ and for different values of the delay δ , denoted here as ϑ to avoid confusion. The horizontal axis measures time in units of the numerical integration step $\delta t = 0.01$. Upper, $\vartheta = \pi/3$ (with initial condition $x(t) = 0.01$ for $t < 0$); in this case the equilibrium at $x = 1$ is still stable, and the system approaches equilibrium through smaller and smaller oscillations. Center, $\vartheta = (2/3)\pi$ (with initial condition $x(t) = 0.99$ for $t < 0$); in this case the equilibrium $x = 1$ is unstable, and the system goes away from it, reaching a stable (regular, albeit not a simply harmonic) oscillation dynamics. Lower, $\vartheta = (4/3)\pi$ (with initial condition $x(t) = 0.99$ for $t < 0$); in this case the equilibrium $x = 1$ is still unstable and the system goes away from it reaching a stable oscillation dynamics with period and oscillation amplitude definitely larger than in the previous case. In this case the oscillations are definitely non-harmonic, and actually one observes periodic *bursts*.

Chapter 4

The Lotka-Volterra model

So far we have discussed the dynamics of a single population. In real world, populations are interacting, and we should consider interacting populations. The simplest, first and most famous model tackling this task is the *Lotka-Volterra model*, which we will discuss in this Chapter.

This model takes the name of Alfred J. Lotka (1880-1944), born in L'vov (then in the Austrian empire, nowadays in Ukraine), and of Vito Volterra (1860-1940), born in Ancona (then in the Church state, nowadays in Italy).¹

4.1 The Lotka-Volterra model

We will consider two populations, one feeding on the other. We will refer to these as Preys and Predators, and denote their consistence at time t as $x(t)$ and $y(t)$ respectively.

We will describe their dynamics as the dynamics of isolated populations plus the effect of interaction.

In the absence of predators, preys – who feed on the environment – would grow according to the logistic model. If we assume that the presence of predators, rather than the scarcity of resources, is the limiting factor, we can model the dynamic of preys in the absence of predators as a Malthusian growth (the limiting counter term will be introduced in a moment), thus of type

$$\dot{x} = A x .$$

On the other hand, in the absence of preys, predators would not have any food, thus become extinct. We will describe their dynamics as an isolated population

¹The work on this subject by Lotka was first published by him in a book titled *Elements of Physical Biology* (1925), and then reprinted in 1956 under the title *Elements of Mathematical Biology*. The work by Volterra first appeared in two papers in 1926, and then in a comprehensive book in 1931. See the Bibliography at the end of the Chapter for exact references.

as a linear but decreasing dynamics, thus of type

$$\dot{y} = -B y .$$

Note that with this notation we are implicitly choosing to consider all parameters as positive, and introduce explicitly the signs entering in our equations.

Now we should introduce the interaction terms; we will of course have this entering with a minus sign in the equation for prays, and with a plus sign in that for predators. But the problem is what functional form we should choose for them.

Let us see things from the point of view of prays: if there is a close encounter with a predator, this will try to capture (i.e. eat) the pray, and this will happen with some probability that depends on the speed of the two actors, on how close was the encounter, and on random factors. But in the whole, any encounter leads to capture with some probability. On the other hand, the probability of encounter itself will depend on the concentration of predators, thus be directly proportional to the number $y(t)$ of predators. This was from the point of view of a single pray; when we consider the situation for the whole pray population, we get that the number of captures (per time interval δt^2) is proportional to both $x(t)$ and $y(t)$; that is, is of the form

$$\alpha x(t) y(t) \delta t .$$

Similarly, for the predators the possibility of reproduction rests eventually of the availability of food (i.e. of prays) and reasoning in the same way we get that the number of new births in the predators population (in a time interval δt) is proportional – obviously, with a rather different proportionality coefficient – to both x and y , i.e. is of the form

$$\beta x(t) y(t) \delta t .$$

Thus in the end our coupled equations for the predator-pray system are

$$\begin{cases} \dot{x} = Ax - \alpha xy & , \\ \dot{y} = -By + \beta xy & . \end{cases} \quad (4.1)$$

These are the *Lotka-Volterra equations* (in the following, we will denote them as LVE).

They are conveniently rewritten, collecting an x and an y factor in the r.h.s. of the two equations respectively, as

$$\dot{x} = (A - \alpha y) x \quad (4.2)$$

$$\dot{y} = -(B - \beta x) y . \quad (4.3)$$

Thus we see that they are still of the logistic type, but now for each population the growth rate depends on the level reached by the *other* population.

²Provided this is small enough.

4.1.1 Equilibria and stability

A simple analysis of the LV equations shows that there are only two equilibrium points $p_e = (x_e, y_e)$:

$$p_0 = (0, 0), \quad p_* = (B/\beta, A/\alpha). \quad (4.4)$$

As all of our parameters are positive, we see immediately that the nontrivial equilibrium p_* corresponds to a biologically acceptable situation, i.e. the populations $x_* = B/\beta$ and $y_* = A/\alpha$ are both positive.

In order to study the stability of these equilibria, we consider the linearization

$$\frac{d}{dt} \begin{pmatrix} x \\ y \end{pmatrix} = M_e \begin{pmatrix} x \\ y \end{pmatrix}$$

of the LVE. By elementary computations,

$$M_e = \begin{pmatrix} A - \alpha y_e & -\alpha x_e \\ \beta y_e & -(B - \beta x_e) \end{pmatrix}.$$

Thus the matrix M at the two equilibrium points is given respectively by

$$\begin{aligned} M_0 &= \begin{pmatrix} A & 0 \\ 0 & -B \end{pmatrix}; \\ M_* &= \begin{pmatrix} A - \alpha(A/\alpha) & -\alpha(B/\beta) \\ \beta(A/\alpha) & -(B - \beta(B/\beta)) \end{pmatrix} = \begin{pmatrix} 0 & -B(\beta/\alpha) \\ A(\alpha/\beta) & 0 \end{pmatrix}. \end{aligned}$$

The stability of equilibria p_e is controlled by the eigenvalues $\lambda_i^{(e)}$ of the linearization matrix M_e ; more precisely, by the real part of these eigenvalues: if all of these real parts are negative, the equilibrium is stable, if at least one of them is positive the equilibrium is unstable.

For M_0 there is nothing to discuss: we obviously have

$$\lambda_1^{(0)} = A, \quad \lambda_2^{(0)} = -B,$$

and we have a saddle point. Note that this corresponds to the behavior when we have only one population: the dynamics around p_0 is expanding in the x direction, contracting in the y direction.

The analysis is also very simple for p_* . In fact, we have

$$\text{Tr}(M_*) = 0, \quad \text{Det}(M_*) = AB,$$

and it follows immediately that

$$\lambda^{(*)} = \pm i\omega, \quad \omega := \sqrt{AB}.$$

Thus p_* is, at least at the lineal level, a *center*.

4.1.2 Trajectories and full solutions

Actually we can describe the solutions also far from the equilibria. In order to do this, let us consider the equation for *trajectories*, i.e.

$$\frac{dy}{dx} = \frac{dy/dt}{dx/dt} = \frac{-(B - \beta x) y}{(A - \alpha y) x} . \quad (4.5)$$

This is a separable equation, and is easily integrated³, yielding

$$I(x, y) := (Ax + By) - (A \log x + B \log y) = K . \quad (4.6)$$

Thus the function $I(x, y)$ is a constant of motion (i.e. a conserved quantity) for the dynamics of the LVE, and the motion remains on the level set $I(x, y) = K$ determined by the initial conditions $(x(0), y(0))$.

4.2 Structural instability of the Lotka-Volterra equations

The presence of a conserved quantity is rather surprising: in fact, there is no reason the dynamics should conserve some quantity, even less this one, which has no special meaning and is not related to any qualitative property of the system.

Actually, we noted before that the LVE make sense only because we know that predators will act as a limiting factor for the growth of the preys; but the dynamics in itself is not well defined, in that on the line $y = 0$ we have an exponential, thus unlimited, growth.

The obvious cure for this problem is to introduce a logistic term taking care of limiting the growth of the preys even in the absence of predators. With this, we have the "logistic Lotka-Volterra equations"

$$\begin{cases} \dot{x} = Ax - \alpha xy - \mu x^2 & , \\ \dot{y} = -By + \beta xy & . \end{cases} \quad (4.7)$$

If we try to determine trajectories of this, we get the equation

$$\frac{dy}{dx} = \frac{dy/dt}{dx/dt} = \frac{-(B - \beta x) y}{(A - \alpha y - \mu x) x} . \quad (4.8)$$

This is *not* separable; thus we cannot integrate it by separation of variables and we do not have a conserved quantity.

Actually, in this case we have an unstable nontrivial equilibrium and an attracting periodic orbit; we will now proof this assertion.

First of all, we study equilibria. We observe that we still have the trivial equilibrium $p_0 = (0, 0)$, and this is still a saddle point. Next, we do now also

³The integration – by separation of variables, indeed – is left to the reader as an easy exercise.

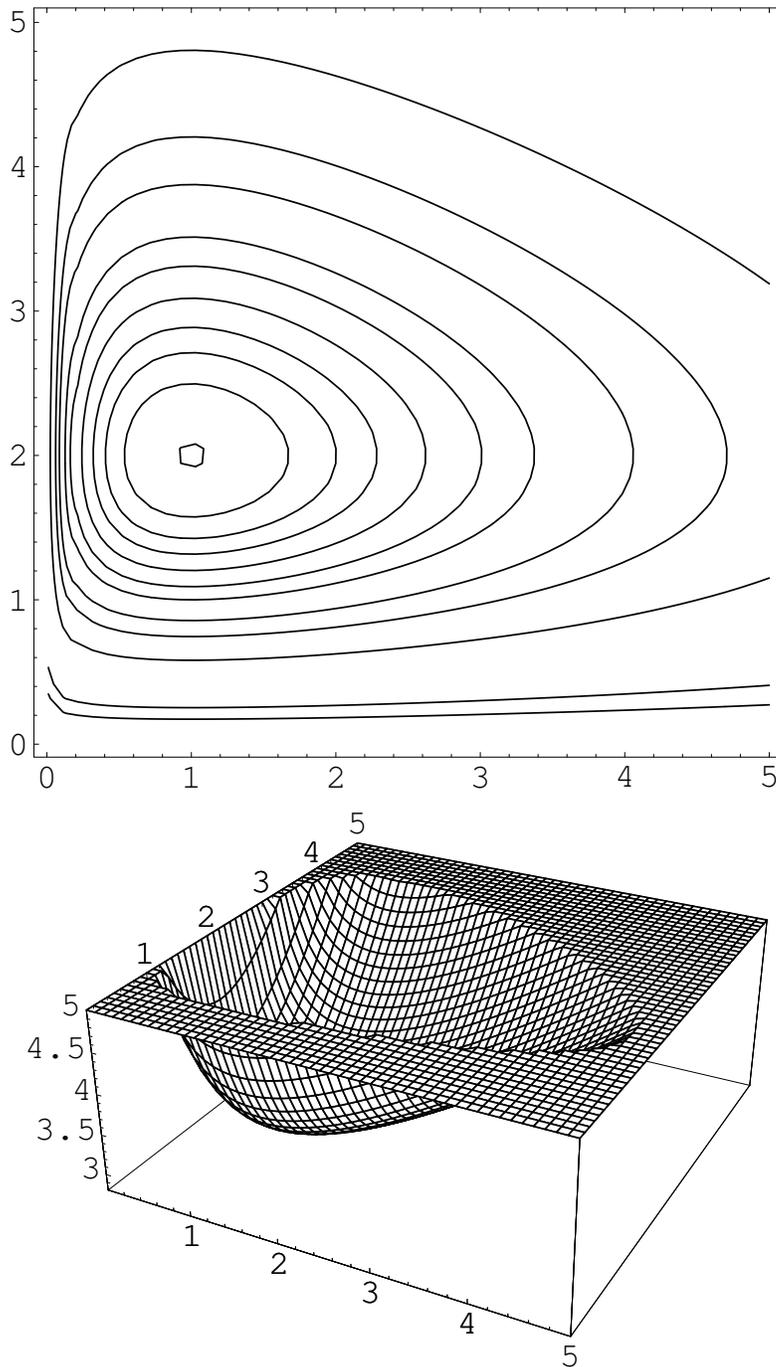


Figure 4.1: Level sets and three-dimensional plot for the function $I(x, y)$ given by (4.6). To ensure better readability, only values up to some upper limit are displayed.

have a partially trivial (in that it does not correspond to coexistence of the two species) equilibrium point at

$$p_x = (\alpha/\mu, 0) .$$

Finally, we have the nontrivial equilibrium

$$p_* = \left(\frac{B}{\beta}, \frac{A\beta - B\mu}{\alpha\beta} \right) .$$

Needless to say, this is acceptable only under the condition

$$A\beta > B\mu ; \tag{4.9}$$

in the following we assume this is satisfied.

