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Spatiotemporal Patterns in Ecology and Epidemiology

Theory, Models, and Simulation

Horst Malchow, Sergei V. Petrovskii, and Ezio Venturino
Dynamics has always been a core issue of all natural sciences. What are the driving forces and the “mechanisms” that result in motion of the system parts and/or lead to its evolution as a whole, whatever that system may be (particular cases range from a single rigid body to the human society)? What are the properties and scenarios of this motion and evolution? What can be the meaning or implication of this dynamics if considered in a wider context, e.g., through interaction with other systems or other sciences? These have been challenging and exciting problems for philosophers and scientists throughout at least 30 centuries.

The focus of interest has evolved, too. At the dawn of contemporary science, stationary processes were regarded as the essence of dynamics. Non-stationary motion was often either attributed to a specific cause (such as periodic forcing) or considered as a mere transient that was bound to die out after a relatively short relaxation time. The corresponding system’s geometry was assumed to be smooth and regular. Those phenomena that did not fit into this philosophy, turbulent flow being the renowned example, were regarded as exotic and rare and, possibly, not self-sustained.

Although this mainstream thought of science has been challenged from time to time from as early as ancient Greece, it was not until the late twentieth century that it was widely realized that the dynamics of even very simple systems can be – and often is – completely different and much more complicated. Relaxation to steady states and periodic motion (with the limit cycle as its mathematical paradigm), which used to be the main elements of dynamics, were displaced by the concept of deterministic chaos. Smooth surfaces and simple curves gave way to objects with fractal properties. Moreover, chaos and fractals were eventually found (if not empirically, then at least theoretically) nearly everywhere, from lasers to animal behavior.

Even more importantly, it was realized that complex temporal dynamics and, especially, spatial structures do not just exist per se but can arise as a result of a system’s self-organization. In an open system, i.e., a system with an inflow of mass and/or energy, the dynamics can become intrinsically unstable, resulting either in the formation of periodic spatial patterns or in a complicated turbulence-like spatiotemporal behaviour.

A similar evolution of concepts and ideas occurred in ecology and population biology. Ecologists have long been aware that population distribution in a natural environment is normally distinctly heterogeneous; however, that was usually regarded as a separate phenomenon that was not directly related
to the main properties of population dynamics in time such as its persistence, reproductive success, etc. Correspondingly, earlier studies tended to focus mainly on the dynamics of “nonspatial” systems (i.e., systems where the spatial distribution of all factors and agents was regarded to be homogeneous under any circumstances). The results of the last two decades, however, proved that the impact of spatial dimensions can be crucial. The dynamics of a spatially extended system can be qualitatively different from the dynamics of its nonspatial counterpart due to self-organized, “spontaneous” pattern formation.

It should be mentioned that recent progress in theoretical ecology would unlikely have become possible without extensive use of mathematical modeling. There are several reasons why a complete and thorough study of ecosystem dynamics is hardly possible if based only on field data collection. Field observations are often very expensive and field experiments can sometimes be dangerous for the environment. Moreover, a regular experimental study implies replicated experiments; however, this is hardly possible in ecology because of the virtual impossibility of reproducing the same initial and environmental conditions. Note that mathematical models have been used in ecology from as early as the nineteenth century (cf. the work by Malthus) but they became a really powerful research tool after the development of numerical simulation approaches and modern computers.

Importantly, although the spatial dimension of ecosystems dynamics is nowadays widely recognized, the specific mechanisms behind species patterning in space are still poorly understood and the corresponding theoretical framework is underdeveloped. In particular, existing textbooks and research monographs on theoretical/mathematical ecology, when addressing its spatial aspect, practically never go beyond the classical Turing scenario of pattern formation. This book is designed to fill this gap, in particular, by giving an account of the significant progress made recently (and published in periodic scientific literature) in understanding these issues through mathematical modeling and numerical simulations using some basic, conceptual models of population dynamics.

A special remark should be made regarding terminology. Apparently, the term “pattern” has originally appeared in application to spatial processes where some kind of heterogeneity is observed. In the context of this book, however, we use this term somewhat more broadly, embracing also the purely temporal dynamics of spatially homogeneous systems; cf. “patterns of temporal behaviour.”

Another tendency in scientific periodics over the last decade has been convergence between ecology and epidemiology. Although it seems to be common knowledge that a disease can change population dynamics essentially, mathematical approaches to these issues remained distinctly different until recently. Remarkably, both population dynamics and disease dynamics exhibit many similar properties, especially with regards to pattern formation in space and time. This book provides a first attempt at a unified approach to popula-
tion dynamics and epidemiology by means of considering a few “ecoepidemiological” models where both the basic interspecies interactions of population dynamics and the impact of an infectious disease are considered explicitly.

The book is organized as follows. Part I starts with a general overview of relevant phenomena in ecology and epidemiology, giving also a few examples of pattern formation in natural systems, and then proceeds to a brief synopsis of existing modeling approaches.

Part II deals with nonspatial models of population dynamics and epidemiology. We have already mentioned that the dynamics of spatial and corresponding nonspatial systems can be essentially different and the results of nonspatial analysis may, in some cases, be misleading. Nevertheless, it is also clear that the properties of nonspatial dynamics provide a certain “skeleton” important for a thorough understanding of spatiotemporal dynamics. Correspondingly, Part II gives a wide panorama of existing nonspatial approaches, starting from basic ideas and elementary models and eventually bringing the reader to the state-of-the-art in this area.

In Part III, we introduce space by means of including “diffusion” of the individuals and consider the main scenarios of spatial and spatiotemporal pattern formation in deterministic models of population dynamics.

Finally, in Part IV, we address the issue of interaction between deterministic and stochastic processes in ecosystem/epidemics dynamics and consider how noise and stochasticity may affect pattern formation.

When writing this book, we were primarily thinking about experienced researchers in theoretical and mathematical ecology and/or in relevant areas of applied mathematics as the “target audience,” and that affected its structure and style. In particular, from the beginning of Part I we use some advanced terminology from applied dynamical systems, mathematical modelling, and differential equations, which requires the reader to have at least some basic education in these topics (even in spite of the fact that much of that terminology is actually explained later in the text). However, we do hope that many researchers from neighboring fields and also postgraduate students will find this book useful as well; in order to encourage them to read it, we give enough calculation details. For the same purpose, the presentation of some introductory items is, at times, made on a rather elementary level.

A considerable part of the results included into this book was obtained in numerical simulations. Therefore, although numerical results by no means can be regarded as an adequate substitute to rigorous analysis, we think that it will be only fair if the reader is given an opportunity to reproduce the main results and (which is probably even more important) to make a deeper look into the system’s dynamics in his/her own numerical experiments. For that purpose, a CD is attached to this book that contains many of the computer programs that we have used in our work. The programs are written in MATLAB®; it must be mentioned here that MATLAB® and Simulink® are trademarks of The MathWorks, Inc., and are used with permission. The MathWorks does not warrant the accuracy of the programs on the CD. This
CD’s use or discussion of MATLAB® and Simulink® software or related products does not constitute endorsement or sponsorship by The MathWorks of a particular pedagogical approach or particular use of MATLAB® and Simulink® software.

In conclusion, it is our pleasure to express our gratitude to numerous people who helped this book to appear through many fruitful discussions and helpful comments, both during manuscript preparation and during the equally important time preceding this work. We are particularly grateful to Ulrike Feudel, Nanako Shigesada, Michel Langlais, Michael Tretyakov, Lutz Schimansky-Geier, Vitaly Volpert, Andrey Morozov, Frank Hilker, Jean-Christophe Poggiale, Hiromi Seno, Bai-Lian (Larry) Li, Herbert Hethcote, Alexander Medvinsky, Joydev Chattopadhyay, Olivier Lejeune, Michael Sieber, Guido Badino, Francesca Bona, and Marco Isaia. S.P. is very thankful to his colleagues in the Department of Mathematics of the University of Leicester for their continuing encouragement and support. E.V. thanks the Max–Planck–Institut für Mathematik in Bonn, where, during an informal visit, parts of this book were written. E.V. is also very much indebted to the persons who long ago introduced him to the fascinating field of mathematical modeling, especially to Brian Conolly, Edward Beltrami, and James Frauenthal. Last but not least, we all are thankful to Sunil Nair, publisher of mathematics and statistics, CRC Press/Chapman & Hall, for inviting us to write this book and for his patience and encouragement.

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Part I

Introduction
Chapter 1

Ecological patterns in time and space

1.1 Local structures

Ecological systems are open systems, highly nonlinear and, hence, one has to cope with all challenges of nonlinear, nonequilibrium dynamics. The simplest growth and interaction laws are already nonlinear and, together with the variable environment, drive ecosystems away from a static equilibrium, which is meant as thermodynamic equilibrium with maximum entropy. The dynamic or flux equilibria far from thermodynamic equilibrium are called steady states.

Steady-state multiplicity

The most simple but important nonlinear effect is the emergence of steady-state multiplicity. Already the logistic growth of a single population has two of them, the unstable extinction and the stable carrying capacity. Bistability appears if this population is prey for a Holling type III predator like in the one-dimensional spruce budworm system (Ludwig et al., 1978; Wissel, 1985). Populations with strong Allee effect also have two stable steady states, contrary to logistic growth the extinction state and again the carrying capacity (Courchamp et al., 1999, 2000). Prey–predator interactions like in the Rosenzweig-MacArthur model (1963), with logistically growing prey and Holling type II predator may generate two alternative stable states in the presence of a top predator as planktivorous fish in Scheffer’s plankton model (1991a). In a certain range of control parameters a stable constant and an oscillating state may also coexist. If the predator is of Holling type III, the system may become bistable through intra-specific competition in the predator population and even tristable by the mentioned top predator. However, the multiple stability masks a just as interesting property of this model type: it is excitability, i.e., supercritical perturbations of the only stable steady state may lead to an outbreak in the prey population with long relaxation time. This effect is used for modeling recurrent phytoplankton blooms (Truscott and Brindley, 1994) and outbreaks of infectious diseases (Malchow et al., 2005). However, back to steady-state multiplicity: Multiple states are known in rivers and lake ecosystems (Scheffer, 1998; Dent et al., 2002), but also in terrestrial such as semi-arid grazing systems (Rietkerk and van de Koppel, 2002) or in
climate (Higgins et al., 2002). In a deterministic model in a uniform, change-
less environment, the initial condition would determine which stable constant
or oscillating state will be approached once and forever. But ecosystems are
also exposed to noisy variability of conditions like climate and weather. If such
fluctuations become supercritical, the systems may jump between alternative
stable states. It is not only noise but also continuously changing environmen-
tal conditions, such as the current global warming crisis, that can lead to such
jumps. The latter is related to the observations and theory of regime shifts
in ecosystems, e.g. from clear to eutrophicated water or from wet to dry land
(Wissel, 1981; Rietkerk, 1998; Scheffer et al., 2001; Scheffer and Carpenter,
2003; Foley et al., 2003; Rietkerk et al., 2004; Greene and Pershing, 2007).
The return, if possible at all, is very slow, usually on a hysteresis loop.

Regular population oscillations

Since Elton (1924; 1942), Lotka (1925), Volterra (1926a), Gause and Vitt
(1934), and others, oscillations in populations bother experimental and theo-
retical ecologists. There are ongoing discussions about the underlying mech-
isms. One side underlines the role of population interactions as predation
or competition, the other side the control through environmental variability,
and there are examples for both. Corresponding prominent cases are the cy-
cles in a vertebrate prey–predator community of the collared lemming in the
high-Arctic tundra in Greenland that is preyed by four predators (Gilg et al.,
2003; Hudson and Bjørnstad, 2003; Gilg et al., 2006), the predation behind
pine beetle oscillations in the southern United States (Turchin et al., 1999;
Turchin, 2003) as well as the oscillations in different fish that are induced
by climate fluctuations (Stenseth et al., 2002). However, the truth will be
somewhere in between, as in the Canada lynx oscillations that are induced by
stochastic climatic forcing and density-dependent processes (Stenseth et al.,
1999), and we do not want to participate in this discussion. The importance of
oscillations for biodiversity maintenance and noninvadibility has been stressed
by Vandermeer (2006) with an earlier application to plankton (Huisman and
Weissing, 1999).

Irregular population oscillations

There is even more dispute about the existence and role of deterministic
chaotic oscillations in ecology (Berryman and Millstein, 1989; Pool, 1989;
Scheffer, 1991b; Ascioti et al., 1993; Hastings et al., 1993; Ellner and Turchin,
1995; Cushing et al., 2001; Rai and Schaffer, 2001; Cushing et al., 2003).
Though there might be no convincing proof of chaos in wildlife, except for
some signs in boreal rodents (Hanski et al., 1993) or in the epidemics of a
few childhood diseases (Olsen et al., 1988; Olsen and Schaffer, 1990; Engbert
and Drepper, 1994); there are examples in laboratory experiments (Dennis
et al., 1997; Becks et al., 2005). We believe that there is chaos in popu-
Ecological patterns in time and space

![Graph showing prey–predator oscillations in lemmings (solid line) and stoats (dashed line). Data courtesy of Olivier Gilg, Helsinki.](image)

**FIGURE 1.1**: Prey–predator oscillations in lemmings (solid line) and stoats (dashed line). Data courtesy of Olivier Gilg, Helsinki.

Population dynamics and that it is masked by environmental and demographic noise. If one accepts the existence of intrinsic population oscillations, then the superposition of extrinsic forcings may naturally lead to quasiperiodic and aperiodic dynamics (Evans and Parslow, 1985; Truscott, 1995; Popova et al., 1997; Ryabchenko et al., 1997). However, we do not overemphasize the role of chaos in ecological systems; it is just another form of variability.

**Noise**

Environmental variability is not purely deterministic, but also noisy. Therefore, the description by ordinary differential equations is always an approximation. One has to consider stochastic differential equations to account for the noise (Gardiner, 1985; Anishenko et al., 2003). There are noise-induced regime shifts between alternative stable states in ecosystems that are possible (Scheffer et al., 2001; Scheffer and Carpenter, 2003; Collie et al., 2004; Rietkerk et al., 2004; Steele, 2004; Freund et al., 2006) as well as counter-intuitive phenomena such as quasideterministic oscillations (Hempel et al., 1999; Neiman et al., 1999; Malchow and Schimansky-Geier, 2006), noise-enhanced stability, noise-delayed extinction, stochastic resonance (Freund et al., 2002), or noise-induced spatial pattern formation (García-Ojalvo and Sancho, 1999; Lindner et al., 2004; Spagnolo et al., 2004; Sieber et al., 2007).
1.2 Spatial and spatiotemporal structures

Ecology happens in time and space, and, therefore, its modeling also requires time and space. Not only growth and interactions but also spatiotemporal processes like random or directed and joint or relative motion of species as well as the variability of the environment must be considered. The interplay of growth, interactions, and transport causes the whole variety of spatiotemporal population structures that includes regular and irregular oscillations, propagating fronts, target patterns and spiral waves, pulses, and stationary as well as fuzzy dynamic spatial patterns.