The linearization of the dynamics at the equilibrium $p_e = (x_e, y_e)$ is given by

$$M = \begin{pmatrix} A - 2\mu x_e - \alpha y_e & -\alpha x_e \\ \beta y_e & -B + \beta x_e \end{pmatrix} ;$$

at the different equilibria we have

$$\begin{aligned} M_0 &= \begin{pmatrix} A & 0 \\ 0 & -B \end{pmatrix} , \\ M_x &= \begin{pmatrix} -A & -A(\alpha/\mu) \\ 0 & -B + A(\beta/\mu) \end{pmatrix} , \\ M_* &= \begin{pmatrix} -B(\mu/\beta) & -B(\alpha/\beta) \\ (A\beta - B\mu)/\alpha & 0 \end{pmatrix} . \end{aligned}$$

It is obvious that M_0 is a saddle point – the unstable and the stable manifolds being respectively the x and the y axes – while for the other equilibria some simple computations are needed.

The eigenvalues of M_x turn out to be

$$\lambda_1^{(x)} = -A , \quad \lambda_2^{(x)} = A(\beta/\mu) - B .$$

Under condition (4.9), we have $\lambda_2^{(x)} > 0$, and hence p_x is also a saddle point.

Finally, the eigenvalues of M_* are

$$\lambda_{\pm}^{(*)} = \frac{-B\mu \pm \sqrt{B(B\mu(4\beta + \mu) - 4A\beta^2)}}{2\beta} .$$

If, looking back at (4.9), we write

$$A = B(\mu/\beta) + \gamma^2$$

, these read as

$$\lambda_{\pm}^{(*)} = -\frac{B\mu}{2\beta} \left[1 \pm \sqrt{1 - 4\frac{\beta^2\gamma^2}{B\mu^2}} \right] .$$

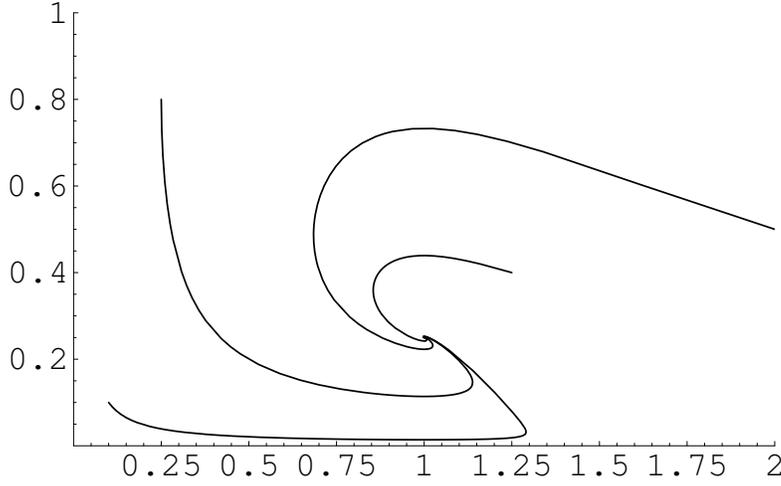


Figure 4.2: Dynamics of the logistic Lotka-Volterra equation. We plot the flow of eq.(4.7) originating from several initial conditions for the parameters value $A = 1$, $B = 1$, $\alpha = 1$, $\beta = 1$, $\mu = 3/4$; with these choices, condition (4.9) is satisfied, and $\lambda_{\pm}^{(*)} \approx -0.375 \pm 0.33i$. Note that all trajectories fall into the coexistence equilibrium point $p_* = (1, 1 - \mu) = (1, 1/4)$. The initial points q_i used in this plot are: $q_1 = (2, 0.5)$, $q_2 = (0.25, 0.8)$, $q_3 = (0.1, 0.1)$, and $q_4 = (1.25, 0.4)$.

That is, again under the assumption (4.9) – which guarantees γ is real – these eigenvalues can be either real or complex conjugate depending on the values of the parameters, but in all cases have negative real part. Thus, when it exists, the coexistence equilibrium p_* is stable.⁴

4.3 Bibliography

The original works by Lotka and Volterra are mentioned below; we take the occasion to point out also the PNAS note by Lotka on natural selection, actually dealing with a predator-prey system and remarkably without a single equation in it. As usual, the book by Bacaer is the source of a wealth of historical information. The Lotka-Volterra model is dealt with in virtually any book on Mathematical Biology or Population Dynamics, and we mention here the usual favorite ones in this (large) set.

- A. J. Lotka, *Elements of Physical Biology* (1925); reprinted by Dover in 1956 as *Elemental of Mathematical Biology*

⁴Note that when (4.9) is *not* satisfied, the equilibrium p_* does not exist, and p_x is stable.

- A. J. Lotka, "Natural selection as a physical principle", *Proc. Natl. Acad. Sci. USA* **8** (1922), 151-154
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Chapter 5

Competition, cooperation, hostility

In chapter ?? we considered the Lotka-Volterra model; in this model the interaction among species – or more precisely the groups of species – consists in predation. In Nature there are different kinds of interactions as well, and while predation is obviously detrimental to the preys and advantageous to predators, more nuanced situations are possible.

In this chapter we will consider these more general cases, focusing again on the case of only two species. The discussion conducted in chapter ?? about the extension of Lotka-Volterra model to higher dimension¹ (i.e. to a larger number of interacting groups) should be a sufficient reason to be cautious about the general relevance of the precise results obtained here.

5.1 Inter-species and intra-species competition

We start going back to the LV model, and focusing on the meaning of the parameters appearing in it.

We recall that in the Malthusian (exponential) growth model, the population evolves according to

$$\frac{dp}{dt} = \alpha p ; \quad (5.1)$$

the parameter α is the *growth coefficient*.

According to the logistic model, eq.(5.1) should be replaced by an equation taking into account the limitedness of the carrying capacity p_* of the environment. We characterized the model through the simplest equation implementing this feature, i.e.

$$\frac{dp}{dt} = \beta p (p_* - p) ; \quad (5.2)$$

¹In effetti li' non era proprio LV, mi sa che va spostato qui...

this yields $p'(t) > 0$ for $0 < p < p_*$, and $p'(t) < 0$ for $p > p_*$. We found convenient to see (5.2) as a version of eq.(5.1) in which the growth rate is not a constant, but depends on the level reached by p . This requires, comparing the two equations,

$$\alpha = \beta(p_* - p) = \beta p_* \left(1 - \frac{p}{p_*}\right). \quad (5.3)$$

We denote by α_0 the value of α when competition for resources is not present (i.e. in the limit $p \rightarrow 0$); thus $\alpha_0 = \beta p_*$. With this notation (5.3) reads

$$\alpha = \alpha_0 - \beta p ; \quad \beta = \alpha_0/p_* . \quad (5.4)$$

We thus obtain that β measures the competition within individuals of the same population, or of the same species. It is the *intra-specific competition coefficient*.

When considering two populations with generic interactions – described anyway by quadratic terms – we should consider the model

$$\begin{cases} dx/dt = \alpha_1 x + a_{11}x^2 + a_{12}xy \\ dy/dt = \alpha_2 y + a_{21}xy + a_{22}y^2 \end{cases} . \quad (5.5)$$

Note that here we made no *a priori* assumption about the signs of the coefficients (it is in this sense that the model is generic).

We stress that in the absence of the other species the carrying capacities for the two species are, according to (5.5),

$$x_* = -\frac{\alpha_1}{a_{11}} ; \quad y_* = -\frac{\alpha_2}{a_{22}} . \quad (5.6)$$

Needless to say these make sense – within population modelling – only when they are positive.

We want now to use this “general” model for the interaction of two species to study situations different from the predators/preys setting. In particular, we are interested in the case where the two populations are in some sense very similar², and compete for the same resources; we will consider several subcases of this framework: namely, the case in which the two groups are in competition, the one in which they cooperate, and the one where they have mutual hostility. Each of these will correspond to certain assumptions on the relative value of the coefficients appearing in (5.5).

5.2 The principle of competitive exclusion

We will start considering the case where the two species – or in this case it will be better to speak of “groups” – are very similar, and compete for just the same resources.

²A specially interesting particular case is provided by the two groups corresponding to the same species but differ only for a character, due to a mutation.

Then our model will lead to a maybe unexpected conclusion, i.e. to the *principle of competitive exclusion*: when there is competition for the same resources, the species less adapted to the environment, no matter how small is its disadvantage, becomes extinct.

In particular, when the two “species” are actually variants of the same species differing for a genetical mutation, this provides a simple – actually, far too simple – way to understand Evolution in Darwin’s sense, and its idea of the “survival of the fittest”.³

In order both to emphasize the similarity of the two groups and to simplify our analysis, we will assume that they grow at the same rate when resources are abundant; that is,

$$\alpha_1 = \alpha_2 = \alpha > 0 .$$

With the same goal, we will moreover assume that they are equivalent in the inter-specific competition, i.e.

$$a_{12} = a_{21} = -\gamma < 0 .$$

We will however assume that one of the two, say y , is slightly better adapted; this is the sense that the carrying capacity of the environment is slightly higher for y than for x . By (5.6), and writing $a_{ii} = -\beta_i < 0$ (thus we assume $\beta_i > 0$), this means

$$\frac{\alpha}{\beta_2} > \frac{\alpha}{\beta_1}$$

and therefore, recalling $\alpha > 0$,

$$\beta_2 < \beta_1 .$$

We will thus write

$$\beta_1 := \beta ; \beta_2 = \beta - \delta .$$

With these notation, the model (5.5) reads

$$\begin{cases} dx/dt = \alpha x - \beta x^2 - \gamma xy \\ dy/dt = \alpha y - \gamma xy - (\beta - \delta)y^2 \end{cases} . \quad (5.7)$$

Note that now all coefficients are positive (that is, all signs are explicit) and $0 < \delta \ll \beta$. Moreover, for the model to make any sense, it is needed that both population may get to a level higher⁴ than one. Thus we also need

$$\alpha > \beta > \beta - \delta > 0 .$$

³In this respect, one should immediately note that when one considers not only the “mean” flows, but also random fluctuations, it may very well happen that the less adapted variants survives and the better adapted one becomes extinct; in particular this may easily be the case when the better adapted variants arise due to a mutation, so initially with an extremely small population in the background of the dominating genome. We will discuss this at length in the following.

⁴Actually, much higher: we are considering average behaviors and for this to make sense from the modelling point of view we need that the average is taken over large populations, so that fluctuations may be overlooked

As for the coefficient γ , it is – at least at first – natural to assume it is intermediate between a and β , i.e. between β and $\beta - \delta$:

$$\beta - \delta < \gamma < \beta .$$

This relation is also expressed writing

$$\gamma = \beta - \nu \delta , \quad 0 < \nu < 1 . \quad (5.8)$$

5.2.1 Coexistence equilibrium

We say that an equilibrium $p_e = (x_e, y_e)$ is a coexistence equilibrium if both x_e and y_e are nonzero. The coexistence equilibria for (5.7) are given by

$$\begin{cases} \alpha - \beta x - \gamma y = 0 ; \\ \alpha - \gamma x - (\beta - \delta)y = 0 . \end{cases}$$

In other words, they are at the intersection of the two lines

$$y = \frac{\alpha}{\gamma} - \frac{\beta}{\gamma}x ; \quad y = \frac{\alpha}{\beta - \delta} - \frac{\gamma}{\beta - \delta}x .$$

With simple computations we obtain that this intersection is given by

$$(x_e, y_e) = \left(\frac{\alpha(\beta - \delta - \gamma)}{\beta^2 - \beta\delta - \gamma^2} , \frac{\alpha(\beta - \gamma)}{\beta^2 - \beta\delta - \gamma^2} \right) . \quad (5.9)$$

Requiring that these are both positive amounts to the condition

$$\begin{cases} (\beta - \delta - \gamma) > 0 , \beta^2 - \beta\delta - \gamma^2 > 0 , (\beta - \gamma) > 0 & \text{or} \\ (\beta - \delta - \gamma) < 0 , \beta^2 - \beta\delta - \gamma^2 < 0 , (\beta - \gamma) < 0 & . \end{cases} \quad (5.10)$$

In the following we assume this condition is satisfied.

We should still investigate the stability of p_e . In order to do this, we consider the linearization of (5.7) around p_e , and compute its eigenvalues. These turn out to be

$$\lambda_1 = -\alpha ; \quad \lambda_2 = -\alpha \frac{(\beta - \gamma)(\beta - \gamma - \delta)}{\beta^2 - \beta\delta - \gamma^2} . \quad (5.11)$$

Thus both eigenvalues are real, with λ_1 always negative; but the sign of λ_2 is not immediately determined. It follows from (5.10) that the numerator of the fraction expressing λ_2 is always positive, while its denominator can take different signs depending on the values of β , γ , and δ .

The reader has surely noted that we performed our computation for a general value of γ , i.e. without using (5.8).⁵

⁵The reason for this is of course that we wish to consider more general situations later on, and in this way we will be able to re-use our computations so far.

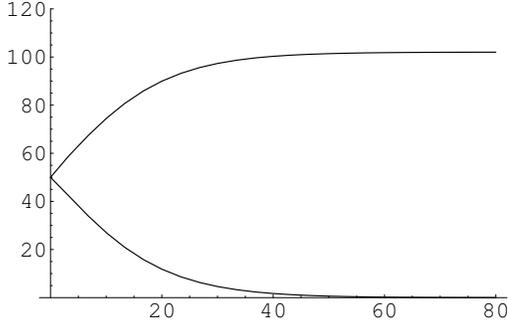


Figure 5.1: Numerical integration of (5.7) for $\alpha = 10$, $\beta = 0.1$, $\gamma = 0.099$, $\delta = 0.002$, with initial condition $x(0) = y(0) = 40$. In this case the carrying capacities are $x_* = 100$, $y_* = 102$. Albeit y has a modest 2% advantage, the x population becomes extinct.

5.2.2 Competition

When we impose (5.8), i.e. insert this into (5.9), we get

$$(x_e, y_e) = \left(\frac{\alpha(1-\nu)}{\beta(1-2\nu) + \delta\nu^2}, \frac{-\alpha\nu}{\beta(1-2\nu) + \delta\nu^2} \right). \quad (5.12)$$

We note the denominator appearing in the two fractions is just the same, and it follows from (5.8) that the two numerators have opposite sign.⁶

In other words, if (5.8) is satisfied, it is *not* possible to have a stable coexistence equilibrium. In this case, one of the two species become extinct, whatever the initial condition.

A simple analysis of the non-coexistence equilibria show that – again under the assumption (5.8) is satisfied – it is always the less adapted one to disappear, and this no matter how largely prevalent it may be at time $t = 0$.

5.2.3 Cooperation

As anticipated, the simple model (5.5) also allows to study the case where there is *cooperation* between the two groups: each of them, in the presence of the other, can reach a stable population *higher* than when it lives alone.

In terms of (5.5), in this case we have $a_{ii} := -\beta_i < 0$, and $a_{12} = \gamma_1 > 0$, $a_{21} = \gamma_2 > 0$. With these notations, (5.5) reads

$$\begin{cases} dx/dt = \alpha_1 x - \beta_1 x^2 + \gamma_1 xy \\ dy/dt = \alpha_2 y + \gamma_2 xy - \beta_2 y^2 \end{cases} . \quad (5.13)$$

We have three different possible cases:

⁶We reach the same conclusion considering $y_e/x_e = \nu/(\nu-1)$, and recalling that $0 < \nu < 1$.

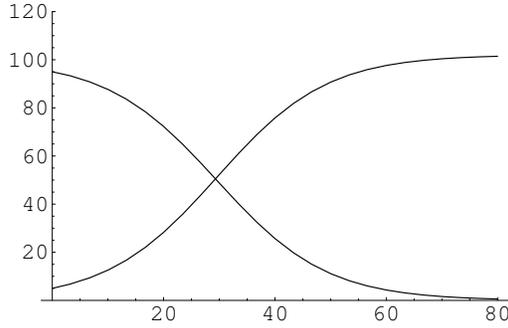


Figure 5.2: Numerical integration of (5.7) for $\alpha = 10$, $\beta = 0.1$, $\gamma = 0.099$, $\delta = 0.002$ (as in the previous figure 5.1), with initial condition $x(0) = 95$, $y(0) = 5$. Again, population x becomes extinct, despite its strong initial predominance.

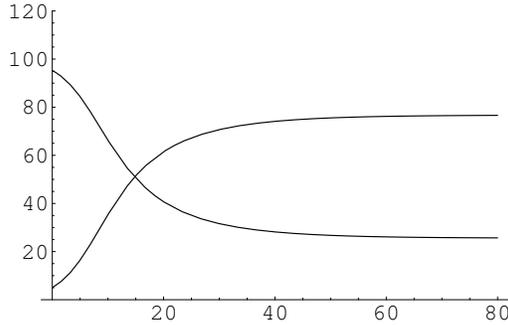


Figure 5.3: Numerical integration of (5.7) for $\alpha = 10$, $\beta = 0.1$, $\gamma = 0.097$, $\delta = 0.002$ (note that now $\gamma < \beta - \delta$) with initial condition $x(0) = 95$, $y(0) = 5$. In this case the carrying capacities are $x_* = 100$, $y_* = 102$. The coexistence equilibrium corresponds to $x = 26$, $y = 77$, and is stable, see (5.9) through (5.11). In fact, after a transient in which the population x has a rapid decrease and y increases rapidly too, equilibrium is reached.

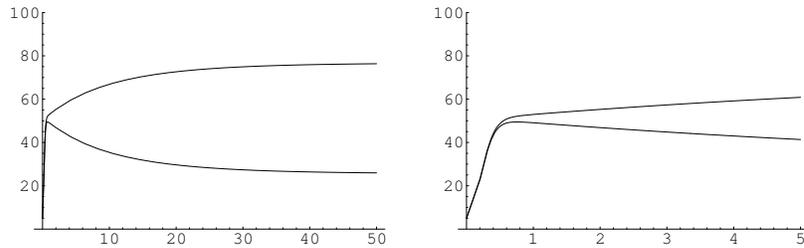


Figure 5.4: Left: numerical integration as in Fig. 5.3, but with initial condition $x(0) = y(0) = 5$. Right: detail of initial evolution. The two populations grow in essentially the same way until $x \simeq y \simeq 50$; afterwards the evolutions become substantially different.

- (a) The two populations can live also one in the absence of the other; this happens for $\alpha_i > 0$;
- (b) They can only live each in the presence of the other; this happens for $\alpha_i < 0$;
- (c) One of them, say x , can live alone, while the other, say y , needs the first one to survive; this happens for $(\alpha_1 > 0, \alpha_2 < 0)$.

The coexistence equilibrium is still corresponding to the intersection of the lines $(\alpha_1 + a_{11}x + a_{12}y) = 0$ and $(\alpha_2 + a_{21}x + a_{22}y) = 0$. With the notation used in (5.13), these are written as

$$(\alpha_1 - \beta_1 x + \gamma_1 y) = 0 ; \quad (\alpha_2 + \gamma_2 x - \beta_2 y) = 0 .$$

The intersection of these is in the point p_e of coordinates

$$x_e = \frac{\alpha_1 \beta_2 + \alpha_2 \gamma_1}{\beta_1 \beta_2 - \gamma_1 \gamma_2} , \quad y_e = \frac{\alpha_1 \gamma_2 + \alpha_2 \beta_1}{\beta_1 \beta_2 - \gamma_1 \gamma_2} . \quad (5.14)$$

Recall that now β_i and γ_i are positive, while the α_i do not have a definite sign; but they do in each of the different cases listed above, and we are now going to see what happens in each of these.

- In case (a) requiring $x_e > 0, y_e > 0$ amounts to requiring

$$\gamma_1 \gamma_2 < \beta_1 \beta_2 ; \quad (5.15)$$

- In case (b) the same requirement amounts to the opposite condition,

$$\gamma_1 \gamma_2 > \beta_1 \beta_2 ; \quad (5.16)$$

- Finally, in case (c) one should consider several sub-cases; we leave this to the reader.

Let us focus on case (a), and hence suppose (5.15) holds. The linearization of (5.13) at (x_0, y_0) is obtained writing $x = x_0 + \xi, y = y_0 + \eta$, and working at first order in the component of the vector $\zeta = (\xi, \eta)$. We obtain a linear system

$$\frac{d\zeta}{dt} = A \zeta$$

where the matrix A is

$$A = \begin{pmatrix} \alpha_1 - 2\beta_1 x_0 + \gamma_1 y_0 & \gamma_1 x_0 \\ \gamma_2 y_0 & \alpha_2 + \gamma_2 x_0 - 2\beta_2 y_0 \end{pmatrix} .$$

Choosing $(x_0, y_0) = (x_e, y_e)$, using (??), and writing for ease of notation

$$\Delta = (\beta_1 \beta_2 - \gamma_1 \gamma_2)^{-1} ,$$

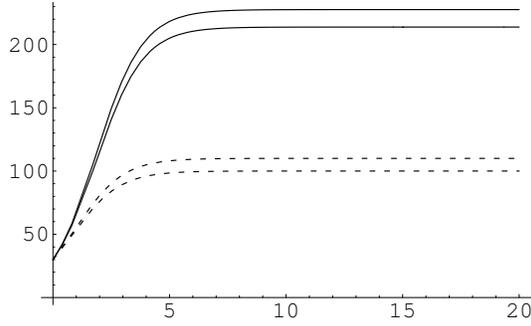


Figure 5.5: Numerical integration of (5.13) with $\alpha_1 = \alpha_2 = 1$, $\beta_1 = 1/100$, $\beta_2 = 1/110$, and $\gamma_1 = \gamma_2 = 0.005$, for different initial conditions. Dashed curves correspond to $x(0) = 30, y(0) = 0$, i.e. for the population x alone (lower curve); and to $x(0) = 0, y(0) = 30$, i.e. for the population y alone (upper curve). Solid curves represent the solutions for $x(t)$ (lower curve) and for $y(t)$ (upper curve) with initial condition $x(0) = 30, y(0) = 30$. The cooperative effect is clearly apparent.

we get

$$A_e = \Delta \begin{pmatrix} -\beta_1(\alpha_1\beta_2 + \alpha_2\gamma_1) & \gamma_1(\alpha_1\beta_2 + \alpha_2\gamma_1) \\ \gamma_2(\alpha_1\gamma_2 + \alpha_2\beta_1) & -\beta_2(\alpha_1\gamma_2 + \alpha_2\beta_1) \end{pmatrix}. \quad (5.17)$$

One can easily compute the eigenvalues of A , but these have some cumbersome expression; it is more convenient to consider the trace and determinant of A , which are

$$\begin{aligned} \text{Tr}(A_e) &= -\Delta (\alpha_2\beta_1(\beta_2 + \gamma_1) + \alpha_1\beta_2(\beta_1 + \gamma_2)); \\ \text{Det}(A_e) &= \Delta (\alpha_1\beta_2 + \alpha_2\gamma_1) (\alpha_2\beta_1 + \alpha_1\gamma_2). \end{aligned} \quad (5.18)$$

In our case, i.e. in case (a), all the parameters are positive, and (5.15) holds. It follows then from (5.15) that

$$\text{Tr}(A_e) < 0, \quad \text{Det}(A_e) > 0;$$

hence both eigenvalues are negative. In other words, the coexistence equilibrium (x_e, y_e) – when it exists, i.e. when (5.15) is verified – is stable and actually attractive.