Diffusive fronts and spatial critical sizes

The simplest known spatiotemporal structures are diffusive invasion fronts of growing populations. For exponential growth, Luther (1906) estimated the front speed, which is numerically the same as the minimum speed of a logistically growing population (Fisher, 1937; Kolmogorov et al., 1937). The textbook example of the invasion of an exponentially growing population is the spread of muskrats in Europe (Skellam, 1951; Okubo, 1980; Okubo and Levin, 2001).

![Figure 1.2: Spread of muskrats over Europe. With permission from Skellam (1951).](image)

For populations with multiple steady states, e.g., bistability in a population with a strong Allee effect, the spatial competition and spread of these states are known. In one spatial dimension, a critical radius of the spatial extension
Ecological patterns in time and space

of a population can be defined (Schlögl, 1972; Nitzan et al., 1974; Ebeling and Schimansky-Geier, 1980; Malchow and Schimansky-Geier, 1985). Population patches greater than the critical size will survive, while the others will become extinct. However, bistability and the emergence of a critical spatial size do not necessarily require an Allee effect; logistically growing preys with a parametrized predator of type II or III functional response can also exhibit two stable steady states and the related hysteresis loops, cf. Ludwig et al. (1978); Wissel (1989).

**FIGURE 1.3**: Diffusive fronts in systems with logistic growth and bistable dynamics. For logistic growth, the front moves to the right-hand side, and, finally, the space $X$ is filled with population $U$ at its capacity $K$. In bistable systems, the direction of the front depends on the initial condition: If the initial patch size exceeds a certain critical value, the picture will be the same as for logistic growth. However, if the initial patch is not large enough, the population will go extinct, though the local model would have predicted its survival.

**Diffusion-driven instabilities**

Alan Turing (1952) was among the first to emphasize the role of nonequilibrium diffusion–reaction processes and patterns in biomorphogenesis. Since then, dissipative nonequilibrium mechanisms of spontaneous spatial and spatiotemporal pattern formation in a uniform environment have been of uninterrupted interest in experimental and theoretical biology and ecology. The interaction of at least two species with considerably different diffusion coefficients can give rise to spatial structure. A spatially uniform population distribution that is stable against spatially uniform perturbations (or in the local model without diffusion) can be driven to diffusive instability against spatially heterogeneous perturbations, e.g., a population wave or local out-
break, for sufficient differences of diffusivities. First, Segel and Jackson (1972) applied Turing’s idea to a problem in population dynamics: the dissipative instability in the prey–predator interaction of algae and herbivorous copepods with higher herbivore motility. Levin and Segel (1976) suggested this scenario of spatial pattern formation as a possible origin of planktonic patchiness. Rietkerk et al. (2002) propose this mechanism as possible for the formation of tiger bushes; see Section 9.3.

Differential-flow-induced instabilities

The Turing mechanism depends on the strong requirement of a sufficient difference of the diffusion coefficients. The latter neither exists for chemical reactions in aqueous solutions nor for micro-organisms in meso- and large-scale aquatic systems where the turbulent diffusion is relevant. Differential-flow-induced instabilities of a spatially uniform distribution can appear if flowing reactants or moving species like prey and predator possess different velocities, regardless of which one is faster. This mechanism of generating patchy patterns is more general. Thus, one can expect a wider range of applications of the differential-flow mechanism in population dynamics (Malchow, 2000b; Rovinsky et al., 1997; Malchow, 2000a). Conditions for the emergence of three-dimensional spatial and spatiotemporal patterns after differential-flow-induced instabilities (Rovinsky and Menzinger, 1992) of spatially uniform populations were derived (Malchow, 1998, 1995, 1996) and illustrated by patterns in Scheffer’s model (1991a).

Complex spatial group patterns may also be generated by different animal communication mechanisms (Eftimie et al., 2007).

Also, vegetation can form a number of spatial patterns, especially in arid and semi-arid ecosystems. Usually, there is a combination of diffusive and advective mechanisms that yield gaps, labyrinths, stripes (tiger bush), or spots (leopard bush) (Lefever and Lejeune, 1997; Klausmeier, 1999; Lefever and Lejeune, 2000; Rietkerk et al., 2002, 2004).

![FIGURE 1.4: Tiger bush in Niger. Courtesy of Charlie Walthall, James R. Irons, and Philip W. Dabney, NASA Goddard Space Flight Center; see also Brown de Colstoun et al. (1996).](image-url)
Target patterns and spiral waves

Target patterns and spiral waves were first known from oscillating chemical reactions, cf. Field and Burger (1985), but have only later been observed as biologically controlled structures in natural populations. Spirals have been found to be important in models of parasitoid-host systems (Boerlijst et al., 1993). For other motile microorganisms, traveling waves like targets or spirals have been found in the cellular slime mold *Dictyostelium discoideum* (Gerisch, 1968; Keller and Segel, 1970; Gerisch, 1971; Segel and Stoeckly, 1972; Segel, 1977; Newel, 1983; Alt and Hoffmann, 1990; Siegert and Weijer, 1991; Steinbock et al., 1991; Vasiev et al., 1994; Ivanitskii et al., 1994; Höfer et al., 1995; Polezhaev et al., 2005). These amoebae are chemotactic species, i.e., they move actively up the gradient of a chemical attractant and aggregate. Chemotaxis is a kind of density-dependent cross-diffusion (Keller and Segel, 1971a,b). Agladze et al. (1993) have shown that colliding taxis waves may generate stationary spatial patterns, and they suggest this as an alternative to the classical Turing mechanism. Bacteria like *Escherichia coli* or *Bacillus subtilis* show a number of complex colony growth patterns (Shapiro and Hsu, 1989; Shapiro and Trubatch, 1991), some of them similar to diffusion-limited aggregation patterns (Witten and Sander, 1981; Matsushita and Fujikawa, 1990). Their emergence also requires cooperativity and active motion of the species, which has been modeled as density-dependent diffusion and predation (Kawasaki et al., 1995, 1997).

**FIGURE 1.5**: Spirals in an amoeba population (*Dictyostelium discoideum*). The base line of the photo is about 28.9 mm. Courtesy of Christiane Hilgardt and Stefan C. Müller, University of Magdeburg.
New routes to spatiotemporal chaos

Space also provides new routes to chaotic dynamics. The emergence of diffusion-induced spatiotemporal chaos along a linear nutrient gradient has been found by Pascual (1993) in a Rosenzweig-MacArthur phytoplankton-zooplankton model. Chaotic oscillations behind propagating diffusive fronts are found in a prey–predator model (Sherratt et al., 1995, 1997). Furthermore, it has been shown that the appearance of chaotic spatiotemporal oscillations in a prey–predator system is a somewhat more general phenomenon and need not be attributed to front propagation or to an inhomogeneity of environmental parameters (Petrovskii and Malchow, 1999; Petrovskii and Malchow, 2001b; Petrovskii et al., 2003; Petrovskii and Malchow, 2001a; Petrovskii et al., 2005).

Patterns of biological invasion and epidemic spread

A problem of increasing concern is the spread of non-native species and epidemic diseases (Drake and Mooney, 1989; Pimentel, 2002; Sax et al., 2005; Allen and Lee, 2006). Biological invasions are regarded as one of the most severe ecological problems, being responsible for the extinction of indigenous species, sustainable disturbance of ecosystems, and economic damage. Therefore, there is an increasing need to control and manage invasions. This requires an understanding of the mechanisms underlying the invasion process. Recently, many factors have been identified that affect the speed and the pattern of the spatial spread of an introduced species or pathogen, such as spatial heterogeneity, resource availability, stochasticity, environmental borders, predation, competition, infection, etc.; cf. recent reviews by Fagan et al. (2002), Hastings et al. (2005), Hilker et al. (2005), Holt et al. (2005), and Petrovskii et al. (2005). Mathematics, mathematical modeling, and computer science play an increasing if not central role in exploring, understanding and predicting these complex processes. This is especially true in studies of biological invasions where laboratory experiments cannot embrace appropriate spatial scales, and manipulative field experiments are, if at all ethically acceptable, very expensive, potentially dangerous, and lack the necessary time series.

The transmission dynamics of infectious diseases is one of the oldest topics of mathematical biology. As early as 1760, Daniel Bernoulli provided the first known mathematical result of epidemiology, that is, the defense of the practice of inoculation against smallpox (Brauer and Castillo-Chavez, 2001). The amount of works in this area has exploded in the last decades. Different aspects are dealt in the literature, from human health assessment to environmental assessment.
Chapter 2

An overview of modeling approaches

The great variety of different environmental settings and ecological interactions existing in mother nature requires, in order to make their theoretical study more effective, an equally broad range of modeling approaches. Quite typically, an adequate model choice depends not only on the ecosystem or community type, but also on the goals of the study, in particular, on the spatial and/or temporal scales where the given phenomena are developing. Discreteness of populations is a fundamental property (cf. Durrett and Levin, 1994), yet on a spatial scale much larger than the size of a typical individual description of the population dynamics by a continuous quantity such as the population density (the number of individuals of a given species per unit area or unit volume) was proved to be effective, e.g., see Murray (1989) and Shigesada and Kawasaki (1997). On a larger scale, however, environmental heterogeneity and habitat fragmentation become important, which may make space-discrete models more appropriate. A similar duality arises in the temporal dynamics. Moreover, due to the possible overlapping of spatial and/or temporal scales associated with different processes, sometimes a hybrid approach might be required that describes some of the processes continuously and some of them discretely; one example is given by a fish school feeding on plankton (Medvinsky et al., 2002).

The very first step to be done in choosing the model is a decision about the “state variables,” i.e., the quantities that give sufficient (for the purposes of a given study) information about the state of the system. There is an apparent fundamental difference between the modeling approaches that take into account each individual separately, cf. “individual-based modeling,” and the approaches that describe the system state in a collective way, e.g., by means of introducing the population density. Obviously, an individual-based approach\(^1\) gives more information about the population system than an approach based on the population density; however, its disadvantage is that this information can rarely be obtained other than through extensive numerical simulations. On the contrary, the density-based models often allow rigorous mathematical analysis and analytical treatment. Another important distinction is that predictions of individual-based approaches are usually restricted to the spa-

\(^1\)For the basics and state-of-the-art in that field, an interested reader is advised to check the books by DeAngelis and Gross (1992) and Grimm and Railsback (2005).
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Having chosen a density-based approach, the next step is to decide whether details of system’s spatial structure may be important. In case they are not, then one arrives at a nonspatial model where the population density or densities are functions of time but not of space. Specific mathematical settings for writing down the model depend on whether the dynamics of a given population is more adequately described as time-discrete or time-continuous. The former approach works better for the populations with nonoverlapping generations, the latter is valid when the generations are tangled. Throughout this book, we will mostly focus on the second case; a relevant mathematical technique is then given by ordinary differential equations:

$$\frac{dU_i(t)}{dT} = f_i(U_1, U_2, \ldots, U_n)$$

where $U_i$ is the population density of the $i$th species at time $T$, $n$ is the number of species in the community, and functions $f_i$ take into account effects of birth and mortality; in most biologically meaningful situations, the functions $f_i$ are nonlinear with respect to at least some of their arguments.

Now, a very subtle issue is the decision about how many equations should the system (2.1) contain and (a closely related question) what are the properties of functions $f_i$, which define the species responses and the types of interspecific interactions. Obviously, the population community even in a very simple ecosystem consists of dozens (more typically, hundreds or even thousands) of different species. Therefore, an idea to write a separate equation for each species is totally unrealistic. One way around this difficulty is to consider some “functional groups” instead of particular species. Most straightforwardly, these groups would correspond to different trophic levels. A classical example is given by phytoplankton and zooplankton; although in any natural aquatic ecosystem each of these two groups consists of many different species (sometimes interacting with each other in a very complicated way), ecologists use these rough “binary” description quite successfully, in both empirical and theoretical studies. Correspondingly, application of the model (2.1) to the plankton system dynamics would result in a two-species prey–predator system.

An alternative approach to minimize the number of equations in (2.1) is to focus on the dynamics of particular species. The reasons behind the choice of those “key species” depends on the ecosystem properties but also on the purpose of the study; for instance, in the case of biological invasion, one of them should obviously be the alien pest. The system (2.1) then may be reduced to either just a single equation or to a few-species system described by two or three equations, e.g., accounting for the given species and its immediate consumers or competitors. The impact of other species can be taken into account in an indirect way by means of either adjusting parameter values (e.g., introducing additional mortality rates in order to account for other
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predators) or by including additional terms into the equations, cf. “closure terms” (van den Bosch et al., 1988; Steele and Henderson, 1992a; Edwards and Yool, 2000).

Note that, while the predictive power of the few-species “conceptual” models is usually not very high, they are very important in a wider theoretical aspect because they make it possible to study the implications of basic interspecific interactions thoroughly.

It should be also mentioned that, apart from the population dynamics, the generic system (2.1) has been effectively used for modeling the dynamics of infectious diseases, up to a somewhat different meaning of the state variables (such as density of susceptibles instead of density of prey, etc.) and to the choice and meaning of the functions $f_i$, e.g., see Busenberg and Cooke (1981), Capasso (1993), and Dieckmann et al. (2002).

Once the model is specified, the next step is to reveal its main properties such as existence and stability of the steady states, existence of periodic solutions, solution boundedness, invariant manifold(s), etc. Careful analytical study of these issues often requires application of some rather advanced mathematical techniques; cf. Part II. The goal of this analysis is twofold. First, the model must be biologically reasonable and thus should exclude some obviously artificial situations (such as, for instance, population growth from a vanishingly small value of the population density). Second – and this is, in fact, the principal idea of a mathematical modeling approach – a change in the model properties with respect to a change in a certain controlling parameter is usually assumed to reflect the changes in the dynamics of the given natural (eco)system and thus has immediate biological implications.

The system (2.1) creates an appropriate modeling framework in the case of a “well-mixed” community in a homogeneous environment, i.e., when the community may be in all circumstances regarded as spatially homogeneous. Obviously, this is not always the case, and this affects the model choice. For instance, the spatial structure of a given population or community can be predefined by the environmental heterogeneity. In the case of small environmental gradients, a relevant mathematical model can still be space-continuous; however, in an extreme case of large environmental gradients or a fragmented habitat, a space-discrete approach will sometimes be more insightful. A mathematical model would then consist of a few systems such as (2.1) where different systems describe the dynamics of different subpopulations, being coupled together due to migration between the habitats (Jansen and Lloyd, 2000; Jansen, 2001; Petrovskii and Li, 2001).

A separate branch of space-discrete models is made by metapopulation models (Gilpin and Hanski, 1991; Hanski, 1999), where the state of the “metacom-

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2In the time-discrete case, a model would consist of coupled difference equations or maps; cf. Comins et al. (1992); Allen et al. (1993).
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“Community” may be described by variables other than population density, e.g., giving a proportion of all sites where the given species is present.

Another source of spatial heterogeneity is the formation of self-organized patterns as a result of inter- and intra-specific interactions, and this is going to be the main focus of this book.