Finally we note that for $y = 0$ the equilibrium level for x is given by $x_* = \alpha_1/\beta_1$; and for $x = 0$ the equilibrium level for y is given by $y_* = \alpha_2/\beta_2$. Looking back at (5.14), we note that

$$x_e > x_*, \quad y_e > y_* :$$

each of the two species has actually an advantage from the presence of the other.

Problems

Problema 1. Al termine della sezione 2 si è affermato che (come ragionevole attendersi, una volta mostrato che solo una specie sopravvive) è sempre la specie meglio adattata a sopravvivere. Dimostrare questa asserzione a partire dal modello (5.7) e dalla discussione svolta nella sezione 2.

[*Suggerimento:* mostrare che la specie più adattata y ha sempre $dy/dt > 0$ per y abbastanza piccolo ($0 < y < y_*$), quindi non può estinguersi.]

Problema 2. Linearizzare la dinamica del sistema (5.7) intorno al punto di equilibrio (5.9), verificando che essa è descritta dalla matrice

$$A_e = - \frac{1}{\beta^2 - \beta\delta - \gamma^2} \begin{pmatrix} \alpha\beta(\beta - \delta - \gamma) & \alpha\gamma(\beta - \delta - \gamma) \\ \alpha\gamma(\beta - \gamma) & \alpha(\beta - \delta)(\beta - \gamma) \end{pmatrix}.$$

Verificare inoltre che gli autovalori di questa matrice sono forniti dalla (5.11).

Problema 3. Con riferimento alla classificazione proposta all'inizio della sezione 3, studiare il caso (b) (in cui $\alpha_i < 0$) nell'approssimazione $\beta_i = 0$; si richiede in particolare di determinare esattamente (in questa approssimazione) la relazione tra x ed y prevista dal modello.

Problema 4. Determinare la velocità di approccio all'equilibrio (x_e, y_e) per il sistema (5.13) nel caso (a).

[*Suggerimento:* questa è determinata dagli autovalori della matrice (5.17).]

Problema 5. Nel quadro della discussione della sezione 3, definiamo il *vantaggio cooperativo* come la differenza tra la taglia della popolazione all'equilibrio in presenza dell'altra popolazione e la stessa taglia in assenza dell'altra popolazione. Con le notazioni introdotte sopra, questo è $\Delta_x = (x_e - x_*)$ per la popolazione x , $\Delta_y = (y_e - y_*)$ per la popolazione y . Mostrare che (sempre nel caso (a) della classificazione) $\Delta_x = (\gamma_1/\beta_1)(\alpha_2\beta_1 + \alpha_1\gamma_2)\Delta$, $\Delta_y = (\gamma_2/\beta_2)(\alpha_1\beta_2 + \alpha_2\gamma_1)\Delta$.

Problema 6. Considerare la discussione condotta nella sezione 2 rinunciando all'assunzione (5.8); in particolare, mostrare che per $\gamma < \beta - \delta$ si può avere un equilibrio stabile tra i due tipi, come mostrato dalle figure 3 e 4.

5.3 Bibliography

As mentioned above, the principle of competitive exclusion was apparently first stated by Gause; it should be noted that he was not a mathematician but a biologist; and not just a theorist, but also conducted experiments (on yeast) to support his analysis (we only quote one of his many experimental papers on the subject). An analysis of the genesis of the principle – which could perhaps be called the Gause-Lotka-Volterra principle – is given in the paper by Hardin. The principle is discussed in most books on Mathematical Biology, including those we already quoted several times.

- G.F. Gause, *The Struggle for Existence: A Classic of Mathematical Biology and Ecology*, reprinted by Dover 2019
- G.F. Gause, “Experimental analysis of Vito Volterra’s mathematical theory of the struggle for existence”, *Science* **79** (1934), 16-17
- G. Hardin, “The competitive exclusion principle”, *Science* **131** (1960), 1292-1297
- J.D. Murray, *Mathematical Biology. I: An Introduction*, Springer 2002
- T. Britton, *Essential Mathematical Biology*, Springer 2005
- L. Edelstein-Keshet, *Mathematical Models in Biology*, SIAM 2005

Chapter 6

Interacting population models in arbitrary dimension

The Lotka-Volterra model considers two interacting populations. In Nature, we never have just two populations, albeit we may be able to regroup the existing populations in two classes (e.g. preys and predators) for the sake of modeling. It is thus natural to wonder if, within the Lotka-Volterra type of models¹ it is possible to obtain results holding for an arbitrary number of interacting species. We are now going to discuss this matter, mainly following the book by Hofbauer and Sigmund.

6.1 Lotka-Volterra systems and equilibria

We should first of all specify what we mean by “LV systems”, i.e. what is the class of systems we are going to analyze. These will be systems describing the evolution of the populations of n species or groups, which we denote by $x_i(t)$ ($i = 1, \dots, n$) of the form (no sum on the i index here)

$$\frac{dx_i}{dt} = a_i x_i + x_i \sum_j b_{ij} x_j . \quad (6.1)$$

Thus, the interactions among different species – and also the “self-interactions” of each species with itself – are described by *quadratic* terms.

The interaction coefficients b_{ij} can be grouped in a matrix B with elements $B_{ij} = b_{ij}$, and this will be denoted as the *interaction matrix*. Similarly, the raw growth coefficients a_i can be collected in a vector $\mathbf{a} = (a_1, \dots, a_n)$.

¹Whatever this means; this point will be discussed below.

We note that now all the coefficient can have any sign; we will suppose that $a_i \neq 0$ for all the $i = 1, \dots, n$, while some of the elements of B may very well vanish.

In principles, the system (6.1) is defined in \mathbf{R}^n ; but from the biological point of view we are only interested in its behavior for $x_i \geq 0$, i.e. in $\mathcal{B} = (\mathbf{R}_+)^n$. It is obvious that if the initial datum $\mathbf{x}(0)$ satisfies $\mathbf{x}(t) \in \mathcal{B}$, we will have $\mathbf{x}(t) \in \mathcal{B}$ for all $t \geq 0$.

We also note that if we restrict the system (6.1) to a subspace in which some of the x_i are set equal to zero², we still have a system of the same general form (6.1).

Now, let us consider the stationary solutions (if any) of the system (6.1). From the biological point of view, it is interesting to know if all the initially present species will still be present in some proportion at the equilibrium, or is some of them will disappear³. If some species are actually absent, this means their population(s) is (are) $x_i = 0$, so the system ends up being on the frontier $\partial\mathcal{B}$ of the admitted phase space $\mathcal{B} = \mathbf{R}_+^n$. We say that an equilibrium in which all species are present is an *internal equilibrium point*, as it belongs to the interior of \mathcal{B} .

In general, an equilibrium is a solution to

$$x_i \left(a_i + \sum_j b_{ij} x_j \right) = 0 . \quad (6.2)$$

If we only consider internal equilibria, we rule out the solutions with some $x_i = 0$, and we just have to consider the system

$$a_i + \sum_j b_{ij} x_j = 0 ; \quad (6.3)$$

this is readily rewritten in vector/matrix form, with an obvious notation, as

$$B \mathbf{x} = -\mathbf{a} . \quad (6.4)$$

If B is invertible, then (6.3) admits a single solution,

$$\mathbf{x} = -B^{-1} \mathbf{a} . \quad (6.5)$$

Note that this is acceptable – in view of the meaning of our variables and of our hypothesis – only if $\mathbf{x} \in \mathcal{B}$, and actually only if \mathbf{x} belongs to the interior of \mathcal{B} . In fact, as we made no hypotheses on the signs of the components of \mathcal{B} and of \mathbf{a} , we are by no means guaranteed that the components of \mathbf{x} defined by (6.5) are all positive.

Problem. Discuss the degenerate case where B has vanishing determinant.

²The reader can easily check this is a *positively invariant subspace*.

³As remarked above, the analysis of a situation in which one or more of the species are actually absent reduces to a mathematical problem of the same type.

6.2 Food chains

We can get some general result about *food chains*, i.e. in the case where the first species feeds on resources present in the environment, but each subsequent species feeds on the previous one, up to species n . This discussion will follow Hofbauer and Sigmund; they claim that in Nature this kind of chains exist for $n \leq 6$.

In this food chain case, we have many zero coefficients, and definite signs for the non-zero ones. In fact, as for the a_i , we have $a_1 > 0$ e $a_i < 0$ per $i > 1$. As for the matrix B , its diagonal terms (i.e. the coefficients b_{ii}) represent the competition among individuals of the same species, and are always negative: $b_{ii} < 0$. The off-diagonal terms are zero excepts for those in the diagonals next to the principal one, i.e. $b_{ij} = 0$ per $|i - j| > 1$; that is, B is a tri-diagonal matrix. As the species i is a prey for the species $i + 1$, we have $b_{i,i+1} < 0$ and $b_{i,i-1} > 0$.

In other words, the zeroes and the signs of the B matrix are as follows:

$$\begin{pmatrix} - & - & 0 & 0 & 0 & 0 \\ + & - & - & 0 & 0 & 0 \\ 0 & + & - & - & 0 & 0 \\ 0 & 0 & + & - & - & 0 \\ 0 & 0 & 0 & + & - & - \\ 0 & 0 & 0 & 0 & + & - \end{pmatrix}. \quad (6.6)$$

It is convenient to deal with positive coefficients, i.e. write down explicitly the signs we have just determined; we thus write $\alpha_i = |a_i|$, so that $\mathbf{a} = (\alpha_1, -\alpha_2, \dots, -\alpha_n)$; similarly we set $\beta_{ij} = |b_{ij}|$, hence B is of the form

$$B = \begin{pmatrix} -\beta_{11} & -\beta_{12} & 0 & 0 & 0 & 0 \\ +\beta_{21} & -\beta_{22} & -\beta_{23} & 0 & 0 & 0 \\ 0 & +\beta_{32} & -\beta_{33} & -\beta_{34} & 0 & 0 \\ 0 & 0 & +\beta_{43} & -\beta_{44} & -\beta_{45} & 0 \\ 0 & 0 & 0 & +\beta_{54} & -\beta_{55} & -\beta_{56} \\ 0 & 0 & 0 & 0 & +\beta_{65} & -\beta_{66} \end{pmatrix}. \quad (6.7)$$

Equations (6.1) are now written as

$$\begin{aligned} dx_1/dt &= (\alpha_1 - \beta_{1,1}x_1 - \beta_{1,2}x_2) x_1, \\ dx_h/dt &= (-\alpha_h + \beta_{h,h-1}x_{h-1} - \beta_{h,h}x_h - \beta_{h,h+1}x_{h+1}) x_h, \\ dx_n/dt &= (-\alpha_n + \beta_{n,n-1}x_{n-1} - \beta_{n,n}x_n) x_n. \end{aligned} \quad (6.8)$$

We will now proof that if (6.8) admits an internal equilibrium, this is stable and actually all solutions starting from the interior of \mathcal{B} are attracted to this.

It will be convenient to have a compact notation for (6.8); we write these as

$$dx_i/dt = f_i(\mathbf{x}) = \Psi_i(\mathbf{x}) x_i, \quad (6.9)$$

where of course $\Psi_i = a_i + \sum_j b_{ij} x_j$. The internal equilibrium \mathbf{x}_* is characterized by

$$\Psi_1(\mathbf{x}_*) = \Psi_2(\mathbf{x}_*) = \dots = \Psi_n(\mathbf{x}_*) = 0 . \quad (6.10)$$

We now consider the matrix with entries $\partial f_i / \partial x_j$ and evaluate it in \mathbf{x}_* . It follows immediately from (6.9) that

$$\frac{\partial f_i}{\partial x_j} = \frac{\partial \Psi_i}{\partial x_j} x_i + \delta_{ij} \Psi_i ; \quad (6.11)$$

in \mathbf{x}_* it is $\Psi_i = 0 \forall i = 1, \dots, n$. Moreover, the explicit form of the Ψ_i yields

$$\partial \Psi_i / \partial x_j = b_{ij} ,$$

hence

$$\left(\frac{\partial f_i}{\partial x_j} \right)_{\mathbf{x}_*} = (\mathbf{x}_*)_i b_{ij} := h_{ij} . \quad (6.12)$$

As by assumption $(\mathbf{x}_*)_i > 0 \forall i$, we conclude that the matrix H with entries h_{ij} is of the same type – for what concerns both the zeros and the signs – of the matrix B . We are thus led to consider again matrices of the type (6.7), and we should show that all of its eigenvalues are negative (or at least have negative real part). For this to be the case, it suffices to show that all of its principal minors of rank $k = 1, \dots, n$ are of alternating signs $(-1)^k$. The writing (6.7) shows immediately that this is indeed the case.⁴

We conclude that with our assumptions on the signs of coefficients, the internal equilibrium point \mathbf{x}_* is stable and asymptotically stable. Moreover, if some of the coefficients b_{ii} vanish, then \mathbf{x}_* is stable but not necessarily asymptotically stable; note that in this case we are not guaranteed \mathbf{x}_* is an isolated equilibrium.

In order to prove that (provided $b_{ii} \neq 0 \forall i$) \mathbf{x}_* is asymptotically stable requires to use a *Lyapounov function*; the appropriate one (which can also be used to analyze the degenerate case in which some of the b_{ii} vanish) is

$$V = \sum_i c_i [x_i - (x_*)_i \log x_i] , \quad (6.13)$$

where the c_i are positive constants defined by

$$c_1 = 1 , \quad c_{i+1} = c_i (\beta_{i,i+1} / \beta_{i+1,i}) . \quad (6.14)$$

In this case V evolves on the flow of (6.9) as

$$\frac{dV}{dt} = - \sum_i c_i \beta_{ii} [x_i - (x_*)_i]^2 \leq 0 . \quad (6.15)$$

⁴One should note that this statement holds provided that the element on the main diagonal are nonzero. If they are instead zero, then the principal minors of odd rank are null, while those of even rank are positive.

6.3 Models with three species

In the general case, i.e. when the n species are *not* organized in a food chain, the simple conclusions obtained in the previous section do not hold, and actually the system (6.1) can behave in virtually whatever way when $n \geq 3$: the case $n = 2$ is special in many ways, essentially because two-dimensional dynamical systems are very special, and different from generic ones – i.e. from those in dimension $n \geq 3$.

It may be interesting to consider the simplest “generic” case, i.e. interaction among three species. The general case, even within the three species setting, would lead to an exceedingly complicate classification. We will instead limit to consider the case of a “cyclic” interaction: A competes with B in the same way as B competes with C, and this is the same way as C competes with A.⁵

That is, the system we are going to consider is

$$dx_i/dt = a x_i - x_i \sum_j B_{ij} x_j := f_i(x), \quad (6.16)$$

where now the matrix B is

$$B = \begin{pmatrix} b_1 & b_2 & b_3 \\ b_3 & b_1 & b_2 \\ b_2 & b_3 & b_1 \end{pmatrix}. \quad (6.17)$$

The coefficients a, b_1, b_2, b_3 are assumed to be all positive, i.e. the interaction among the species is of competitive type. Changing the time scale we can always set $a = 1$; changing the units in which we measure populations we could also set one of the coefficients to unity.

The system (6.16) admits algebraically eight points of equilibrium; some of these might be trivial or non-acceptable; there is only one internal equilibrium point (we recall this means in the interior of $\mathcal{B} = \mathbf{R}_+^n$), given by

$$x_1 = x_2 = x_3 = \frac{a}{b_1 + b_2 + b_3}. \quad (6.18)$$

The Jacobian is

$$\frac{\partial f_i}{\partial x_j} = J = \begin{pmatrix} a - \chi_1 & -b_2 x_1 & -b_3 x_1 \\ -b_3 x_2 & a - \chi_2 & -b_2 x_2 \\ -b_2 x_3 & -b_3 x_3 & a - \chi_3 \end{pmatrix}, \quad (6.19)$$

where we have written, for graphical convenience,

$$\begin{aligned} \chi_1 &= 2b_1 x_1 + b_2 x_2 + b_3 x_3, \\ \chi_2 &= b_3 x_1 + 2b_1 x_2 + b_2 x_3, \\ \chi_3 &= b_2 x_1 + b_3 x_2 + 2b_1 x_3. \end{aligned}$$

⁵This should ring a bell to the reader familiar with *game theory*.

Evaluating this in the internal equilibrium point (6.18) we have

$$J_* = -\frac{a}{b_1 + b_2 + b_3} \begin{pmatrix} b_1 & b_2 & b_3 \\ b_3 & b_1 & b_2 \\ b_2 & b_3 & b_1 \end{pmatrix}. \quad (6.20)$$

The eigenvalues of J_* are

$$\lambda_0 = -a, \quad \lambda_{\pm} = \frac{1}{2(b_1 + b_2 + b_3)^2} \left(P \pm \sqrt{-Q} \right),$$

where we have written

$$\begin{aligned} P &= -a (2b_1^2 + b_1(b_2 + b_3) - (b_2 + b_3)^2), \\ Q &= 3a^2 (b_2 - b_3)^2 (b_1 + b_2 + b_3)^2. \end{aligned}$$

It is immediately apparent that $Q > 0$, so that $\lambda_{\pm} = \mu \pm i\nu$ with $\nu = \sqrt{Q}$. As for the real part μ , it is easy to check that it is negative (respectively, positive) for $b_1 < (b_2 + b_3)/2$ (respectively, for $b_1 > (b_2 + b_3)/2$).⁶

Thus, for $b_1 < (b_2 + b_3)/2$ the internal equilibrium point is stable and actually asymptotically stable, while for $b_1 > (b_2 + b_3)/2$ it is a saddle point and hence unstable.

When the internal equilibrium is unstable, one can have behaviors which are somewhat surprising from the point of view of population dynamics, albeit rather natural from the point of view of abstract dynamical systems. In fact, in this case the system drifts towards the frontier $\partial\mathcal{B}$, and the dynamics near to $\partial\mathcal{B}$ is a sequence of phases in which one of the populations is by far dominant, followed by a sharp transition in which this nearly disappears and its dominant role is taken by another one. This with an increasing “slow” dynamics, i.e. with pseudo-equilibrium phases which are longer and longer.⁷

We are in the presence of a problem already met in previous chapters: an observer who is not aware of these aspects, which are natural in – and well described by – the theory of dynamical systems, could believe that the “pseudo equilibrium” was a true ecological equilibrium, and that the following sharp transition was not something built in the natural dynamics of the system, but due to some sudden change in the environmental conditions, or to some “hidden” environmental catastrophe.

It is instead a behavior of *intermittent* type, and often nearly catastrophic⁸, entirely due to the internal dynamics of the system. This is specially deceiving

⁶Obviously this condition, obtained recalling that by assumption $a > 0$, could also be expressed singling out any other of the b_i coefficients; we would get as threshold value $b_2 = 2b_1 - b_3$ in terms b_2 , or $b_3 = 2b_1 - b_2$ in terms of b_3 .

⁷A proof of these statements is given in section 5.5 of Hofbauer and Sigmund. This model was proposed and studied by R. May and W. Leonard, “Nonlinear aspects of competition between three species”, *SIAM Journal of Applied Mathematics* **29** (1975), 243-252. See also A. Chenciner, “Comportement asymptotique de systèmes différentiels du type compétition d’espèces”, *Comptes Rendus Académie de Sciences de Paris* **284**, 313-315.

⁸In the technical sense, i.e. as a near phase transition.

in that it is *not* a periodic behavior, given that the time scale on which the sharp transitions occur becomes longer and longer.⁹

On the other hand, real populations are integer, i.e. require in particular $x_i \geq 1$; so at some point one of the populations will actually become extinct. At this point we have a two-species system, and the analysis of a previous chapter applies. In particular, if the principle of competitive exclusion applies, the less adapted species will in turn disappear.

Our discussion was conducted for three species, but similar results also applies for N species under the assumption that their interactions are “cyclic”. This assumption (which corresponds more precisely to assuming a symmetry of the system under cyclic permutation) allows to compute exactly the eigenvalues of J_* , the Jacobian at the internal equilibrium, but is not biologically realistic. The discussion given here is however, in our opinion, useful in that it presents a phenomenology which is surprising in several ways and which can occur also for systems which do not satisfy the cyclicity condition.

In order to concretely illustrate our three-species discussion, we consider numerical simulations choosing

$$a = 1; \beta_1 = 0.01, b_2 = 0.018, b_3 = 0.005 . \quad (6.21)$$

In Fig.6.1 we show a numerical integration of this system with initial condition

$$x_1(0) = 20 , x_2(0) = 10 , x_3(0) = 30 . \quad (6.22)$$

In Fig. 6.3 we show a numerical integration of the same system with a different initial condition,

$$x_1(0) = 60 , x_2(0) = 90 , x_3(0) = 30 . \quad (6.23)$$

It appears that, apart from differences in the initial transient behavior, the system reaches the same asymptotic dynamics.

⁹Reader familiar with the Physics of Matter may call this kind of behavior “glass-like”.

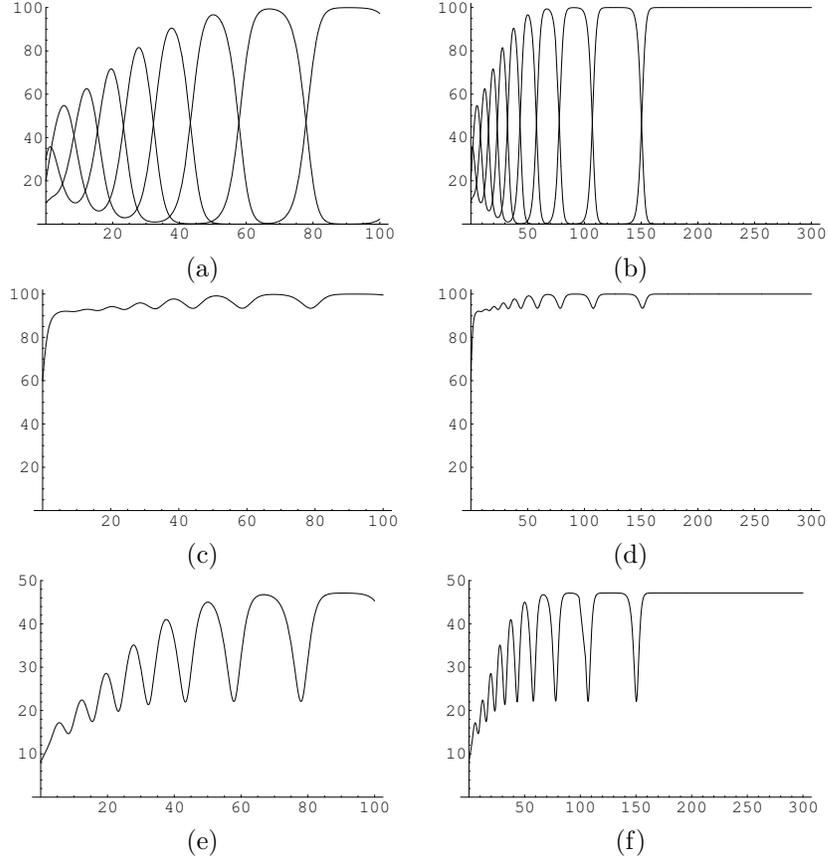


Figure 6.1: Numerical integration of (6.16) for parameters as in (6.21) and initial condition (6.22). (a): Variation of the three populations $x_i(t)$ for $t \in [0, 100]$; (b) same for $t \in [0, 300]$. (c) Variation of the total population $X(t) = x_1(t) + x_2(t) + x_3(t)$ for $t \in [0, 100]$; (d) same for $t \in [0, 300]$. (e) Dynamics of the dispersion $\langle x^2 \rangle - \langle x \rangle^2$ for $t \in [0, 100]$; (f) same for $t \in [0, 300]$.