Obviously, for the spatial aspect of population dynamics to be nontrivial, there must exist a mechanism of population redistribution in space. The most common one is due to individual motion. The motion can be either active (i.e., due to self-motion) or passive (e.g., when an individual of an air-borne species is carried about by wind). Also, motion can take place with or without a preferred direction. The simplest (but yet biologically meaningful) case is given by random isotropic motion, i.e., diffusion. In Chapter 8, we will talk in more details about diffusion in the population dynamics; for the moment we simply assume that the spatial aspects can be taken into account by adding the diffusion terms to Equations (2.1):

\[
\frac{\partial U_i(R, T)}{\partial T} = D_i \nabla^2 U_i(R, T) + f_i(U_1, U_2, \ldots, U_n) \tag{2.2}
\]

\((i = 1, \ldots, n)\), where \(R = (X, Y, Z)\) is the position in space, \(D_i\) is the diffusion coefficient of the \(i\)th species, and \(\nabla^2\) is the Laplace operator:

\[
\nabla^2 = \frac{\partial^2}{\partial X^2} + \frac{\partial^2}{\partial Y^2} + \frac{\partial^2}{\partial Z^2}.
\]

The system (2.2) is a system of nonlinear partial differential equations and, as such, is a difficult mathematical object to study. Although some regular analytical approaches are available [e.g., see Petrovskii and Li (2006) and also Chapter 9 of this book], more often its properties are studied through computer simulations by means of solving Equations (2.2) numerically (cf. Thomas, 1995).

Correspondingly, the next important step is scaling. In analytical approaches it may be easier to work with the original systems (2.1) or (2.2) because it sometimes makes the interpretation of the results more straightforward. In numerical simulations, however, we actually work with numbers rather than with dimensional quantities. Therefore, it is more convenient to first transform a given model to a dimensionless form. Remarkably, it is always possible; the corresponding procedure is usually called either dimensions analysis or scaling. Indeed, the functions \(f_i\) depend not only on the population densities but also on a number of parameters, such as the birth/death rate(s), population carrying capacity(-ies), etc. These parameters provide an intrinsic scale for each of the variables. Some typical examples showing how to do it in practice are given below; a very general description of the procedure along

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\[It\ should\ be\ mentioned\ here\ that\ scaling\ by\ itself\ is\ a\ powerful\ method\ of\ analysis,\ and\ it\ often\ allows\ one\ to\ arrive\ at\ some\ important\ conclusions\ without\ even\ specifying\ the\ model;\ see\ Barenblatt\ (1996).\]
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with its application to a wide range of problems can be found in Barenblatt (1996).

A considerable part of this book, especially in Parts III and IV, will be concerned with the dynamics of a prey–predator system continuous in space and time. According to the general theoretical framework introduced above, it is described by the following equations:

\[
\frac{\partial U(R, T)}{\partial T} = D_1 \nabla^2 U(R, T) + P(U) - E(U, V), \tag{2.3}
\]

\[
\frac{\partial V(R, T)}{\partial T} = D_2 \nabla^2 V(R, T) + \kappa E(U, V) - \mu(V), \tag{2.4}
\]

where \(U\) and \(V\) are the population densities of prey and predator, respectively. For biological reasons, the corresponding functions \(f_1\) and \(f_2\) are now split to separate terms such as prey population growth \(P\), predation \(E\), and predator mortality \(\mu(V)\), and the coefficient \(\kappa\) is called the predation efficiency or conversion rate.

Prey–predator systems have been at the focus of mathematical biology for several decades, starting from the works by Lotka and Volterra, yet there is still a lot of controversy regarding the optimal choice of the predator functional response to the prey density, e.g., see Arditi and Ginzburg (1989) and van Leeuwen et al. (2007). Presently, the forms that are used most often are the so-called Holling types II and III, that is,

\[
(a) \quad E(U, V) = A \frac{UV}{U + H} \quad \text{and} \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad 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forms:

\[
\frac{\partial u(x, t)}{\partial t} = \epsilon \frac{\partial^2 u}{\partial x^2} + u(1 - u) - \frac{uv}{u + h}, \quad (2.8)
\]

\[
\frac{\partial v(x, t)}{\partial t} = \frac{\partial^2 v}{\partial x^2} + k \frac{uv}{u + h} - mv, \quad (2.9)
\]

where \( k = \frac{\kappa A}{\alpha} \), \( m = \frac{M}{\alpha} \), \( h = \frac{H}{K} \), and \( \epsilon = \frac{D_1}{D_2} \) are dimensionless parameters. Correspondingly, the properties of \( u(x, t) \) and \( v(x, t) \) now depend not on all the original parameters separately but only on their combinations \( k \), \( m \), \( h \), and \( \epsilon \).

Note that (2.8)–(2.9) contain fewer parameters than the original Equations (2.6)–(2.7), i.e., four instead of eight. A decrease in parameter number due to the transition to dimensionless variables is a typical result. It gives another advantage of scaling: The less parameters a given model contains, the more effective is its study by means of numerical simulations.

An important remark that should be made here is that in most cases the choice of dimensionless variables is not unique. Indeed, talking about the prey–predator system (2.6)–(2.7), it is readily seen that we can introduce dimensionless variables as \( t = \frac{T}{\kappa AK/H} \), \( x = \frac{X}{\kappa AK/(D_2 H)^{1/2}} \), \( u = \frac{U}{K} \), and \( v = \frac{V}{\kappa K} \). From Equations (2.6)–(2.7), we then obtain:

\[
\frac{\partial u(x, t)}{\partial t} = \epsilon \frac{\partial^2 u}{\partial x^2} + a u(1 - u) - \frac{uv}{1 + \alpha u}, \quad (2.10)
\]

\[
\frac{\partial v(x, t)}{\partial t} = \frac{\partial^2 v}{\partial x^2} + \frac{uv}{1 + \alpha u} - mv, \quad (2.11)
\]

where \( \alpha = \frac{K}{H} \), \( a = \frac{\eta HK}{\kappa A} \), \( m = \frac{MH}{\kappa AK} \), and \( \epsilon = \frac{D_1}{D_2} \).

Unfortunately, there are no accepted standards regarding the scaling procedure, and which of these two schemes to choose is to a large extent a matter of personal preference. Different authors use different schemes, and that often makes quantitative comparisons between published results rather difficult, even in the case when the principal implications are clear.

Prey–predator interactions are ecologically meaningful but surely they do not exhaust all possible types of interspecific interactions. Another important case is given by competition. In contrast to the prey–predator system, where predation is beneficial for one species and detrimental for the other, competition hampers the population growth of both involved species. Mathematically, it is usually described by a bilinear function, i.e., a general time- and space-continuous model of a community of \( N \) competing species looks as

\[\text{There can be some variations within each of these two approaches, such as using } D_1 \text{ instead of } D_2 \text{ for scaling } X, \text{ or } X \text{ can be scaled to the domain length, etc.}\]
follows:

\[
\frac{\partial U_i(R, T)}{\partial T} = D_i \nabla^2 U_i(R, T) + \left( \alpha_i - \sum r_{ij} U_j \right) U_i ,
\]

\(i = 1, \ldots, N\)

(cf. May, 1973), where \(\alpha_i\) is the inherent population growth rate of the \(i\)th species and \(R = (r_{ij})\) is the so-called “community matrix.” In a general case, \(r_{ij} \neq r_{ji}\) because different species possess different abilities for competition.

The properties of the competing species system appear to be significantly different from those of a prey–predator system. In particular, it is readily seen that, contrary to a prey–predator system where cycles are generic, a nonspatial system of two competing species cannot have periodic solutions but only equilibrium states.

In agreement with the general idea of scaling, the number of parameters in (2.12) can be reduced (by means of choosing relevant scaling factors for the population densities, space, and time) from \(N^2 + 2N\) to \(N^2 + N - 2\). One particular case will be considered in Section 10.3.

Note that, in the general systems like (2.1) and/or (2.2), the state variables \(U_1, \ldots, U_N\) must not necessarily be regarded as the densities of species 1 to \(N\), respectively. Instead, at least some of them may have the meanings of the densities of subpopulations of the same species, provided that given species is in some way structured. In this book, we will be especially interested in one particular case when a given species is affected by an infectious disease, and thus its population is split to susceptible, infected, and removed/recovered parts.

Remarkably, a mathematical description of the disease dynamics appears to be similar (at least, in the simplest cases) to that of the prey–predator system. Indeed, denoting the densities of the susceptibles and infected as \(S\) and \(I\), respectively, instead of the system (2.3)–(2.4) we obtain

\[
\frac{\partial S(R, T)}{\partial T} = D_S \nabla^2 S(R, T) + P(S, I) - \Sigma(S, I) - M_SS ,
\]

\[
\frac{\partial I(R, T)}{\partial T} = D_I \nabla^2 I(R, T) + \Sigma(S, I) - M_I I ,
\]

where \(\Sigma\) describes the disease transmission and other terms have the same meanings as above. In a general case, the “kinetics” of the disease transmission can be very complicated (cf. Fromont et al., 1998); however, having assumed that it is described by the so-called mass-action law, \(\Sigma = \sigma SI\), and that only susceptibles can produce offspring, the system (2.13)–(2.14) is then reduced to the classical Lotka–Volterra prey–predator model.

Finally, the systems (2.1) and/or (2.2) can be of a mixed origin, i.e., some of the state variables may correspond to population densities and others to the subpopulations of the infected species. In that case, we arrive at an
ecoepidemiological system. We will provide a detailed consideration of the corresponding models in Chapter 7.

All the models mentioned above are deterministic in the sense that they are described by deterministic equations. As such, they largely neglect the impact of stochasticity and noise. To distinguish between the cases when stochasticity may or may not be important is a highly nontrivial problem. Indeed, although the population dynamics is intrinsically stochastic, in many cases it is very well described by the deterministic equations. In practice, the choice of the model (i.e., deterministic or stochastic) is often purely heuristic. Also, it of course depends on the goals of the study. In order to take into account possible effects of stochasticity and noise, one can either apply statistical modeling (cf. Czárán, 1998) or include stochastic terms/factors into the deterministic model. The latter approach will be used in Part IV of this book, where we make an insight into the issue and reveal, by means of considering several specific cases, how the system’s dynamics can be modified by the impact of stochasticity.
Part II

Models of temporal dynamics
Chapter 3

Classical one population models

Population theory, with its long history, dates back to the Malthus model, formulated for economic reasons in the early nineteenth century, predicting the population problems due to an exponential growth not supported by unlimited resources. The basic equation was then corrected by Verhulst (1845; 1847) to compensate for the shortcomings of the earlier model. The logistic equation states instead the existence of a horizontal asymptote to which the population tends as time flows. This asymptote has a biological interpretation, namely the carrying capacity the environment possesses, to support the population described by the model.

Later on, in the twenties of the past century, the works by Lotka and Volterra extended the mathematical modeling to prey–predator situations (Lotka, 1925; Volterra, 1926a,b). Since then the field has developed, and the biomathematical literature is now very large.

In this chapter we present the basic models in population theory, at first considering an isolated population and in Section 3.2 models including migrations. The purpose of Section 3.1 is to introduce some basic notation and terminology, while in Section 3.2 the most elementary bifurcations will naturally arise from the analysis. We stress the fact that in some cases the effort is made to keep the considerations simple enough to help the less familiar reader to grasp the basic concepts. Also, occasionally very elementary examples are made in both this as well as in subsequent chapters. In Section 3.3, simple discrete versions of the basic one-population models are discussed, to pave the way for the topic of the following Section 3.4, a short introduction of chaos.

3.1 Isolated populations models

Consider a single population in an idealized situation, in which neither deaths nor migration occur. Since then the only process that can happen is reproduction, the population must be a function of time $T$, i.e., $U \equiv U(T)$. Taking the time unit to be months, for instance, suppose we count the individuals at different times and find the following results:

\[ U(0) = 1000, \quad U(10) = 1200, \quad U(20) = 1440. \]
If we take the population differences first with respect to the origin and then with respect to the previous measurement, we find
\[ U(10) - U(0) = 200, \quad U(20) - U(0) = 440, \quad U(40) - U(20) = 240. \]

One question we may ask is, how do we know the estimate size of the population at intermediate times, such as \( U(1) \) and \( U(2) \)? An intuitive answer would then be \( U(1) = 1020, \quad U(2) = 1044. \) The reason is that we tend to think linearly in terms of time. If 200 newborns occur in a time of 10 months, we expect 20 in just one. In other words, we are saying that the monthly increment in population is \( \delta_U = 20. \)

Suppose now we take a second population, \( V(T) \), in the same environmental conditions, the situation from the previous case differing only in the initial conditions. We would then find \( V(0) = 2000, \quad V(10) = 2400, \quad V(20) = 2880, \) again as 1000 individuals contribute 200 newborns in 10 months. The reason is once again that we also think linearly in terms of the population size. However, in this case we have \( V(10) - V(0) = 400, \quad V(20) - V(0) = 880, \quad V(40) - V(20) = 480. \)

Thus, comparing the examples, the monthly population increment \( \delta_V = 40 \neq \delta_U \) must be a function of population size \( V, \delta_V \equiv \delta_V(V). \)

Consider a third example, again in different environmental circumstances, but still not accounting for deaths and migrations, in which the population \( F \) is counted at other times giving the following data:
\[ F(T_0) = 100, \quad F(T_1) = 105, \quad F(T_2) = 110. \]

We may now ask which of the two populations \( F \) or \( U \) reproduces faster. Now, taking differences, we find
\[ F(T_1) - F(T_0) = 5, \quad F(T_2) - F(T_1) = 5, \quad F(T_2) - F(T_0) = 10, \]
and these absolute numbers seem to be smaller than the previous ones, so that we may think that \( F \) reproduces the slowest. But if time is now measured in weeks, so that \( T_1 = 1, \ T_2 = 2, \) and we make the proportions, clearly \( F \) reproduces the fastest of all populations. Indeed, at the end of one month, keeping the same growth proportion, we find \( F(1) = 120, \) and at the end of 10 months then \( F(10) = 300, \) assuming here that newborns do not start to reproduce in the whole period taken into account. The absolute increment is the same as for \( U, \) but here the percentage increase is clearly different. Indeed, in 10 months the increase of \( F \) was 200%, while the one of \( U \) was only 20%.

This indicates first of all that the population increment is also a function of time,
\[ \delta_F \equiv \Delta F \equiv F(T + \Delta T) - F(T) \equiv \delta_F(T). \]
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Secondly, we should take the reference value of the population into account, thus not speaking of absolute increases, but rather of percentage accruals, i.e., relative increases $\Delta F/F$.

The simplest form for the population population increment over time that we can write is then

$$\frac{1}{U} \frac{\Delta U}{\Delta T} \equiv \frac{1}{U} R(U) \equiv r_M(U) = r,$$

with $r_M = r$ denoting a constant. Dimensionally, $U$ is a number, and $R(U)$ is then a per capita rate, i.e., a per capita frequency of reproduction. The word “per capita” refers to the fact that we were naturally induced to divide by the whole population size $U$, to determine the individual reproduction rate. The function $r_M(U)$ is then the individual frequency of reproduction, in this case constant. Equivalently, the function $R(U)$ is thus a linear function of $U$, $R(U) = rU$. Finally, we can let the time interval tend to zero, to get a differential equation. We have thus obtained the Malthus model,

$$\frac{dU}{dT}(T) = rU(T),$$  \hspace{1cm} (3.1)

in which case the (constant) function $r_M = r$ denotes the per capita instantaneous reproduction rate. More generally, as seen before, it may be a function of time. The equation is dimensionally sound as on the left there is a rate measured in numbers by time, and on the right a frequency times a number. Its solution is easily obtained:

$$U(T) = U(0) \exp(rT),$$  \hspace{1cm} (3.2)

with $U(0)$ denoting the initial value of the population. The problem with this model is that for $r > 0$, the solution goes to $\infty$, while for $r < 0$, it goes to zero.

To correct this behavior Verhulst proposed the following modification. The constant $r$ should be replaced by a function, namely, the simplest nonconstant function of the population, $r_L(U)$, which implicitly becomes a function of time. This means that it should be taken as a linear function of $U$, namely, $r_L \equiv r_L(U) = r\left(1 - \frac{U}{K}\right)$. Notice indeed that the slope of this straight line must be negative, since otherwise $r_L$ would be bounded below by the earlier constant $r$, and therefore the solution of the corrected equation would also be bounded below by the exponential function of the Malthus equation; thus, for a positive slope, the modification will not solve the problem, because the solution would still diverge for large times. We thus have the logistic equation

$$\frac{dU}{dT}(T) = r \left(1 - \frac{U(T)}{K}\right) U(T),$$  \hspace{1cm} (3.3)

in which now geometrically $r$ has the meaning of the intercept at the origin and therefore must be nonnegative; otherwise, as seen above, the population
Spatio-temporal patterns in ecology and epidemiology will vanish. The solution of (3.3) can be analytically evaluated, by separation of variables,

\[
\frac{dU}{(1 - \frac{U}{K})U(T)} = \frac{dU}{U(T)} + \frac{dU}{K(1 - \frac{U}{K})} = [d\ln(U) - d\ln(1 - \frac{U}{K})] = d\ln(\frac{U}{1 - \frac{U}{K}}) = rdT,
\]

followed by integration, to give

\[
\ln\frac{U}{1 - \frac{U}{K}} = rT + C, \quad \frac{U}{1 - \frac{U}{K}} = \exp(C) \exp(rT) \equiv C_0 \exp(rT)
\]

and finally

\[
U(t) = \frac{C_0 K \exp(rT)}{K + C_0 \exp(rT)}. \tag{3.4}
\]

Thus, for \( T \to \infty \), the solution tends to a horizontal asymptote, \( U \to K \). The value of this constant represents the population that the environment can support in the long run, and it is called the carrying capacity. It also represents the root of the linear function \( r(U) \) in the \( RU \) phase plane. Notice that \( R(U) \geq 0 \) for \( 0 \leq U \leq K \), and conversely for \( U \geq K \). It thus follows that in the logistic equation, \( \frac{dU}{dT} \geq 0 \) for \( 0 \leq U \leq K \), and \( \frac{dU}{dT} \leq 0 \) for \( U \geq K \), thus implying that \( U \) must grow when it has a value below \( K \), while it should decrease when it exceeds it. These qualitative results then confirm the analysis we performed above: In both cases the population ultimately tends to the value \( K \). Notice finally that, as mentioned earlier, for \( r < 0 \) the solution obtained also shows that \( U \to 0 \) as \( T \to \infty \), allowing us to then discard this case for the reproduction parameter.

A further second order correction consists in taking a parabola instead of a straight line for the reproduction function, namely,

\[
r_A \equiv r_A(U) = r(U - B)(1 - \frac{U}{K}), \quad 0 < B < K,
\]

where again we take \( r > 0 \) in view of the previous discussion, so that the differential equation now takes into account the impact of the Allee effect:

\[
\frac{dU}{dT}(T) = r(U - B)(1 - \frac{U}{K})U(T). \tag{3.5}
\]

In this model the reproduction function \( r_A \) becomes negative for population values smaller than \( B \). This models the well-known fact that for small populations that live in a large environment in which individuals cannot easily find each other, reproduction becomes difficult. The phase plane analysis shows that the equilibria now are the same as for the logistic model, the origin, and the carrying capacity \( K \), but there is also the equilibrium at the value \( B \).
The rate of change of the population is positive only for \( B < U < K \), so that again \( K \) is a stable equilibrium since \( U \) tends to grow if it is \( B < U < K \) and decreases to this value if it exceeds it. For the equilibrium \( B \), the reverse is true, thus, as just seen, \( U \) increases away from it if it is larger than \( B \), but if \( 0 \leq U < B \), then the derivative is negative so that in such case \( U \to 0^+ \), meaning that ultimately the population vanishes, but from positive values. Thus, in the above-described environment, the population will be wiped away when it drops below a threshold population value represented by \( B \). A further difference with the logistic model is given by the fact that here \( K \) is not a globally asymptotically equilibrium, while in the logistic case it is. Starting from any value of \( U \), indeed, in the latter case \( K \) is ultimately reached, while for the Allee model (3.5), this is not true for \( 0 \leq U \leq B \).

### 3.1.1 Scaling

Let us consider now the effect of scaling. Taking into account the Malthus model, with a net growth rate \( r \in \mathbb{R} \) that may be positive or negative, let us rescale the variables by setting

\[
U(T) = \beta u(t), \quad T = \alpha t.
\]  

(3.6)

The constants \( \beta \) and \( \alpha \) must be positive, because biologically we account only for nonnegative populations and since we look at the future evolution of the system. By suitably differentiating we find

\[
\frac{dU(T)}{dT} = \frac{\beta}{\alpha} \frac{du(t)}{dt},
\]

(3.7)

and substituting into Malthus equation upon simplification we have

\[
\frac{du(t)}{dt} = \alpha ru(t).
\]

(3.8)

In view of \( \alpha > 0 \) and \( r \in \mathbb{R} \), we cannot impose \( r\alpha = 1 \), but rather consider the two cases \( r\alpha = \pm 1 \), i.e., work separately with the two equations

\[
\frac{du(t)}{dt} = u(t), \quad \frac{du(t)}{dt} = -u(t).
\]

This is not practical, so in the case of unrestricted net growth rate for the Malthus model, adimensionalization does not bring too much of an advantage. In the case of the logistic model, instead we must have \( r > 0 \), otherwise the model would show the population to have a negative carrying capacity to keep the “crowding competition” term negative, i.e., to still ensure \(-\frac{r}{K}U^2 < 0 \). In any case, the growth of the population would always be negative, leading to its disappearance, and thus vanifying the benefit of the introduction of the correction term. In the logistic case with \( r > 0 \), scaling can be performed...
FIGURE 3.1: The rows refer to the three models considered: the first row to the Malthus model (3.1), the second one to the logistic one (3.3), and the third one to the Allee model (3.5). The first column contains the picture of the per capita reproduction function $r(U)$, the second one the reproduction function $R(U)$, and the last one the analytic solution of each model for three different initial conditions and for each value of the function $r(U)$ plotted in the corresponding row. In the logistic and Allee models, however, we do not show the case $r < 0$ as it is not meaningful. For the Malthus growth, a negative net growth function leads to the disappearance of the population. For the logistic model, all solutions tend to the horizontal asymptote; for the Allee model, this is the case only if the initial conditions are above the threshold value $B$, corresponding to the extra root, with respect to the logistic case, of the reproduction function in the second column.

with the same substitutions indicated above to get

$$\frac{du(t)}{dt} = \alpha u(t) \left( 1 - \frac{\beta u(t)}{K} \right) = u(1 - u),$$

(3.9)
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with the choice $\beta = K$ and $\alpha = r^{-1}$. For the Allee model (3.5), again assuming $r > 0$ and using the same substitutions, we find

$$\frac{du(t)}{dt} = \alpha ru(t)(\beta u(t) - B) \left(1 - \frac{\beta u(t)}{K}\right) = u(u - b)(1 - u),$$

(3.10)

choosing $\beta = K$, $\alpha = (Kr)^{-1}$ and setting $b = BK^{-1} < 1$.

3.2 Migration models

Consider now a population $U(T)$ that does not reproduce; rather, it is subject only to a constant rate migration $m$ and competition for resources. Its evolution equation then reads

$$\frac{dU}{dT} = m - U^2 \equiv f(U).$$

(3.11)

From the logistic model we take the quadratic death rate term, while the constant term represents the external feed (or loss) of new individuals. Indeed, if we take $m < 0$, the first term represents an emigration term, and seeking equilibria, there is no way of zeroing out the right-hand side, as $U^2 = -m$ has no real roots. Thus the population $U$ will decrease at all times as in the $Uf$ phase plane, $f(U) = 0$ is a parabola always below the horizontal axis. For $m = 0$, i.e., no migration of any sort occurs, the same parabola will move upwards and become tangent to the $U$ axis at the origin. The origin will then become a semistable equilibrium, but the behavior of the model will essentially be unchanged. A more interesting situation arises for an immigration rate $m > 0$. In such a case there are two real roots, $U_{\pm} = \pm \sqrt{m}$. The parabola $f(U)$ is above the $U$ axis in between them, so that $f(U) \geq 0$ for $U_{-} \leq U \leq U_{+}$, and therefore $U$ grows in such an interval and decreases for $U \geq U_{+}$. This means that the equilibrium $U_{+}$ is stable, and conversely $U_{-} < 0$ is unstable, but since it is unfeasible, we disregard it. When $m$ increases, so does $U_{+}$. By drawing a picture of $U_{+}$ as a function of $U$ we obtain what is called a bifurcation diagram; see Figure 3.2.

We can also consider a more complex situation, a logistic model in which we allow the growth rate $r$ to be of either sign. In such a case, the model should not be written as (3.3) of Section 3.1 because the term containing the carrying capacity would become positive for $r < 0$, which does not correspond to the population pressure. We then write instead

$$\frac{dU}{dT} = aU - bU^2,$$

(3.12)
FIGURE 3.2: Bifurcation diagram for the logistic model (3.11), with immigration and no reproduction, \( \frac{dU}{dT} = m - U^2 \equiv f(U) \). The stable feasible branch is represented by \(*\); we also show the infeasible unstable branch \(\circ\).

with \(a \in \mathbb{R}\) and \(b \geq 0\). Let us rescale the equations by introducing new positive parameters \(\alpha\) and \(\beta\), so that \(U = \beta u\) and \(T = \alpha t\) give

\[
\frac{dU}{dT} = \frac{du}{dt} = \frac{\beta}{\alpha} \frac{du}{dt}
\]

and the rescaled model becomes

\[
\frac{du}{dt} = a\alpha u - b\alpha\beta u^2.
\] (3.13)

Now we can choose the free parameters so as to simplify the equation, but the choice \(a\alpha = 1\) is not possible, because in such a case the growth rate \(a\) would be positive. If we want to account for \(a \in \mathbb{R}\), we then need to take \(b\alpha\beta = 1\), i.e., \(\beta = \frac{1}{b\alpha}\), and therefore we must then set \(a\alpha = \frac{\alpha}{\beta} \equiv \mu \in \mathbb{R}\). The rescaled model then reads

\[
\frac{du}{dt} = \mu u - u^2 \equiv F(u).
\] (3.14)

The equilibria are the origin and the point \(u_m = \mu \in \mathbb{R}\). The parabola \(F(u)\) is positive once again in between the roots, implying with the discussion as above that the right root is always stable and the left one is unstable. For \(\mu < 0\), the right root is the origin, while for \(\mu > 0\) it is the point \(u_m > 0\). At \(\mu = 0\), the origin is a double root, a semistable equilibrium in this case, as it seems stable if approached from the right and unstable from the left.

If we draw the roots as function of the parameter \(\mu\), we find the diagram of
a transcritical bifurcation. Notice that for $\mu < 0$ the stable branch is given by the origin and the bisectrix is unstable, while for $\mu > 0$ the two branches exchange their stability properties. Clearly, however, for $\mu < 0$, the bisectrix would give an unfeasible equilibrium; see Figure 3.3.

![Figure 3.3](image-url)

**Figure 3.3:** Logistic model (3.14) with reproduction and no immigration, $\frac{du}{dt} = \mu u - u^2 \equiv F(u)$. Bifurcation diagram as a function of reproduction rate $\mu$. The stable feasible branches are represented by $*$; the partly infeasible unstable branch is denoted by $\circ$.

We now consider a particular case of (3.5), namely,

$$\frac{dU}{dT} = \mu U - U^3,$$

(3.15)

with $\mu \in \mathbb{R}$, obtained from (3.5) again relaxing an assumption. We allow here a negative threshold $B$, so that $K + B = 0$, and rescale as indicated above, choosing $\alpha \beta^2 \tau = K$ and setting $\mu = -B \alpha \tau$. Note that, although (3.15) formally gives a particular case of the Allee model (3.5), the population growth described by (3.15) is no longer damped by the Allee effect. It is readily seen that for any $B \leq -K$, the right-hand side of (3.15) becomes a convex function and thus is qualitatively similar to the logistic growth. Correspondingly, Equation (3.15) is sometimes called a model with a “generalized logistic growth.”

Figure 3.4 shows that we obtain a pitchfork bifurcation diagram.
3.2.1 Harvesting

We now consider a term $H(U)$ denoting harvesting of the population,

$$\frac{dU(T)}{dT} = R(U) - H(U).$$

The exploitation term can generally be chosen in one of the following ways:

$$H(U) = h, \quad H(U) = hU, \quad H(U) = h \frac{U}{A + U},$$

or

$$H(U) = h \frac{U^2}{A + U^2},$$

(3.16)

where $A$ and $h$ are positive parameters. The first two expressions in (3.16) correspond to constant and linear exploitation, respectively, allowing for possibly unbounded harvests if the population is large enough. The last two assume an upper bound on the return, in a sense analogous to the limitations provided by the logistic equation. They differ essentially near the origin, the former starting with a slope at the origin, while the last one has zero derivative there. Compare Figures 3.11 and 3.13.

Combining now different types of harvestings of the population, let us look in detail at its consequences, with respect to the different growth mechanisms.
introduced before. For the Malthus model, with the net growth rate \( \tilde{r} \in \mathbb{R} \) unrestricted in sign, we have

\[
\frac{dU(T)}{dT} = \tilde{r}U(T) - h.
\]  

(3.17)

After adimensionalization again with \( U(T) = \beta u(t) \), \( T = \alpha t \), we find

\[
\frac{du(t)}{dt} = \alpha \tilde{r}u(t) - \frac{\alpha}{\beta} h
\]  

(3.18)

and setting \( \beta = \alpha h \), \( r = \alpha \tilde{r} \), we finally end up with

\[
\frac{du(t)}{dt} = ru(t) - 1, \quad r \in \mathbb{R}.
\]  

(3.19)

The right-hand side is thus linear in \( u \), positive for \( u > u_0 \equiv r^{-1} \), and negative conversely, so that \( u \) grows if it has a larger value than \( u_0 \), and vice versa. This fact renders \( u_0 \), if feasible, i.e., for \( r > 0 \), an unstable equilibrium while the origin in a such case is a stable one, contrary to what happens in the classical model without harvesting. Thus exploitation changes the model’s behavior. For \( r < 0 \), as for the original Malthus model, \( O \) is always stable on biological grounds, as negative populations make no sense. Mathematically, it would not even be an equilibrium, as in a such case the only one would be \( u_0 < 0 \), which would be infeasible. The reader should compare the very simple bifurcation diagram as a function of \( r \) reported in Figure 3.5 with the above considerations.