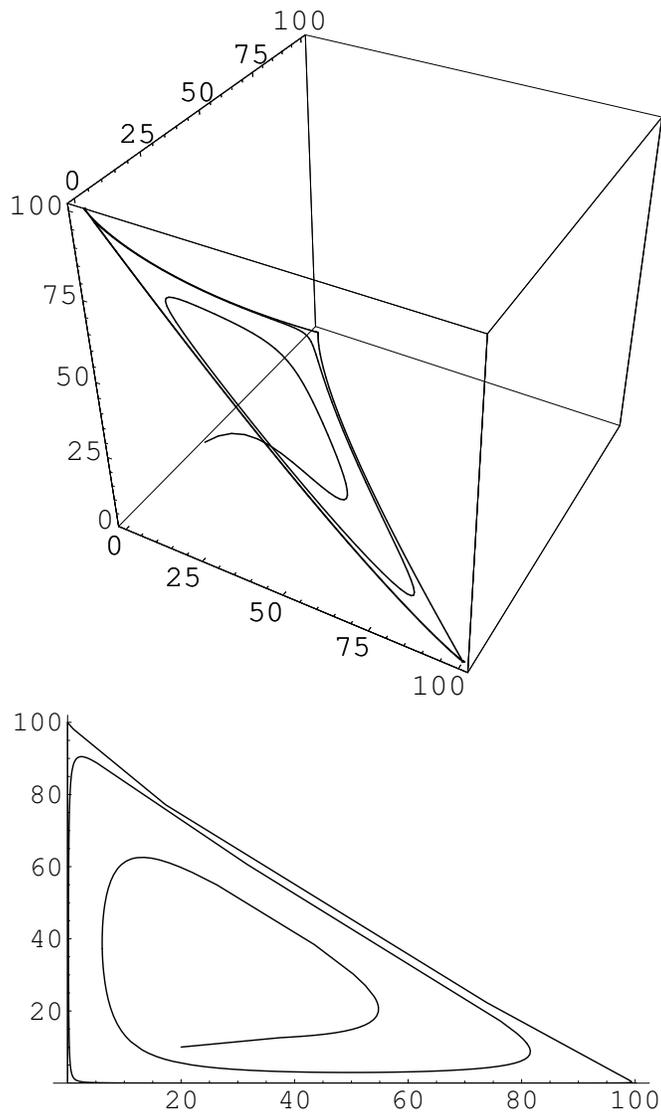


Figure 6.2: The data of Fig.6.1 are reported here through parametric plots, representing thus the trajectory of the system, in three dimensions (upper) and projecting them in the plane (x_1, x_2) (lower).

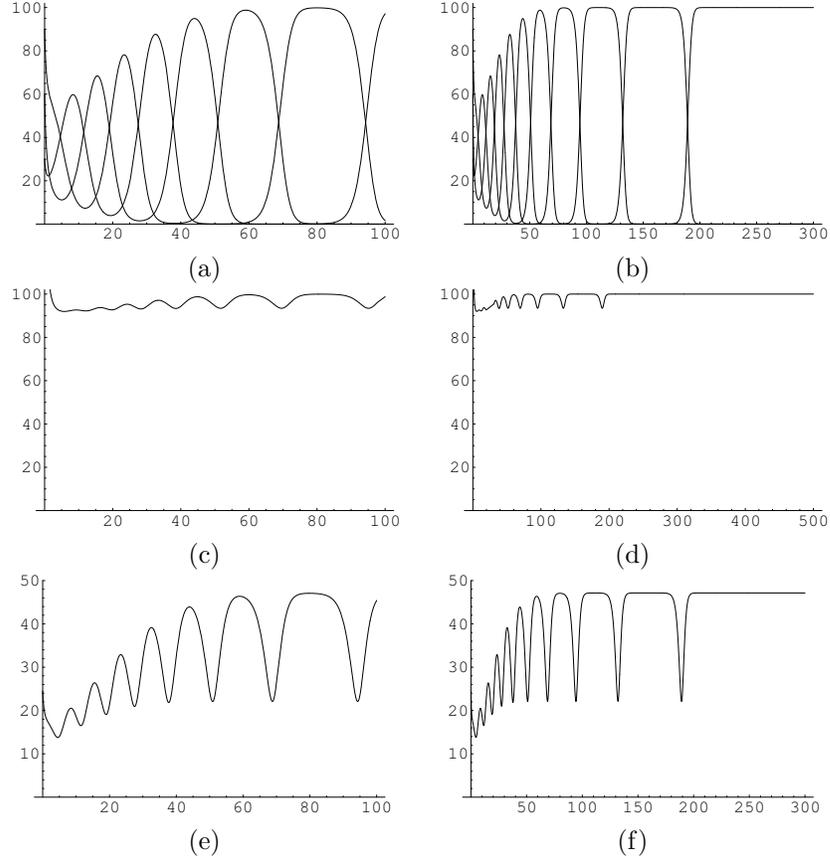


Figure 6.3: Numerical integration of (6.16) for parameters as in (6.21) and initial condition (6.23). (a): Variation of the three populations $x_i(t)$ for $t \in [0, 100]$; (b) same for $t \in [0, 300]$. (c) Variation of the total population $X(t) = x_1(t) + x_2(t) + x_3(t)$ for $t \in [0, 100]$; (d) same for $t \in [0, 300]$. (e) Dynamics of the dispersion $\langle x^2 \rangle - \langle x \rangle^2$ for $t \in [0, 100]$; (f) same for $t \in [0, 300]$.

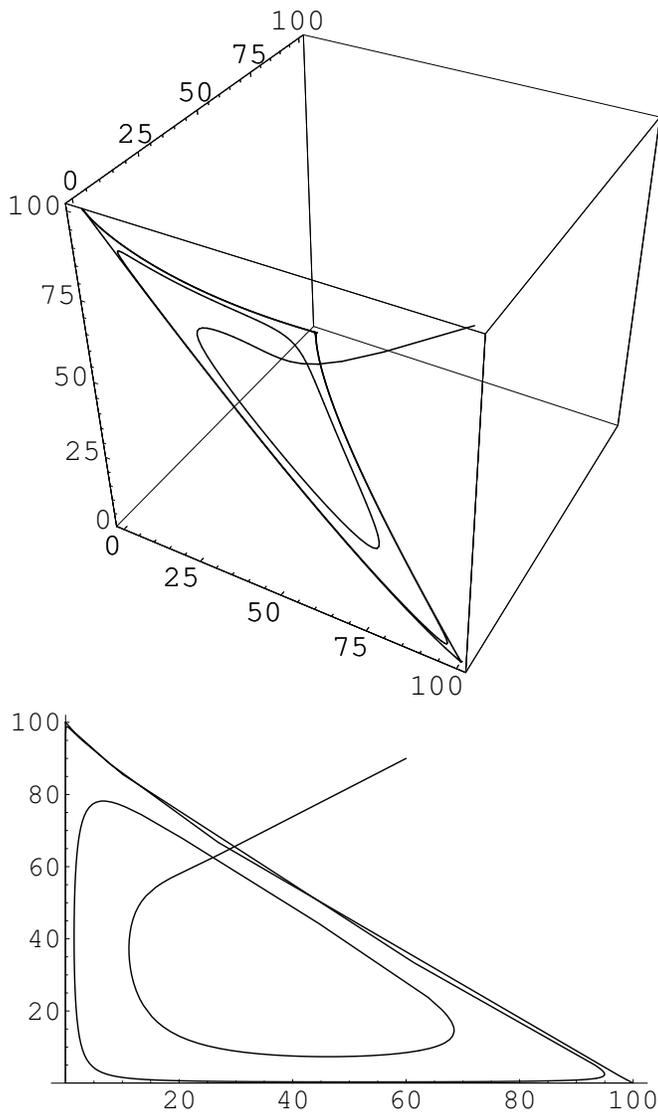


Figure 6.4: The data of Fig.6.3 are reported here through parametric plots, representing thus the trajectory of the system, in three dimensions (upper) and projecting them in the plane (x_1, x_2) (lower).

6.4 Bibliography

Our discussion of the N species and in particular of the three species case follows Hofbauer and Sigmund; the cyclic model was introduced and discussed by May and Leonard and also dealt with by Chenciner. The reader may be warned that often (but not always!) in the mathematical literature reference to “Lotka-Volterra systems” is meant in the sense of dynamical systems with quadratic r.h.s., independent of dimension and of biological applications.

- J. Hofbauer & K. Sigmund, *Evolutionary Games and Population Dynamics*, Cambridge UP 1998
- R. May & W. Leonard, “Nonlinear aspects of competition between three species”, *SIAM Journal of Applied Mathematics* **29** (1975), 243-252
- A. Chenciner, “Comportement asymptotique de systèmes différentiels du type compétition d’espèces”, *C. R. Acad. Sci. Paris* **284**, 313-315

Chapter 7

Hysteresis

In this Chapter we are considering a slightly more realistic model for a population. In particular we will consider a logistic model for a population of trees parasites – concretely, worms – in which we introduce a term taking into account the presence of (non-specific) predators – concretely, birds – feeding on them. This will also illustrate how difficult it may be to recover an equilibrium once it has been destroyed (maybe due to human actions).

The model we are going to discuss was introduced in 1978 by Ludwig, Jones and Holling to describe the proliferation of a parasite, the spruce budworm, infesting conifer forests in North America¹; the mechanism at play is however much more general – as it will be obvious in a moment – and describes in simple, and hence general, terms why once an ecological equilibrium has been destroyed it is often not sufficient to restore the initial environmental conditions.

7.1 The model

Let us consider a population of parasites, e.g. insects infesting a forest; this population will be described by a variable $p(t)$. As prescribed by the logistic model, the evolution of $p(t)$ will be described, in the absence of predators feeding on it, by the logistic equation

$$\frac{dp}{dt} = (\alpha_0 - \beta p) p ; \quad (7.1)$$

On the other hand, if the forest is also the habitat to a population of birds feeding on insects, it will be necessary to introduce in eq.(7.1) a predation term. We will suppose the birds population to be constant in time and equal to \mathcal{B} ; this

¹D. Ludwig, D.D. Jones and C.S. Holling, “Qualitative analysis of insect outbreak systems: the spruce budworm and forest”, *J. Anim. Ecol.* **47** (1978), 315-332; see also D. Ludwig, D.G. Aronson and H.F. Weinberger, “Spatial patterning of the spruce budworm”, *J. Math. Biol.* **8** (1979), 217-258. This later work uses a more sophisticated Mathematics than the previous one, and describes the situation in a more detailed way.

will actually be considered later on as a varying parameter, being influenced on other variables, e.g. the use of pesticides.

It is natural to assume that the predation term depends on both B and $p(t)$, and more precisely that it is directly proportional to B and depends in some more general way on $p(t)$. We will thus write

$$\frac{dp}{dt} = (\alpha_0 - \beta p) p - \mathcal{B} f(p) . \quad (7.2)$$

Our first problem is of course to understand what may be a correct way to describe the predation, i.e. what can be the general form of the function $f(p)$, and possibly a concrete expression for it.

First of all we note that as B is constant, there is a maximal amount of worms which can be eaten by them per unit of time; so we must have a finite limit F_* for the predation when the worms population increases. In mathematical terms, we require

$$\lim_{p \rightarrow \infty} f(p) = F_* < \infty . \quad (7.3)$$

Moreover, obviously, if there are no worms, the birds will not be able to feed on them; that is, we also require

$$\lim_{p \rightarrow 0} f(p) = 0 . \quad (7.4)$$

Now, we have supposed that the birds are non-specific predators. That is, they feed on the worms we are considering, but also on other preys. This entails that if “our” worms become rare, the birds will direct their attention (i.e. look for) other insects, easier to find. In mathematical terms, this means that for p small the predation term will grow very little: we should require

$$\lim_{p \rightarrow 0} f'(p) = 0 , \quad (7.5)$$

which of course just means $f'(0) = 0$.

In other words, we require that the Taylor expansion for p small is of the form

$$f(p) \approx f_2 p^2 + O(p^3) . \quad (7.6)$$

A function satisfying all of these requirements is just our old friend the *sigmoid*²

$$f(p) = \frac{p^2}{A + Bp^2} ; \quad (7.7)$$

here we should take

$$A > 0 , \quad B = F_*^{-1} > 0 .$$

The latter condition guarantees that (7.3) is satisfied, and obviously the limit for $p \rightarrow \infty$ of $f(p)$ defined in (7.7) is just B^{-1} .

²Which, being a function we by now know well, has the nice feature of allowing us to analyze the problem without having to focus on the mathematical detail but keeping instead our attention on the modelling issues.

Summarizing, our concrete model will be

$$\frac{dp}{dt} = (\alpha_0 - \beta p) p - \mathcal{B} \frac{p^2}{A + Bp^2}; \quad (7.8)$$

all the parameters appearing in this equation are assumed to be real and positive, and we will denote for short by $\Phi(x)$ the r.h.s. of our equation,

$$\Phi(p) := (\alpha_0 - \beta p) p - \mathcal{B} \frac{p^2}{A + Bp^2}. \quad (7.9)$$

We are happy in that (7.8) is a separable equation; but it is not possible to express the integral

$$\int \frac{dx}{\Phi(x)}$$

in terms of elementary functions for generic values of the parameters. The reason for this will be apparent on the following: as the parameters are varied, the behavior of the equation changes not only quantitatively, which is obvious, but also qualitatively.

7.2 Equilibria and their dependence on parameters

We are not able to provide the general solution to (7.8), but we may be less ambitious and start by looking for its stationary solutions (thus with $dp/dt = 0$), i.e. the situations where the parasites and the predators are at equilibrium.

For this, it suffices to require $\Phi(p) = 0$. We note first of all that in the expression for $\Phi(p)$ we can collect a common factor p :

$$\Phi(p) = p \left[(\alpha_0 - \beta p) - \mathcal{B} \frac{p}{A + Bp^2} \right]. \quad (7.10)$$

This entails the obvious fact that $p = 0$ is an equilibrium (in fact, if there are no parasites, their population will remain at zero – at least within our model, i.e. unless they enter into the ecological system by migration); we can thus look for other equilibria simply by looking for the values of p which cancel the term in square brackets, which we will denote – again for short – as $\Psi(p)$, with

$$\Phi(x) = x \Psi(x).$$

We have to solve $\Psi(x) = 0$, i.e.

$$(\alpha_0 - \beta x) = \mathcal{B} \frac{x}{A + Bx^2}. \quad (7.11)$$

The l.h.s. member, call it $\rho(x)$, describes a straight line $y = \alpha_0 - \beta x$ with negative slope, which meets the y axis in the point $(0, y_*) = (0, \alpha_0)$ and the x axis in the point $(x_*, 0) = (\alpha_0/\beta, 0)$.

As for the r.h.s. member, call it $h(x)$, it is always positive for $x \geq 0$, and it goes to zero both for $x = 0$ and for $x \rightarrow \infty$.

In particular, it is positive for $x = x_*$; more precisely,

$$h(x_*) = \frac{\alpha_0 \beta \mathcal{B}}{A\beta^2 + \alpha_0^2 B}.$$

Therefore, as $\rho(0) > h(0)$ and $\rho(x_*) < h(x_*)$, and both our functions are continuous, there is *at least* one point in the interval $[0, x_*]$ at which $\rho(x) = h(x)$, and hence $\Psi(x) = 0$.

However, there may be more points with this property. As $\Phi(x)$ is written as a fraction whose numerator is a polynomial of degree three, there may be up to three points at which it vanishes. Note that all the points at which $\Phi(x)$ vanishes are necessarily belonging to the interval $I_\Phi := [0, x_*]$: in fact, for $x < 0$ we have $h(x) < 0$, $\rho(x) > 0$; and for $x > x_*$ we have $h(x) > 0$ and $\rho(x) < 0$ (recall $\Phi(x) = 0$ corresponds to $h(x) = \rho(x)$, see above).

The situation is depicted in Fig.7.1, where we plotted the functions $\rho(x)$ and $h(x)$ for given values of the parameters α_0, β, A, B , and only varied the value of \mathcal{B} . In Fig.7.2 we plotted, for the same values of the parameters, the function $\Phi(x)$.

7.3 Stability of equilibria

Figure 7.2 also allows to immediately determine the stability of equilibria. In fact, $\Phi(x)$ represents dx/dt , hence the population $p(t)$ grows for $\Phi > 0$, and decreases for $\Phi < 0$. It follows that the equilibria in $x = x_0$ with $\Phi'(x_0) > 0$ are unstable, those with $\Phi'(x_0) < 0$ are stable.

On the other hand, $\Phi(x) = x\Psi(x)$, and this entails

$$\Phi'(x) = \Psi(x) + x\Psi'(x); \quad (7.12)$$

this means that the derivative in a point $x_0 > 0$ such that $\Psi(x_0) = 0$ (hence $\Phi(x)$ vanishes as well) is given by

$$\Phi'(x_0) = x_0\Psi'(x_0). \quad (7.13)$$

That is, the sign of $\Phi'(x_0)$ is the same as the sign of $\Psi'(x_0)$ (as we assumed $x_0 > 0$).

IN conclusion, when we have only one nontrivial solution to $\Psi(x) = 0$, this corresponds to a *stable* equilibrium; when we have three nontrivial solutions for $\Psi(x) = 0$, the central one is *unstable* while the side ones are both *stable*. Note that the trivial equilibrium in $x = 0$ is always *unstable*.

Note that it is possible to determine the exact position of the zeros of $\Phi(x)$ as a function of the parameters using the Cardano formula for the solution of a cubic equation; but this would produce a rather involved and not interesting expression, which we therefore omit.

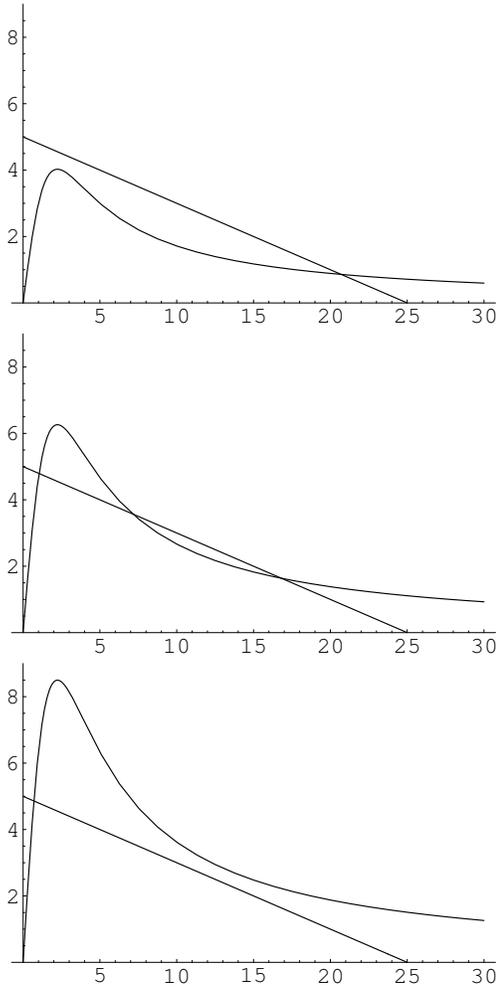


Figure 7.1: Plots of the functions $\rho(x)$ and $h(x)$ for given values of the parameters α_0, β, A, B and different values of the parameter \mathcal{B} . Concretely we have chosen $\alpha_0 = 5, \beta = 0.2, A = 0.5, B = 0.1$, while for \mathcal{B} we considered (from the upper to the lower plot) the values $\mathcal{B} = 1.8, \mathcal{B} = 2.8, \mathcal{B} = 3.8$. The number of points satisfying $h(x) = \rho(x)$, and hence $\Phi(x) = 0$, is one in the first case, three in the second one, and again one in the third one.

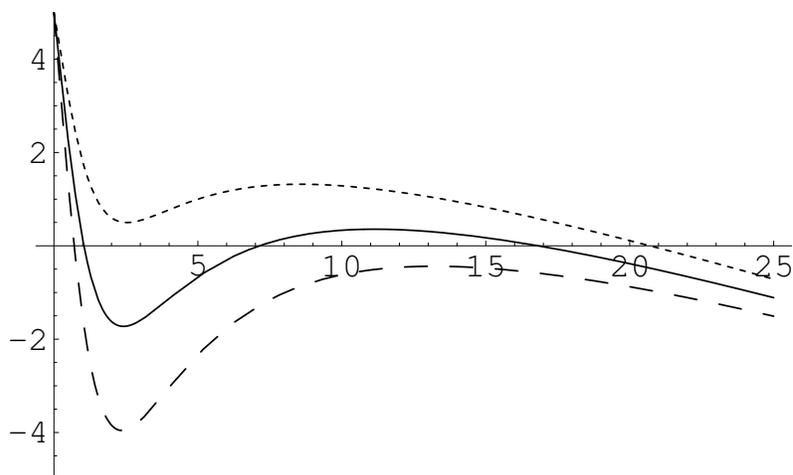


Figure 7.2: The function $\Psi(x)$ for the same values of the parameters considered in Fig.7.1. Here $\mathcal{B} = 1.8$ corresponds to the dotted curve, $\mathcal{B} = 2.8$ to the solid curve, and $\mathcal{B} = 3.8$ to the dashed curve.