Consider now a linear harvesting term

\[
\frac{dU(T)}{dT} = rU(T) - hU(T), \quad r \in \mathbb{R}.
\]  

(3.20)

Scaling as before, we find

\[
\frac{du(t)}{dt} = \theta u(t), \quad \theta = \alpha(r - h) \in \mathbb{R}.
\]  

(3.21)

In this case, then, we see that the Holling type I exploitation leads to the same original Malthus model, with a different growth function. The corresponding bifurcation diagram as a function of \( \theta \) is again elementary to construct. \( O \) is unstable for \( \theta > 0 \), and conversely it is always stable; see Figure 3.6.

The Holling type II harvesting gives

\[
\frac{dU(T)}{dT} = \tilde{r}U(T) - h \frac{U(T)}{A + U(T)}, \quad A, h, \tilde{r} \in \mathbb{R}.
\]  

(3.22)

Scaling in this case gives

\[
\frac{du(t)}{dt} = \alpha u(t) \left[ \tilde{r} - \frac{h}{A + \beta u} \right],
\]
from which, setting $\beta = A$, $\alpha = \frac{A}{h}$, and $r = \alpha \tilde{r}$, we find

$$\frac{du(t)}{dt} = u \left[ r - \frac{1}{1 + u} \right].$$

(3.23)

The equilibria are once again $O$ and the point $u_* = \frac{1}{r}$, which is feasible for $0 < r < 1$. In such a case, however, it is unstable; for $r > 1$, the origin is unstable and for $r < 1$, it is stable. The bifurcation diagram is now composed by a branch of a hyperbola in addition to the line $u = 0$; see Figure 3.7.

We finally consider harvesting using the Holling type III form, on the simple Malthus model:

$$\frac{dU(T)}{dT} = \tilde{r} U(T) - h \frac{U^2(T)}{A + U^2(T)}, \quad A, h, \tilde{r} \in \mathbb{R}.$$  

(3.24)

The scaling procedure with $\beta = \sqrt{A}$, $\alpha = \beta h^{-1}$ and $r = \tilde{r} \alpha$ now leads to

$$\frac{du(t)}{dt} = ru - \frac{u^2}{1 + u^2}.$$  

(3.25)

The equilibria in this case are $O$ once again and $u_{\pm} = \frac{1}{2 \tilde{r}}(1 \pm \sqrt{1 - 4r^2})$, which exist and are feasible for $-\tilde{r} < r < \tilde{r}$, with $\tilde{r} = \frac{1}{2}$. Analysis of the trajectories shows that $O$ for $r < 0$ is stable, for $0 < r < \tilde{r}$ it becomes unstable, and there are also the two equilibria found above, of which $u_-$ is stable and $u_+$ is unstable, for $r > \tilde{r}$ we find only $O$, which retains its instability. The
FIGURE 3.6: Bifurcation diagram for the Malthus model (3.21) with Holling type I harvesting: stable branch $\ast$, unstable branch $\circ$.

The bifurcation diagram (see Figure 3.8) exhibits once again an unstable branch of a hyperbola in the interval $0 < r < \tilde{r}$, which coalesces with the stable branch emanating from $O$ at the point $\tilde{u} = 1$. The latter is easily calculated by imposing tangency between the straight line and the Holling type III term, yielding the equations

$$ru = \frac{u^2}{1 + u^2}, \quad r = \frac{2u}{(1 + u^2)^2}.$$

Substitution of one into the other easily leads to $1 + u^2 = 2$, from which the values of $\tilde{u}$ and $\tilde{r}$ are obtained, in agreement with what was formerly obtained.

We now will consider the logistic model subject to the various forms of exploitation. As mentioned before, here we will assume $r > 0$, which will give an alternative choice for the rescaling. In particular, we will construct bifurcation diagrams now as functions of the harvesting effort $h$. We have, under constant effort,

$$\frac{dU(T)}{dT} = rU(T) \left(1 - \frac{U(T)}{K}\right) - \tilde{h}, \quad \tilde{h} > 0. \quad (3.26)$$

Rescaling with the usual substitutions and setting $\beta = K$, $\alpha = r^{-1}$, and $h = \tilde{h}r^{-1}$ gives the adimensional model

$$\frac{du(t)}{dt} = u(1 - u) - h. \quad (3.27)$$
Clearly the right-hand side represents the logistic parabola through the origin and the normalized carrying capacity $K = 1$ pulled down by an amount $h$. Its roots are intersections of this parabola with the horizontal straight line at height $h$. They then move toward each other the higher $h$ becomes, retaining their stability properties, stable on the right and unstable on the left, until they coalesce and then disappear, at which point the origin becomes the only stable equilibrium of the system. The coalescence point coincides with the vertex of the parabola, thus it occurs for $h = \frac{1}{4}$. The bifurcation diagram (Figure 3.9) thus shows a parabola with a stable and an unstable branch, while the origin for $h > 0$ is always stable. Biologically, this constant harvesting effort thus has on the equilibria the same effect of the Allee model without exploitation. Notice also that in fisheries models the level of the harvesting effort at the stable equilibrium gives the so-called maximum sustainable yield of the exploitation.

We now turn to Holling type I harvest namely,

$$\frac{dU(T)}{dT} = rU(T) \left(1 - \frac{U(T)}{K}\right) - \tilde{h} U(T), \quad \tilde{h} > 0.$$  \hspace{1cm} (3.28)\

Scaling gives

$$\frac{du(t)}{dt} = u(1 - h - u),$$  \hspace{1cm} (3.29)\

with the choices $\alpha r = 1$, $\beta = K$ and setting $h = \tilde{h} \alpha$. In this case, the stable equilibrium at $u = 1$ for $h = 0$ moves leftwards linearly with $h$, as it is given
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by \( u_s = 1 - h \), retaining its stability, until it hits the origin at \( h = 1 \). This corresponds to the logistic and harvesting curves to be tangent to each other at \( O \). The origin that was formerly unstable at this point becomes stable and remains so for every \( h \geq 1 \). Figure 3.10 shows the corresponding bifurcation diagram.

The Holling type II exploitation gives

\[
\frac{dU(T)}{dT} = rU(T) \left( 1 - \frac{U(T)}{K} \right) - \tilde{h} \frac{U(T)}{A + U(T)}, \quad A, \tilde{h} > 0. \tag{3.30}
\]

Upon rescaling we find for \( \alpha = r^{-1}, \beta = A, \theta = \frac{A}{K} \),

\[
\frac{du(t)}{dt} = u(1 - \theta u) - h \frac{u}{1 + u} \equiv P(u) - \bar{H}(u). \tag{3.31}
\]

Notice that the slopes of the parabola \( P \) and the hyperbola \( \bar{H} \) can be evaluated so that at the origin they are, respectively,

\[
\frac{dP}{du} = 1 - 2\theta u, \quad \frac{d\bar{H}}{du} = \frac{h}{1 + u^2}, \quad \left. \frac{dP}{du} \right|_{u=0} = 1, \quad \left. \frac{d\bar{H}}{du} \right|_{u=0} = h.
\]

The equilibria are given by

\[
u_{\pm} = \frac{1}{2\theta} \left[ 1 - \theta \pm \sqrt{(\theta + 1)^2 - 4h\theta} \right].
They are real only for \( h < h_s \equiv \frac{(\theta + 1)^2}{4\theta} \). In such a case, the rightmost one, \( u_+ \), is stable, as the hyperbola given by the harvesting term lies above the logistic parabola for \( u > u_+ \), and conversely for \( u < u_- \). The leftmost one, \( u_- \), will instead be unstable. Feasibility is better described geometrically. We need to ensure that the curves \( P(u) \) and \( \tilde{H}(u) \) meet in the first quadrant. For low values of \( h \), this is always ensured, \( 0 < h < 1 \), but for \( h > 1 \), this happens only if the value of the horizontal asymptote of \( \tilde{H} \) is below the vertex of the parabola \( P \), of height \( (4\theta)^{-1} \). Thus, for \( 1 < h < (4\theta)^{-1} \), there are the intersections \( u_\pm \) described above, and for this to occur, naturally we need to require \( \theta < \frac{1}{4} \). In the contrary case, no intersection between \( P \) and \( \tilde{H} \) is possible for \( h > 1 \), and the origin becomes the only equilibrium of the system. Figure 3.11 shows these situations. Notice also that the origin is always a stable equilibrium when \( u_\pm \) are feasible, and is instead unstable for \( 1 > h > 0 \).

The bifurcation diagram is reported in Figure 3.12.

The logistic growth subject to Holling type III exploitation is ruled by the equation

\[
\frac{dU(T)}{dT} = rU(T) \left( 1 - \frac{U(T)}{K} \right) - \frac{\bar{h}}{A} U^2(T), \quad A, \bar{h} > 0. \tag{3.32}
\]

The rescaling is now done with \( \alpha = r^{-1}, \beta = \sqrt{A} \), and setting \( h = \frac{\bar{h}}{r\sqrt{A}} \), \( \phi = \frac{\sqrt{A}}{K} \), to get

\[
\frac{du(t)}{dt} = u(1 - \phi u) - h \frac{u^2}{1 + u^2} \equiv u[\tilde{\ell}(u) - \tilde{H}(u)]. \tag{3.33}
\]
FIGURE 3.10: Bifurcation diagram for the logistic model (3.29) with linear harvesting: stable branch *, unstable branch °.

This shows that $O$ is again an equilibrium while the other ones come from solving $\hat{\ell}(u) = \hat{H}(u)$. The analysis of $\hat{H}(u)$ shows that it has a maximum located at $(1, \frac{1}{2}h)$ and an inflection point at $Q \equiv (\sqrt{3}, \frac{\sqrt{3}}{4}h)$. We can then choose $\hat{\ell}(u)$ through $Q$ and with a height at the origin located between the heights of the two former points, so that then $\frac{1}{2}h > 1 > \frac{\sqrt{3}}{4}h$. These give the interval $2 < h < \frac{4}{\sqrt{3}}$. The condition ensuring that $\tilde{\ell}(u)$ goes through $Q$ is easily found to be $\phi = \frac{1}{\sqrt{3}} - \frac{h}{4}$, and the constraint $\phi > 0$ is satisfied since it is equivalent to the former requirement $1 > \frac{\sqrt{3}}{4}h$. Thus, in these conditions there are three intersections, otherwise just one. The two different situations are shown in Figure 3.13. The origin is always an unstable equilibrium of the system. If there is only one other equilibrium, it is then stable (Figure 3.14). If there are three more, the intermediate of the two is unstable, while the other two are stable (see Figure 3.15).

Notice also that Figure 3.15 shows the phenomenon of hysteresis. Namely, starting from the population being exploited at the carrying capacity in absence of harvesting, $h = 0$, the equilibrium is the uppermost on the vertical axis. Increasing the exploitation, the stable upper branch of the bifurcation is followed, with the equilibrium decreasing steadily and slowly for increasing $h$. However, when $h \approx .85 \equiv h^c$, the upper critical level, the two stable and unstable branches coalesce, the upper equilibrium for larger harvesting efforts then disappears, and suddenly the population moves toward the only stable equilibrium, which is now situated very near the horizontal axis. If we think
now to go back to the previous situation only by diminishing the harvesting to values just below the critical one, say at $h = 0.8$, the reached stable equilibrium on the lower branch of the bifurcation diagram moves upwards, but only by a very small amount. This is, of course, provided that the whole ecosystem has not collapsed before due to some external disturbances that push the population $u$ to zero. Thus we have the counter-intuitive fact that it is not enough to backtrack a little to push the system to its previous situation. It is not until $h = 0.2 \equiv h_c$, the lower critical value, that $u$ recovers, as in such a case following toward the left the lower branch of the bifurcation diagram, the lower stable equilibrium vanishes and the system is pushed suddenly upwards to the stable branch near the carrying capacity. On increasing again the harvesting effort $h$, the cycle would then be repeated.

We finally turn to the analysis of the Allee model, coupled with harvesting:

$$\frac{dU(T)}{dT} = rU(T)(U(T) - B) \left( 1 - \frac{U(T)}{K} \right) - \tilde{h}, \quad \tilde{h} > 0. \quad (3.34)$$

The adimensionalization procedure in this case, letting $\beta = K$, $\alpha = (Kr)^{-1}$, and $h = \tilde{h}K^{-2r^{-1}}$, $b = \frac{B}{K}$, gives the rescaled model

$$\frac{du(t)}{dt} = u(u - b)(1 - u) - h. \quad (3.35)$$
Classical one population models

The equilibria are found geometrically to move from the values $b$ and 1 toward each other, until they coalesce and disappear, as the cubic in the right-hand side is “pulled down” by an amount $h$. Mathematically, $O$ is not a root of the right-hand side, but biologically it remains a stable equilibrium of the system. The other two retain their type of stability, namely, the one emanating from $b$ is unstable and the one coming from 1 remains stable. Figure 3.16 shows the bifurcation diagram.

For linear exploitation, the unscaled model reads

$$\frac{dU(T)}{dT} = rU(T)(U(T) − B) \left(1 - \frac{U(T)}{K}\right) - \hat{h}U(T), \quad \hat{h} > 0.$$  \hspace{1cm} (3.36)

Adimensionalization is carried out almost as above, with $\beta = K$, $\alpha = (Kr)^{-1}$, and $h = \hat{h}(Kr)^{-1}$, $b = \frac{B}{K}$, to get

$$\frac{du(t)}{dt} = u[(u - b)(1 - u) - h].$$  \hspace{1cm} (3.37)

This shows that $O$ is always an equilibrium and stable since linearization (i.e., dropping the higher order terms, which vanish faster near 0) gives $\frac{du}{dt} \approx -(b+h)u$. Then there are two other, coming from “pulling down” the parabola with roots in $b$ and 1, so that these roots, as above, approach each other and retain their stability properties. The corresponding bifurcation diagram is given in Figure 3.17.
FIGURE 3.13: Diagram of right-hand side for Holling type III exploitation (3.33), on the left for $K = 33.3$, i.e., $\phi = 0.3$, and for integer harvesting rates $h \in \{1, \ldots, 10\}$. On the right is a blowup of the same picture around the origin for $h \in \{0.5, 1, 1.5, 2, 2.5\}$. The lowmost harvesting curve intersects the reproduction curve at two feasible points, and the other ones at four such points.

For a Holling type II harvesting, the unscaled Allee model is

$$\frac{dU(T)}{dT} = rU(T)(U(T) - B) \left(1 - \frac{U(T)}{K}\right) - \tilde{h} \frac{U(T)}{A + U(T)}, \quad A, \tilde{h} > 0.$$  \hspace{1cm} (3.38)

Rescaling is performed choosing $\beta = A$, $\alpha = (Ar)^{-1}$, and $h = \tilde{h}A^{-2}r^{-1}$, $b = \frac{b}{A}$, $\phi = \frac{A}{K}$, to find

$$\frac{du(t)}{dt} = u(u - b)(1 - \phi u) - h \frac{u}{1 + u}.$$  \hspace{1cm} (3.39)

The equilibria in addition to the origin, stable, are two points moving toward each other for increasing $h$ from $b$ and $\phi^{-1}$, the former unstable and the latter stable. Then they coalesce and disappear, leaving only the origin as stable equilibrium. Figure 3.18 shows the bifurcation diagram.

The Allee model with Holling type III harvesting is

$$\frac{dU(T)}{dT} = rU(T)(U(T) - B) \left(1 - \frac{U(T)}{K}\right) - \tilde{h} \frac{U^2(T)}{A + U^2(T)}.$$  \hspace{1cm} (3.40)
FIGURE 3.14: Bifurcation diagram for the logistic model (3.33) with Holling type III harvesting: case of only two feasible intersections between the reproduction and harvesting curves; stable branch $\ast$, unstable branch $\circ$; $K = 1.5$, i.e., $\phi = 0.75$.

FIGURE 3.15: Bifurcation diagram for the logistic model (3.33) with Holling type III harvesting: case of four feasible intersections between reproduction and harvesting curves; stable branch $\ast$, unstable branch $\circ$; $K = 33.3$, i.e., $\phi = 0.3$. 
FIGURE 3.16: Bifurcation diagram for the Allee model (3.35) with constant harvesting: stable branch *, unstable branch o.

FIGURE 3.17: Bifurcation diagram for the Allee model (3.36) with linear harvesting: stable branch *, unstable branch o.
FIGURE 3.18: Bifurcation diagram for the Allee model (3.39) with Holling type II harvesting: stable branch $\ast$, unstable branch $\circ$.

(with $A, \bar{h} > 0$), and rescaling via $\beta = \sqrt{A}$, $\alpha = (\sqrt{Ar})^{-1}$, and $h = \alpha \bar{h} (\sqrt{A})^{-1}$, $b = \frac{B}{\sqrt{A}}$, $\varphi = \frac{\sqrt{A}}{K}$, leads to

$$\frac{du(t)}{dt} = u(u - b)(1 - \varphi u) - h \frac{u^2}{1 + u^2}. \tag{3.41}$$

The equilibria are again the origin and the two points moving toward each other from $b$ and $\varphi^{-1}$, retaining their stability, until they coalesce and vanish. The bifurcation diagram is shown in Figure 3.19.

On comparing Figures 3.16–3.19, we notice that they are only quantitatively different, but their qualitative behavior is exactly the same. This agrees with previous remarks on the intersections of the reproduction and harvesting functions.

There is a further remark we ought to make. On comparing Figures 3.16 to 3.19 with Figure 3.15, where we have seen the hysteresis phenomenon occur, again qualitatively we see that the behavior for the Allee model is similar, but for one important feature. Namely, this model entails the sure disappearance of the population once the critical harvesting effort $h_C$ is reached. In this case, indeed, the lower branch of the bifurcation diagram lies exactly on the horizontal axis so that the population vanishes and thus cannot recover even by drastically reducing the exploitation down to $h = 0$. The only difference, as remarked above for the four cases of harvesting in the Allee situation, is the increasing value of $h_C$, namely, $h_C = 0.08$ for constant effort, $h_C = 0.12$ for Holling type I effort, $h_C = 0.2$ for Holling type II effort, and $h_C = 0.28$ for Holling type III effort.
3.3 Glance at discrete models

We finally consider here models in which the population size is measured at fixed time intervals, in general corresponding to generation times. Let \( T_n \) be the instants in time corresponding to the appearance of the new generation; it could be eggs disclosure for some insects, for instance, which may happen a few times during a good season. Therefore, the new generation of size \( X_n \equiv X(T_n) \) may appear a number of times a year, depending on the species under consideration and the climactic factors. The quantity \( X_{n+1} - X_n \) measures the change in the population size during one such typical generation, and in absence of migrations, it is the balance of births and deaths, namely, \( X_{n+1} - X_n = bX_n - dX_n = rX_n \), where \( b \) and \( d \) denote the per capita birth and death rates per generation time. What we have described is a version of the discrete Malthus model,

\[
X_{n+1} = aX_n, \quad a = 1 + r = 1 + b - d.
\] (3.42)

Attempting the solution of this difference equation by means of a discrete exponential function \( X_n = \alpha^n \), we obtain the characteristic equation \( \alpha = a \), from which the solution \( X_n = X_0\alpha^n \) follows. This model exhibits the same drawbacks as its continuous counterpart, namely, for \( a < 1 \) we find \( X_n \to 0^+ \), and for \( a > 1 \) we have \( X_n \to +\infty \). But a new problem arises for \( -1 < a < 0 \), since now the solution oscillates taking negative values,
which is clearly a nonbiological situation. A natural correction would require $X_{n+1} = \max\{aX_n, 0\}$.

An improvement would be to consider logistic-type models, of which the following are examples:

\[ X_{n+1} = \frac{aX_n}{X_n + A}, \quad (3.43) \]
\[ X_{n+1} = \frac{aX_n^2}{X_n^2 + A}, \quad (3.44) \]
\[ X_{n+1} = X_n + aX_n \left(1 - \frac{X_n}{K}\right), \quad (3.45) \]
\[ X_{n+1} = a \left(1 - \frac{X_n}{K}\right), \quad (3.46) \]
\[ X_{n+1} = e^{a\left(1 - \frac{X_n}{K}\right)}, \quad (3.47) \]
\[ X_{n+1} = aX_n \left(1 - \frac{X_n}{K}\right)^{-\beta}. \quad (3.48) \]

In general, these models are of the form $X_{n+1} = f(X_n)$, and if $X^*$ exists such that $X^* = f(X^*)$, so that it is a fixed point for the map $f$, then it is an equilibrium of the discrete system. We now consider a perturbation, $u_n = X_n - X^*$, so that for a suitable $\bar{X} \in CH\{X^*, X_n\}$, with the symbol $CH$ denoting the convex hull of the points $X^*, X_n$, i.e., the smallest interval that contains them, Taylor’s formula gives

\[ X_{n+1} - X^* = f(X_n) - f(X^*) = \frac{df}{dX}(X^*)u_n + \frac{1}{2} \frac{d^2f}{dX^2}(\bar{X})u_n^2. \quad (3.49) \]

Since upon linearization we have $u_{n+1} = \rho u_n = \rho^2 u_{n-1} = \ldots = \rho^{n+1} u_0$, convergence occurs if $|\rho| < 1$. \hfill (3.50)

Finally, let us mention a very famous model, due to Fibonacci, that gives rise to a “second order” difference equation, namely, it involves the two previous generations to determine the next one, $x_{n+1} = x_n + x_{n-1}$, with initial conditions $x_0 = 0, x_1 = 1$. Its solution involves the golden ratio, which is related to the length of the side of the regular decagon, the polygon with 10 sides. Indeed, upon trying an exponential solution, we find the characteristic equation

\[ \alpha^2 - \alpha - 1 = 0, \quad (3.51) \]

whose solutions are $\alpha_{\pm} = \frac{1}{2}[1 \pm \sqrt{5}]$. Now the solution of the Fibonacci model can be written as a linear combination of exponentials with these bases:

\[ x_n = c_+ \alpha_+^n + c_- \alpha_-^n = c_+ \left\{\frac{1}{2}[1 + \sqrt{5}]\right\}^n + c_- \left\{\frac{1}{2}[1 - \sqrt{5}]\right\}^n. \]
so that as \( n \to \infty \), since \(|\alpha_-| < 1\), we have
\[
\frac{x_{n+1}}{x_n} \to \frac{1}{2}[1 + \sqrt{5}] = \alpha_+ \equiv \phi = 1.61803399.
\]

In classical euclidean geometry, the golden ratio \( \phi \) is determined by the point \( \alpha \) that splits the (unit) segment in two parts, so as to satisfy the proportion \( 1 : \alpha = \alpha : (1-\alpha) = \phi \). On expanding, \( \phi = \frac{\alpha}{1-\alpha} = \frac{1}{\alpha} \) and \( \phi \) is the only positive solution of the resulting quadratic equation, i.e., (3.51), so that \( \phi = \alpha_+ \).

### 3.4 Peek into chaos

Let us specifically investigate Equation (3.45). We find
\[
f(X) = (1 + a)X - \frac{a}{K}X^2, \quad \frac{df}{dX} = 1 + a - \frac{2a}{K}X.
\]
Calculating the fixed points of \( f \), we find the equilibria of the equation, \( X_* = 0 \) and \( X^* = K \). To study the stability, we are led to the characteristic equation and the consequent conditions \( \rho = \frac{df}{dX}(X_*) = \frac{df}{dX}(0) = |1 + a| < 1 \), which implies the stability condition \( 0 > a > -2 \), so that for \( a > 0 \), the origin is unstable, and \( \rho = \frac{df}{dX}(X^*) = \frac{df}{dX}(K) = |1 - a| < 1 \), which gives instead \( 0 < a < 2 \) for stability. Now, to see what happens for \( a > 2 \), we can consider the iterated map \( X_{n+2} = f_2(X_n) \),
\[
f_2(X) = f(f(X)) = (1 + a)f(X) - \frac{a}{K}f(X)^2
\]
\[
= (1 + a)^2X - X^2(a + 1)\frac{a(a + 2)}{K} + 2a^2\frac{a}{K^2}(1 + a)X^3 - \frac{a^3}{K^3}X^4.
\]
To find the equilibria \( f_2(X) = X \), we must then solve the following quartic equation:
\[
aX(1 - \frac{X}{K}) \left[ \left(\frac{a}{K}\right)^2X^2 - a(a + 2)\frac{X}{K} + a + 2 \right] = 0,
\]
which has the roots \( X = 0, X = K, \) and
\[
X_\pm = \frac{K^2}{2a^2} \left\{ \frac{a}{K}(a + 2) \pm \left[ \frac{a^2}{K^2}(a + 2)^2 - \frac{4}{K^2}(a + 2)a^2 \right]^{\frac{1}{2}} \right\}
\]
\[
= \frac{K}{2a} \left[a + 2 \pm \sqrt{a^2 - 4} \right]. \quad (3.52)
\]
The latter are real for \( a \geq 2 \). Moreover, observe that
\[
f(X_+) = X_+ + aX_+ \left( 1 - \frac{X_+}{K} \right) = (1 + a)X_+ - \frac{a}{K}X_+^2
\]
\[
= (1 + a)\frac{a^2 + 2}{2a}K + \frac{1 + a}{2a}K\sqrt{a^2 - 4}
- \frac{a}{4a^2} \left[ (a + 2)^2 + a^2 - 4 + 2(a + 2)\sqrt{a^2 - 4} \right]
\]
\[
= \sqrt{a^2 - 4} \left[ \frac{1 + a}{2a}K - \frac{K}{2a} (a + 2) \right] + \frac{K}{2a} \left[ (1 + a)(2 + a) - (a^2 + 2a) \right]
\]
\[
= \frac{K}{2a} \left[ (2 + a)(1 + a) - \sqrt{a^2 - 4} \right] = \frac{K}{2a} \left[ 2 + a - \sqrt{a^2 - 4} \right] = X_-
\]

Since we have a fixed point for the iterated map, it follows that
\[ X_+ = f_2(X_+) = f(f(X_+)) = f(X_-), \quad \text{i.e.} \quad X_- f(X_+), \quad X_+ = f(X_-) \]

We have thus found a periodic solution for \( f(X) \), of period 2, for \( a > 2 \). We investigate now the stability of this periodic solution. Recalling the convergence condition (3.50) for fixed point iterates of the map \( f_2(X) \), let us differentiate \( f_2(X) - X = 0 \). Since \( X_+ \) solves the quadratic equation (3.52), we find
\[
\frac{df_2}{dX}(X_+) - 1 = a \left( X_+ - \frac{X_+^2}{K} \right) \left[ \frac{2a^2}{K^2}X_+ - \frac{a}{K}(a + 2) \right]
\]
\[
= \frac{K}{2} \left( a + 2 + \sqrt{a^2 - 4} \right) \frac{1}{2a} \left( a - 2 - \sqrt{a^2 - 4} \right)
- \frac{a}{K} \left( a + 2 + \sqrt{a^2 - 4} - a - 2 \right)
\]
\[
= \frac{1}{4} \left[ a^2 - (2 + \sqrt{a^2 - 4})^2 \right] \sqrt{a^2 - 4}
\]
\[
= \frac{1}{4} \left[ a^2 - 4\sqrt{a^2 - 4} - (a^2 - 4) \right] \sqrt{a^2 - 4} = 4 - a^2.
\]
Hence \( \frac{df_2}{dX}(X_+) = 5 - a^2 \) and the condition for stability then becomes \( |5 - a^2| < 1 \), so that the cycle of period 2 for the map \( f \) is stable for the parameter range \( 2 < a < \sqrt{6} \).

The question now arises as to what happens for \( a > \sqrt{6} \). Again a periodic solution of period 4 can be found that is stable for \( \sqrt{6} < a < 2.544 \). Iterating the question and the procedure, a periodic solution of period 8 arises hereafter, which is stable for \( 2.544 < a < 2.564 \). On continuing this procedure, we obtain the so-called period doubling route to chaos. It can be shown that if \( a_n \) is the parameter value for which a periodic solution of period \( 2^n \) appears, then
\[
\lim_{n \to \infty} \frac{a_{n+1} - a_n}{a_{n+2} - a_{n+1}} = 4.6692016... ,
\]
this limiting value being known as the Feigenbaum constant.
Chapter 4

Interacting populations

The Lotka–Volterra model takes its name from the authors of the first investigations of interacting populations. Volterra (1926a; 1926b; 1931) based his considerations on the unexpected results of the field observations of the biologist D’Ancona (1954) on the fishing catches in the Adriatic sea after the First World War. Other models were developed later to avoid the main drawback of the original Lotka–Volterra model, namely, the neutral stability of its interior equilibrium point. This makes the solutions sensitive to variations of the initial conditions of the system, and therefore biologically they are not very satisfactory. These modifications include at first quadratic models, which incorporate a logistic growth term for at least one of the two populations, and then also other nonlinear terms, such as the effect of satiation in feeding.

Competing situations and species living in symbiosis are other examples of interacting populations, extended then to more complicated food webs in which the species at a certain level in the chain is the predator of those in the levels below and a prey for the higher level ones.

To model the quadratic interactions between different populations, we proceed similarly to what is done for the logistic term in a single population growth. If one individual of the first population \( U \) wanders in the environment, the meetings with individuals of the second population \( V \) will occur at a certain per capita rate \( \alpha \). Thus the total encounters of that specific individual per unit time are \( \alpha V \). But this must be multiplied by every individual of species \( V \), so that the total number of interactions in the time unit between the two populations is then \( \alpha UV \). More generally, the interactions for the population \( V \) are described by a per capita rate \( f(U,V)V \). The quadratic case then corresponds to a Holling type I term. If we want to model the saturation effect experienced by predators when the prey abound, we should then take for \( f \) a Holling type II or III function. The latter correspond to what in operations research is called the law of diminishing returns, for the same amount of effort the gain is larger if the effort is small, and tends to a plateau for increasing efforts.