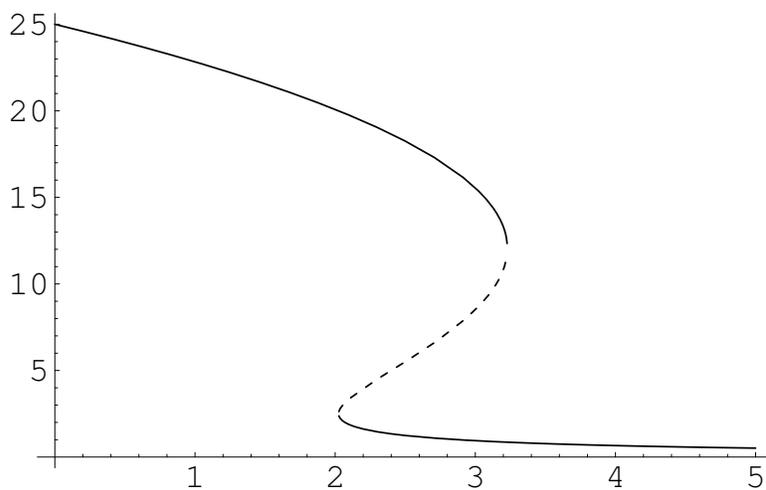


Figure 7.3: The position of the nontrivial equilibria, i.e. the zeros of $\Psi(x)$, as \mathcal{B} is varied while the other parameters remain constant (we used the same values as in Fig.7.1). The stable equilibria x_1 and x_3 are plotted by solid curves, while the unstable equilibrium x_2 by a dashed curve.

The important aspect is the *qualitative* one. In particular, let us see how the situation can change if the number of predators changes for some reason, natural or otherwise (e.g. due to the diffusion of pesticides, used to get rid of the infesting insects but which also affect their predators).

Let us suppose that initially \mathcal{B} is such to be in the situation with three nontrivial equilibria; we denote these as $x_1(\mathcal{B})$, $x_2(\mathcal{B})$, $x_3(\mathcal{B})$, with $0 < x_1(\mathcal{B}) < x_2(\mathcal{B}) < x_3(\mathcal{B})$. As we have seen above, in this case the “external” equilibria x_1 and x_3 are stable; let us suppose to be in $x_1(\mathcal{B})$. As emphasized by our present notation, the exact values of these equilibria depend on the value of \mathcal{B} (and also on the other parameters; but we suppose these to be given).

As \mathcal{B} diminishes, the equilibrium points assume different values; these vary continuously with \mathcal{B} until the latter takes the limiting value \mathcal{B}_* . This is the value at which the curve $h(x)$ lowers down to have only one intersection with $\rho(x)$. This intersection does *not* correspond to $x_1(\mathcal{B}_*)$; it corresponds instead to $x_3(\mathcal{B}_*)$. Thus when \mathcal{B} is just below \mathcal{B}_* , the population x of parasites jumps from $x_1(\mathcal{B}_*)$ to $x_3(\mathcal{B}_*)$.

Suppose now we manage – maybe due to the fact the situation is getting much worse from the point of view of parasite diffusion, and we got aware of the fact this depends on the low level of the birds population – to have the predators population \mathcal{B} increase again until we arrive to $\mathcal{B} > \mathcal{B}_*$. In this process the parasite population changes with \mathcal{B} , and diminishes as \mathcal{B} increases; but now it follows the branch corresponding to the equilibrium $x_3(\mathcal{B})$. In order to recover the more favorable (for the trees and hence for us) initial situation, i.e. in order to drive the parasite population to the branch $x_1(\mathcal{B})$, it is necessary that \mathcal{B} grows up to above the second threshold value \mathcal{B}_{**} , i.e. the value for which $h(x)$ has again only one intersection with $\rho(x)$, now corresponding to the equilibrium x_1 .

The “naturalistic” interpretation of this analysis is quite simple: once an equilibrium is destroyed, it is not sure that in order to recover it one has “just”, so to say, to drive back the environmental parameters to their initial values. In some cases, a much greater effort may be needed; or recovering the initial situation may be just impossible³. Ecological systems are in general not “reversible”, and going back as far as some (or even all of the) control parameters are concerned does not guarantee that the system is driven back to the initial state.

7.4 Two examples

In order to illustrate our discussion in a concrete case, let us consider eq.(7.8) with given values of the parameters apart from \mathcal{B} . We will use the same parameters used in Figg.1–4, i.e.

$$\alpha_0 = 5, \quad \beta = 0.2, \quad A = 0.5, \quad B = 0.1. \quad (7.14)$$

³It is not difficult to imagine different shapes for $f(p)$, such that the equilibrium x_3 is always present.

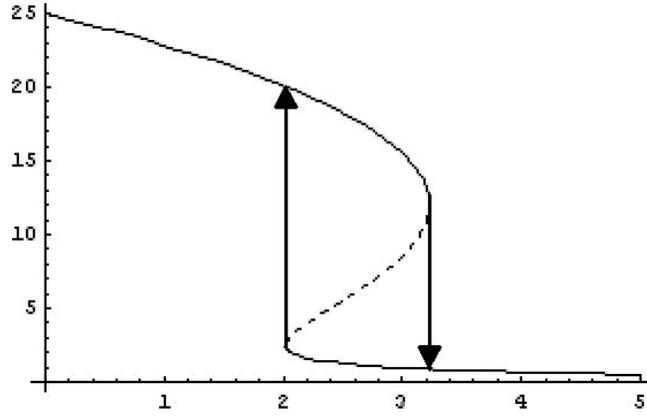


Figure 7.4: The hysteresis cycle. if the parameter \mathcal{B} grows above the limit value \mathcal{B}_* , or decreases below the second limit value $\mathcal{B}_{**} > \mathcal{B}_*$, there is only one equilibrium point, and the population initially at the other equilibrium point will evolve until reaching this remaining equilibrium. If a value of \mathcal{B} for which both stable equilibria are present is recovered, the population will remain on the branch connected continuously with its initial state.

As for \mathcal{B} , we will have it changing slowly in time in a prescribed way. In order to fix ideas, we note that with the values of the parameters given in (7.14), it results

$$\mathcal{B}_* \approx 2.025, \quad \mathcal{B}_{**} \approx 3.23. \quad (7.15)$$

We choose to vary $\mathcal{B}(t)$ as follows:

$$\mathcal{B}(t) = \begin{cases} 4 - \varepsilon t & \text{for } 0 \leq t \leq 500 \\ 1.5 & \text{for } 500 \leq t \leq 1000 \\ 1.5 + \varepsilon t & \text{for } 1000 \leq t \leq 1500 \end{cases} \quad (7.16)$$

and we choose $\varepsilon = 0.005$.

The numerical integration of eq.(7.8) with \mathcal{B} changing as in (7.16) produces the function $X(t)$ plotted in fig.7.5. Showing in the same graph the value of $\mathcal{B}(t)$ on the horizontal axis and that of $X(t)$ on the vertical one (that is, we are producing a parametric plot), we get the plot shown in Fig.7.6; this corresponds to the one seen before in order to illustrate the hysteresis cycle.

The non-smooth behavior of $\mathcal{B}(t)$ in this example could raise some objection; we will thus consider an example in which $\mathcal{B}(t)$ changes smoothly.

We will have $\mathcal{B}(t)$ changing sinusoidally, see Fig.7.7,

$$\mathcal{B}(t) = u_0 - \sin(\omega t) \quad (7.17)$$

and we choose

$$\omega = 0.005 \pi. \quad (7.18)$$

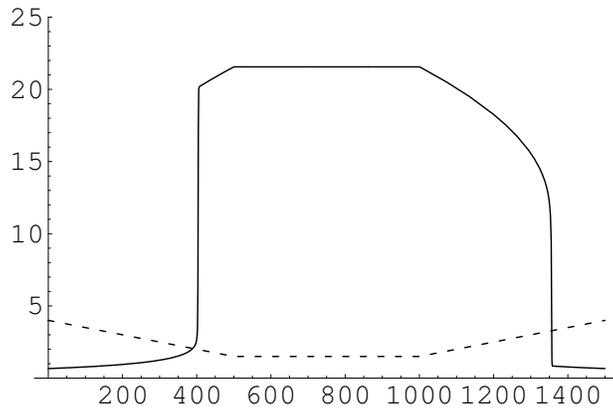


Figure 7.5: Numerical integration of $x(t)$ with $\mathcal{B}(t)$ changing slowly with t in a prescribed way, see text (Sect.7.4). The solid curve represents the result $X(t)$ of the numerical integration; we also show $\mathcal{B}(t)$ (dashed curve) in order to show how $X(t)$ take different values for the same value of $\mathcal{B}(t)$ depending on this being in the growing or the decreasing phase.

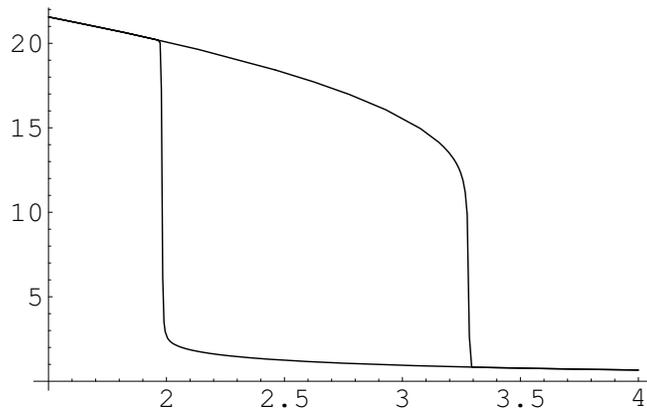


Figure 7.6: Parametric plot of $X(t)$ vs. $\mathcal{B}(t)$ for the numerical integration of Sect.7.4; this clearly shows the hysteresis cycle.

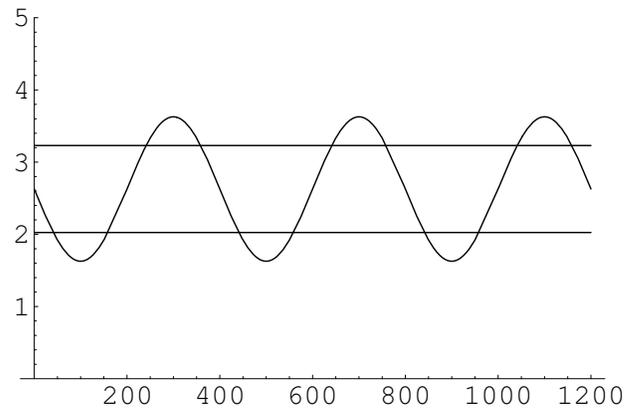


Figure 7.7: The function $B(t)$ defined by (7.18), here shown together with the limit values B_* and B_{**} for the sake of comparison.

The numerical integration of eq.(7.8) produces then the function $X(t)$ shown in Fig.7.8, illustrating again the phenomenon discussed in this chapter.

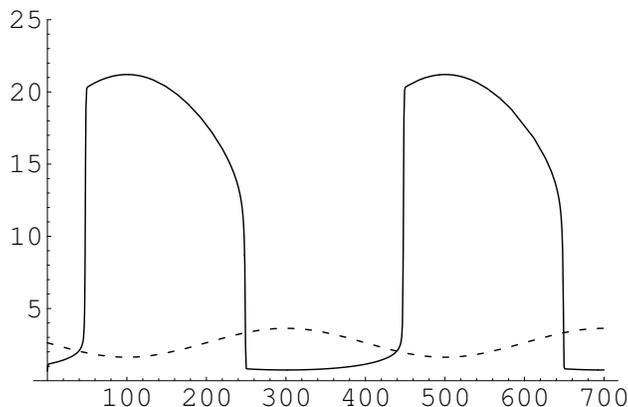


Figure 7.8: The result $X(t)$ of numerical integration of (7.8), with \mathcal{B} given by (7.17), (7.18). The solid line represents $X(t)$, the dashed one represents $\mathcal{B}(t)$.

7.5 Bibliography

The model discussed here was introduced, as mentioned above, by Ludwig and collaborators. Our discussion follows closely enough that in the book by Istas. For a general discussion of hysteresis see e.g. Guckenheimer & Holmes; more focused discussion are provided e.g. by Krasnosel'skii & Pokrovskii, Visintin, and Mayergoyz. The reader is however warned that these discussions focus on rather different fields of application.

- D. Ludwig, D.D. Jones and C.S. Holling, “Qualitative analysis of insect outbreak systems: the spruce budworm and forest”, *J. Anim. Ecol.* **47** (1978), 315-332
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