In this chapter we consider basically the Lotka–Volterra model and its modifications. In addition to presenting the models, we also develop the main mathematical techniques needed for the analysis of these systems, by discussing very simple situations in detail. We start with an example in Section 4.1 that mathematically illustrates the local stability analysis and the tools
needed for it. In Section 4.2 a more in-depth discussion of the Lotka–Volterra model is related and also used for a more in-depth discussion of the scaling procedure, also in view of the later parts of the book. Section 4.2.1 presents another modification of the prey–predator system and the other interesting biological situations of competing and symbiotic species. Quadratic and other classical nonlinear models are discussed. The main mathematical tool for studying the global stability of these models is the Lyapunov function, presented in Section 4.3.1. Then the question of the investigation of possible limit cycles is addressed, with Section 4.3.2 giving positive and negative criteria.

The last two sections are devoted to a step further, beyond the two-species models. In Section 4.3.3, a four-level food web is considered, from minerals through plants and herbivores up to the top predators, the carnivores. Section 4.4 describes a very important problem in ecology, namely, the preservation of cultures, in this case vineyards, by using ecological controls.

From now on all parameters appearing in the models are assumed to be nonnegative constants, unless otherwise specified.

4.1 Two-species prey–predator population model

We consider an idealized ecosystem in which only two populations are accounted for, and one may be hunted by the second one. More specifically, let the prey and the predator populations be denoted by \( U(T) \) and \( V(T) \), respectively. We assume that the environment provides enough resources for the former to ensure its Malthusian growth. The predators instead show logistic growth, where \( r \) denotes their reproduction rate and \( K \) represents the environment carrying capacity. These assumptions imply that the predators have sources of food other than the prey \( U \). In the absence of interactions, the two populations would grow independently as follows:

\[
\frac{dU}{dT} = \alpha U(T), \quad \frac{dV}{dT} = rV(T) \left[ 1 - \frac{V(T)}{K} \right].
\]

Assume now that the predation occurs at rate \( b \), while the conversion rate into new predators is described by the parameter \( c \). Combining the above equations, we thus obtain

\[
\frac{dU}{dT} = U(\alpha - bV), \quad \frac{dV}{dT} = rV \left( 1 - \frac{V}{K} \right) + cUV. \tag{4.1}
\]

To analyze this system, we first find the nullclines, i.e., the lines in the phase plane on which each population does not change. Their intersections also give the equilibria of the system, i.e., the points at which both populations do not change. The two nullclines are respectively defined by the equations \( \frac{dU}{dT} = 0 \),
$dV/dT = 0$, and these in turn give for the equilibria the system of algebraic equations

$$U(\alpha - bV) = 0, \quad V \left[ r \left( 1 - \frac{V}{K} \right) + cU \right] = 0. \quad (4.2)$$

Now, from the first one, we get either $U(T) \equiv U_0 \equiv 0$ or $V(T) \equiv V_2 = \frac{\alpha}{b}$. In the former case, from the second one, we have either $V(T) \equiv V_0 = 0$ or $V(T) \equiv V_1 = K$. The second choice on the first equation gives, upon substitution into the second one, $U(T) \equiv U_2 = \frac{r}{c} (\frac{\alpha b}{K} - 1)$. Thus we have the three equilibria

$$E_0 \equiv (U_0, V_0) \equiv (0, 0), \quad E_1 \equiv (U_0, V_1) \equiv (0, V_1), \quad E_2 \equiv (U_2, V_2).$$

Clearly $E_0$ and $E_1$ are always feasible, while to have a meaningful solution for $E_2$ we need the populations to be nonnegative, and this entails the restriction $K < \frac{\alpha}{b}$, which is then the feasibility condition for $E_2$.

**Local Stability Analysis**

We now study the long-term behavior of the model near the equilibria. To do so, we need to linearize it. The procedure consists in defining new variables in the neighborhood of each equilibrium point and then disregarding perturbations of higher order, so that the system becomes easily integrable. Of course the solutions we find are valid only as a first approximation near the equilibrium, but nevertheless they tell a lot about the model’s behavior.

Consider $E_0$ at first. We investigate what happens to the system in a neighborhood of the origin. As $U, V$ in this case are small, namely $U, V < 1$, then $U^2, V^2, UV < U, V$ so that $U^2, V^2, UV \ll 1$. These second order perturbations are much smaller than the original ones and can be disregarded as mentioned earlier. The system then simplifies to

$$\frac{dU}{dT} = aU(T), \quad \frac{dV}{dT} = rV(T). \quad (4.3)$$

This is easy to solve, giving $U(T) = U(0) \exp(aT)$, $V(T) = V(0) \exp(rT)$. In fact, it is so easy because it is uncoupled. Since $\alpha, r > 0$, the solutions will grow, thus the trajectories will wander away from the origin, even though we chose $U(0) = U_0 \approx 0$, $V(0) = V_0 \approx 0$.

Look now at $E_1$. In this case, $U$ is still small, but $V$ lies near the value $V_1$. It is therefore better to introduce the perturbation $y = V - V_1$, for which $\frac{dy}{dT} \equiv \frac{dV}{dT}$. Again we have $y^2, yU < y$, so that $y^2, yU \ll 1$ and the latter can be disregarded. The first equation of the linearized system in a such case reads

$$\frac{dU}{dT} = U(T)[\alpha - b(y + V_1)] = U(T)[\alpha - bV_1] = U(T)[\alpha - bK],$$
while the second one is
\[
\frac{dy}{dT} = r(y + V_1) \left[ 1 - \frac{(y + V_1)}{K} \right] + cU(T)(y + V_1)
\]
\[
= r(y + K) \left[ 1 - \frac{(y + K)}{K} \right] + cU(T)(y + K)
\]
\[
= -r(y + K) \frac{y}{K} + cKU(T) = -ry + cKU(T).
\]

In this case the system is not so easy to solve. But substitution is still a viable method. Solve the first equation, which is still independent of \(y\), to get
\[
U(T) = U(0) \exp\left[ (\alpha - bK)T \right].
\]

As no powers higher than one, nor product of unknown functions appear in it, this is a linear constant coefficient, first order nonhomogeneous ordinary differential equation. Notice that each expression has its own meaning, namely, the coefficients are indeed constants, not functions of time; the highest derivative is the first one; there is a term independent of the unknown \(y\) making the equation nonhomogeneous, i.e., carrying it to the right-hand side; the latter is nonzero, i.e., nonhomogeneous; and, finally, no partial derivatives arise in the formulation. This classification may be useful for understanding how to solve it, if one wishes to look for the method in books. Instead, we will proceed in a constructive way. If we forget about the nonhomogeneous part, the solution is easily found to be
\[
y(T) = k \exp(-rT),
\]
\(k\) denoting an arbitrary integration constant. This is just the solution of the homogeneous equation. To find the one for our problem, we use the method of variation of parameters, which amounts to letting \(k\) become a function of time, \(k \equiv k(T)\). Differentiation and substitution into the original nonhomogeneous equation give for the left-hand side
\[
\frac{dy}{dT} = \frac{d[k(T) \exp(-rT)]}{dT} = \frac{dk}{dT} \exp(-rT) + k(T) \frac{d[\exp(-rT)]}{dT}
\]
\[
= \frac{dk}{dT} \exp(-rT) - rk(T) \exp(-rT),
\]
while for the right-hand side we have
\[
-ry(T) + cKU(T) = -rk(T) \exp(-rT) + cKU(0) \exp[(\alpha - bK)T].
\]

Observe that two terms on the two sides of the equation are equal. Thus, equating and simplifying, we find
\[
\frac{dk}{dT} = cK \exp(rT)U(0) \exp[(\alpha - bK)T] = cKU(0) \exp[(\alpha + r - bK)T].
\]
Solving this last differential equation for $k$, we have

$$k(T) = C + \frac{cKU(0)}{\alpha + r - bK} \exp[(\alpha + r - bK)T],$$

where $C$ denotes an arbitrary constant of integration. Substituting back into (4.5), we have

$$y(T) = \left[ C + \frac{cKU(0)}{\alpha + r - bK} \exp[(\alpha + r - bK)T] \right] \exp(-rT)$$

$$= C \exp(-rT) + \frac{cKU(0)}{\alpha + r - bK} \exp[\alpha - bK]T.\]$$

This function solves the original nonhomogeneous equation, a fact that can be verified by backsubstitution into (4.4).

We have thus shown that $y$ tends to zero exponentially if $\alpha - bK < 0$, while it tends to infinity exponentially if $K < \alpha b$. Hence $E_1$ is stable in the former case, as $U$ tends always to zero, while it is unstable in the latter. The trajectories in the latter case diverge along the $y$-axis, i.e., the $V$-axis. Note that the condition for stability of $E_1$ is the very same condition for the infeasibility of $E_2$. Thus when the latter is infeasible, then trajectories approach $E_1$, while if $E_1$ is unstable, then the trajectories most likely will approach $E_2$, as the latter would now be feasible, and stable, as we will show shortly.

We now turn to the more complicated analysis for the case of $E_2$. Linearization entails the definition of two new variables, the perturbations in each component $x = U - U_2$ and $y = V - V_2$. We then obtain

$$\frac{dx}{dT} = (x + U_2)[\alpha - b(y + V_2)] = -by(x + U_2) = -bU_2y,$$

$$\frac{dy}{dT} = \{r[1 - \frac{(y + V_2)}{K}] + c(x + U_2)\}(y + V_2).$$

The second one can be further processed as follows:

$$\frac{dy}{dT} = \left\{ r \left[ 1 - \frac{(y + \frac{\gamma}{K})}{K} \right] + c \left[ x + \frac{r}{\alpha} \left( \frac{\alpha}{bK} - 1 \right) \right] \right\} \left( y + \frac{\gamma}{b} \right)$$

$$= -r \frac{y}{K} \left( y + \frac{\alpha}{b} \right) + r \left( y + \frac{\alpha}{b} \right) - \frac{\alpha r}{bK} \left( y + \frac{\alpha}{b} \right)$$

$$+ cx \left( y + \frac{\alpha}{b} \right) + r \left( \frac{\alpha}{bK} - 1 \right) \left( y + \frac{\alpha}{b} \right)$$

$$= -r \frac{y}{K} \left( y + \frac{\alpha}{b} \right) - \frac{\alpha r}{bK} \left( y + \frac{\alpha}{b} \right) + cx \left( y + \frac{\alpha}{b} \right) + \frac{\alpha r}{bK} \left( y + \frac{\alpha}{b} \right)$$

Finally, we have to analyze the linearized system

$$\frac{dx}{dT} = -bU_2y , \quad \frac{dy}{dT} = -\frac{\alpha r}{bK}y + \frac{\alpha c}{b}x .$$

(4.6)
Let us introduce the perturbation vector \( u = (x, y)^T \). We can then write the above system in the form \( \frac{du}{dt} = Mu \), where the matrix \( M \) will now be determined. To do this in a very elementary but constructive way, let us observe that for a generic matrix \( A \) with elements

\[
A = \begin{bmatrix}
A_{1,1} & A_{1,2} \\
A_{2,1} & A_{2,2}
\end{bmatrix},
\]

upon postmultiplication by \( u = (u_1, u_2)^T \), the row by column product gives the vector

\[
(A_{1,1}u_1 + A_{1,2}u_2, A_{2,1}u_1 + A_{2,2}u_2)^T.
\]

If we denote by \( A_{.,j} \) the \( j \)th column of the matrix \( A \), so that \( A = [A_{.,1}, A_{.,2}] \), then the above equations can be rewritten as \( Au = A_{.,1}u_1 + A_{.,2}u_2 \). It is then apparent that the \( j \)th column of the matrix \( A \) acts on the \( j \)th component of \( u \). To construct the matrix \( M \), we have to look at its action on the vector \( u \), action coming from the equations of the system. From the first one, the component \( u_1 \equiv x \) is seen to be absent, while \( u_2 \equiv y \) gets multiplied by \( -bU_2 \). The only number that makes \( u_1 \) disappear is zero. Similarly, in the second equation, \( x \) is multiplied by \( \frac{\alpha c\beta}{\tau} \). Hence the first column of \( M \) must be given by \( [0, \frac{\alpha c\beta}{\tau}]^T \). In the second equation, moreover, \( y \) has the coefficient \( -\frac{\alpha r\beta K}{\tau} \), so that the second column of \( M \) must be given by \( [-bU_2, -\frac{\alpha r\beta K}{\tau}]^T \). In summary,

\[
A = \begin{bmatrix}
0 & -bU_2 \\
\frac{\alpha c\beta}{\tau} & -\frac{\alpha r\beta K}{\tau}
\end{bmatrix}.
\]

If we consider carefully the matrix just obtained, we find that it coincides with the Jacobian of the original system (4.1) evaluated at \( E_2 \). This is a general result. Any time we linearize around an equilibrium, we may skip all the steps undertaken above and just consider directly the Jacobian of the model. This will be done repeatedly in the following chapters.

To get an uncoupled system, let us introduce the change of basis matrix \( W \), yet unknown, and a new vector of unknowns \( z \), such that \( u = Wz \), for which the system is uncoupled. We must then have

\[
W \frac{dz}{dt} = MWz, \quad \frac{dz}{dt} = W^{-1}MWz = \Lambda z,
\]

with \( \Lambda = diag(\lambda_1, \lambda_2) \). Here the entries of this diagonal matrix as well as those of \( W \) are not known. To find these unknowns, let us once again identify the matrix \( W \) by its columns, \( W = [W_{.,1}, W_{.,2}] \). For an arbitrary vector \( z \), it then follows

\[
MWz = W\Lambda z,
\]

that is,

\[
M[W_{.,1} W_{.,2}] = [W_{.,1} W_{.,2}]\Lambda \equiv [W_{.,1} W_{.,2}][L_{.,1} L_{.,2}],
\]

where \( L_{.,1} \) and \( L_{.,2} \) are the \( 2 \times 2 \) identity matrices.
where \( L_{.,j} \) denotes the \( j \)th column of \( \Lambda \) and therefore is the \( \lambda_j \) multiple of the \( j \)th vector in the standard basis of the euclidean space, in view of the action of a matrix over a vector described above,

\[
L_{.,j} = (0, \ldots, 0, \lambda_j, 0, \ldots, 0)^T = \lambda_j (0, \ldots, 0, 1, 0, \ldots, 0)^T = \lambda_j e_j.
\]

Indeed, once again, the action of the \( j \)th column of the matrix \( W \) is on the \( j \)th component of \( L_{.,j} \) of the vector \( L_{.,j} \), since the latter is the \( \lambda_j \) multiple of the \( j \)th vector of the standard euclidean basis \( e_j \). Hence the \( j \)th column gets multiplied by \( \lambda_j \), i.e., \( WA \equiv [\lambda_1 W_{.,1} \, \lambda_2 W_{.,2}] \), so that, equating columns, we find \( MW_{.,1} = \lambda_1 W_{.,1} \) and \( MW_{.,2} = \lambda_2 W_{.,2} \).

More in general, for \( j = 1, 2 \), we have to solve the problem

\[
MW_{.,j} = \lambda_j W_{.,j}. \tag{4.7}
\]

This is is the well-known linear algebra eigenvalue problem. Geometrically, a direction \( z \) is sought, which is invariant under the action of the matrix \( \Lambda \), i.e., the resulting vector \( \Lambda z \) through the eigenvalue \( \lambda \) can change length but not the direction. Note that a zero eigenvalue is allowed. Indeed, the resulting null vector is assumed to possess every direction in space. This follows from the fact that it must be orthogonal to any arbitrary vector \( w^* \) in space, since \( 0^T w^* = 0 \). However, a null eigenvector makes no sense, as it trivially satisfies the eigenvalue equation \( M0 = \lambda0 \) but gives no indication as to which direction is being left invariant by the matrix. To solve the eigenvalue problem, rewrite the equation as \( (M - \lambda I)w = 0 \), having set \( W_{.,j} = w \). This is a homogeneous system, for which we seek a nonzero solution, in view of the above discussion on eigenvectors. We need further tools from linear algebra.

The main result that we are now going to quote is the summary of several equivalent statements, the proofs of which are easily found in elementary linear algebra texts.

\textbf{Fundamental Theorem of Linear Algebra}

For the system \( Ax = b \), the following statements are all equivalent to each other:

- The matrix \( A \) is nonsingular, i.e., it has an inverse \( A^{-1} \).
- The nonhomogeneous system, i.e., the system with \( b \neq 0 \), is uniquely solvable.
- \( \det A \neq 0 \).
- The homogeneous system, i.e., the system with \( b = 0 \), which has clearly always the trivial solution \( x = 0 \), also possesses infinitely many nontrivial solutions.
- The column rank of the matrix is maximal.
The row rank of the matrix is maximal.

The eigenvalues of the matrix are nonzero.

The right-hand side \( \mathbf{b} \) belongs to the subspace spanned by the columns of the matrix \( \mathbf{A} \).

All the columns of the matrix are linearly independent.

All the rows of the matrix are linearly independent.

Thus, since our system is homogeneous and since we seek the eigenvectors, i.e., its nontrivial solutions, we must impose that the matrix \( \mathbf{M} - \lambda \mathbf{I} \) be singular. Using the statements above, we impose that its determinant must vanish, \( \det(\mathbf{M} - \lambda \mathbf{I}) = 0 \). This gives only an algebraic equation, namely, the characteristic equation \( (M_{1,1} - \lambda)(M_{2,2} - \lambda) - M_{1,2}M_{2,1} = 0 \), making the problem easier to tackle. Substituting the values of our situation, we find \( \lambda^2 + \frac{\alpha r}{bhK} \lambda + \alpha c U_2 = 0 \). This algebraic equation has two roots,

\[
\lambda_1 = -\frac{\alpha r}{2bhK} + \Delta, \quad \lambda_2 = -\frac{\alpha r}{2bhK} - \Delta, \quad \Delta \equiv \left( \frac{\alpha r}{2bhK} \right)^2 - \alpha c U_2.
\]

Observe that the second term in the discriminant \( \alpha c U_2 \) is always positive, so that we are really subtracting it from the square. Thus the square root of \( \Delta \) is smaller than \( \frac{\alpha r}{bhK} \), and this entails that \( \lambda_1 \) cannot be positive. Certainly \( \lambda_2 < 0 \), so that both roots, which are either real or complex conjugate, depending on the discriminant, always have a negative real part, \( \Re(\lambda_i) < 0 \). They are real if

\[
\alpha r \left( \frac{1}{2bhK} \right)^2 - \left( \frac{\alpha}{bhK} - 1 \right) > 0.
\]

In both cases, however, the solutions will be either decaying exponentials, for real eigenvalues, or complex conjugate exponentials. In the former case, the solutions apart from arbitrary constants are \( z_1 = \exp(-\lambda_1 T), \ z_2 = \exp(-\lambda_2 T) \). In such a case, the equilibrium is stable, as the perturbations approach zero. Indeed, if this is the case for \( \mathbf{z} \), so it is for the nonsingularly transformed vector \( \mathbf{u} = \mathbf{W} \mathbf{z} \). In the case of complex conjugate roots, Euler’s formula allows the computation of the result, \( \exp(i\theta) = \cos(\theta) + i\sin(\theta) \). Thus, for \( j = 1, 2, \) if \( \lambda_j = \mu_j + i\nu_j \), we have \( \mathbf{z}_1 = \exp(\lambda_j T) = \exp[(\mu_j + i\nu_j)T] \equiv \exp(\mu_j)[\cos(\nu_j T) + i\sin(\nu_j T)] \). The trajectories in a neighborhood of \( E_2 \) in this case approach the equilibrium following spirals, as indicated by the trigonometric terms. The trajectories are indeed spirals as they are “almost” periodic, in the sense that after a time of \( 2\pi \) from a given instant \( T^* \) they are in the same position with respect to the \( x \)-axis, making with this axis the very same angle as they were at time \( T^* \). Hence they have gone around \( E_2 \) once. But their distance from the equilibrium at time \( T^* \) was \( \exp(\mu_j T^*) \) and at time \( T^* + 2\pi \) it is \( \exp[\mu_j(T^* + 2\pi)] \), i.e., it has shrunk. Recall that \( \mu_j < 0 \) in view of the above analysis. This remark will be the heart of the method of Section 4.4.6.
Summarizing discussion

Implications of any model predictions should be interpreted in terms of the science where it stems from. Let us therefore consider what we have learnt from the above analysis. Suppose that \( U \) represents some kind of nuisance species that we would like to fight with biological means, i.e., using one of its natural predators \( V \). The problem may then be that in the environment where \( U \) is present, other species appetible to \( V \) may also be present. In such a case, \( V \) has a choice on which species to feed upon. The interior equilibrium we have found expresses the fact that if the carrying capacity \( K \) for the predators \( V \) is not high enough, they survive by feeding partly on the other populations present and partly on \( U \), but the latter survives in the environment and settles to the value \( U_2 \). On the other hand, if \( K \) is raised high enough, the equilibrium moves on the \( V \)-axis, i.e., the predator will bring the pest \( U \) to extinction. Thus, in order to eliminate the latter, if we cannot act directly on the number of their natural predators, we could at least provide them with enough alternative food sources in order to raise their environment carrying capacity \( K \) so that they are then able to wipe out the pest \( U \).

4.2 Classical Lotka–Volterra model

In contrast to the model presented earlier, the classical Lotka–Volterra considers an aquatic environment, in which the sharks have as their food source the other fish, which all together constitute the prey. In their absence, the predators exhibit a negative growth rate, \(-e\). This assumption is indeed the main difference with the model of Section 4.1. In the classical model, the equations read

\[
\frac{dU}{dT} = U(T)\left[\alpha - bV(T)\right], \quad \frac{dV}{dT} = V(T)\left[-e + cU(T)\right].
\] (4.8)

If one carries out the linearization procedure outlined before, about the equilibrium \((\xi, \eta)\) in the \(UV\) phase space, we would find in this case purely imaginary eigenvalues \( \lambda = \pm i\sigma, \sigma = \sqrt{\alpha e} \), since the Jacobian and characteristic equations are now

\[
M = \begin{bmatrix}
0 & -\frac{1}{2}eb \\
\frac{1}{2}\epsilon\alpha & 0
\end{bmatrix} \quad \text{and} \quad \lambda^2 + \epsilon\alpha = 0,
\]

respectively.

The basis for the linearized solution is then given by \( \exp(\pm \sigma T) = \cos(\sigma T) \pm i\sin(\sigma T) \). Near the equilibrium point the solution trajectories are thus closed curves, giving the so-called neutral stability. If two different initial conditions are considered, the trajectories starting from each one of them will return to
the initial values after a certain time. In other words, perturbing the initial values, the trajectories do not approach the equilibrium, they rather remain confined to a suitable neighborhood of this point. From a practical point of view this is not satisfactory, as biologically an equilibrium should attract or repel trajectories and not be too sensitive to the initial conditions.

4.2.1 More on prey–predator models

Now we consider a more general model than the original Lotka–Volterra model, with the aim of overcoming the weakness it possesses, namely, the neutral stability of its equilibrium. In order to do that, we will assume that the environment for the prey is no longer unlimited, that a carrying capacity exists to limit their growth. Let the prey and predator populations be again expressed respectively by the functions $U(T)$ and $V(T)$. As for the classical Lotka–Volterra system, assume the prey are the sole food source for the predators, so that the mortality rate for the latter is exponential in absence of the former. This is then a different environment than the one discussed in Section 4.1. The system is therefore

$$\frac{dU}{dT} = U(a - bV - eU), \quad \frac{dV}{dT} = V(-c + dU). \quad (4.9)$$

The equilibria are the origin and then the points $E_1 = (a, 0)$ and $E_2 \equiv (U^*, V^*)$, with $U^* = \frac{a}{d}, V^* = \frac{1}{bd}(ad - ce)$. This nontrivial equilibrium point is feasible for $ad > ce$. The system’s Jacobian is easily evaluated,

$$J = \begin{pmatrix} a - bV - 2eU & -bU \\ dV & -c + dU \end{pmatrix}. \quad (4.10)$$

It follows that, when feasible, $E_2$ is also asymptotically stable, as the eigenvalues of the Jacobian are

$$\lambda_{\pm} = -\frac{eU^*}{2} \pm \sqrt{\left(\frac{eU^*}{2}\right)^2 - bdU^*V^*} = -\frac{ce}{2d} \pm \frac{1}{2} \sqrt{\frac{c^2e^2}{d^2} + \frac{4c}{d}(ce - ad)}$$

and thus always have a negative real part. The point $E_2$ is then a focus or a node, depending on whether the argument of the root is negative or positive. Both conditions can arise, as by fixing all parameters, except for the prey net birth rate, we see that for $a = 0$ or very small, the eigenvalues are real, thus giving a node, while for $a \to +\infty$ they become complex conjugate, and $E_2$ would thus be a focus. Worthy of notice is also the fact that should $E_2$ become infeasible, the boundary equilibrium $E_1 = (\frac{a}{d}, 0)$ would then be the new and only equilibrium. Its eigenvalues are $-a, \frac{1}{2}(ad - ce)$. These entail an important consequence, indeed that the infeasibility condition of $E_2$ implies $E_1$ to be stable, and vice versa. Thus the $\omega$-limit set of this model is given only by the points $E_2$ and $E_1$. Notice indeed that the origin is a saddle since
the trajectories escape from it along the $U$-axis. Thus the community can never become extinct, as the prey are prevented from vanishing. On the other hand, it is possible either that both species survive at levels prescribed by $E_2$, or that only the predators disappear, at the equilibrium $E_1$.

### 4.2.2 Scaling

We want to review what was already done in the case of the one-population models, to simplify the equations. In the later chapters we will appreciate this technique, since it is powerful enough to allow substantial reduction of the number of parameters appearing in a system of equations. Bearing in mind the system (4.8), let us introduce new dependent and independent variables by means of the equations

\[
U(T) = Au(t), \quad V(T) = Bv(t), \quad T = Ct,
\]

with $A, B, C$ denoting positive constants to be determined. Taking derivatives, the chain rule yields

\[
\frac{dU}{dT} = A \frac{du}{dt}, \quad \frac{dV}{dT} = B \frac{dv}{dt}.
\]

Thus back substitution into (4.8) gives

\[
\frac{du}{dt} = Cu(t)[\alpha - bv(t)], \quad \frac{dv}{dt} = Cv(t)[-e + cu(t)]. \tag{4.11}
\]

Now we want to choose the scaling constants so as to minimize the number of parameters in the rescaled system. We can thus choose to set $C\alpha = 1$ and $CbB = 1$ in the first equation in (4.11), thus giving $C = \frac{1}{\alpha}, B = \frac{\alpha}{e}$. If we now try to make use of the second equation in (4.11), setting $Ce = 1$, we see that it is not possible. Thus $\theta = \frac{e}{\alpha}$ is a rescaled parameter intrinsic to the model.

On the contrary, we can still use the left degree of freedom by setting $CcA = 1$, i.e., selecting $A = \frac{\alpha}{e}$. The rescaled model then reads

\[
\frac{du}{dt} = u(t)[1 - v(t)], \quad \frac{dv}{dt} = v(t)[-\theta + u(t)]. \tag{4.12}
\]

From the simulation point of view, let us illustrate the advantage of this procedure. Suppose we would like to investigate the model’s behavior for a range of parameter values, saving the plots of the simulations. Fixing the original parameters to some values, say $b = 0.1$, $c = 0.1$, $e = 0.1$, we may let the last parameter vary in a certain range, say $\alpha = 0.1, 0.2, \ldots, 0.9, 1.0$, thus obtaining 10 figures. Then we need to change the value of another parameter, $b = 0.2$ say, and repeat the operation, obtaining another 10 figures. Repeating again the procedure for $b = 0.3, \ldots, 0.9, 1$, we thus have 100 figures, which we can arrange in a 10 by 10 table in a single page. Then we repeat the whole construction for the value $c = 0.2$, obtaining a second “page” with another 100 figures, and then a third one for $c = 0.3$, and so on up to $c = 1.0$. In this way a “chapter” of 10 pages and 1000 figures is obtained, all corresponding
to \( e = 0.1 \). We then let \( e = 0.2 \) and again repeat the procedure, getting a second chapter, and so on, until for \( e = 1.0 \) we have completed a book of 10000 figures. Scaling instead allows us just to run the simulation for a range of values of the new parameter \( \theta \), so that all the behaviors of the model contained in the former simulations can be traced back to some of the latter, for suitable parameter combinations. Indeed, looking at the definition of \( \theta \), it is clear that the simulations for the pairs \( \alpha = 0.1, \ e = 0.2, \alpha = 0.3, \ e = 0.6 \) and \( \alpha = 0.4, \ e = 0.8 \) all correspond to the same figure in the last set obtained for \( \theta = 2 \). The advantage in terms of computational costs and easiness of reading the results is apparent.

4.3 Other types of population communities

Prey–predator interactions are among the most important in ecological communities, yet they of course are not the only ones possible. In this section we give an overview of some other types of interspecific interactions as well as some other and complementary modeling approaches.

4.3.1 Competing populations

Competing species can be either predators directly competing among themselves or species that survive in the same ecosystem exploiting the same resources, e.g., sharing a common pasture. It is known that among wild ungulates, direct competition for the same pastures arises between the red deer (\textit{Cervus elaphus hippelaphus}) and the roe deer (\textit{Capreolus capreolus}), the former suffering the most as the other wild ungulates are more generalist in the habitat selection as well as in feeding habits. The roe deer, in Mediterranean areas, also suffers from the presence of the fallow deer (\textit{Dama dama}). An example for exploitation of the same trophic niches, in the Gran Paradiso National Park in the Northwestern Alps in Italy, is given by goats and the wild chamois (\textit{Rupicapra rupicapra}) and ibex (\textit{Capra ibex}). Another instance involves skuas and seagulls populations, which compete for food, since their trophic niches overlap. Skuas (\textit{Stercorarius} spp.) predate on seagulls’ newborns, while the seagulls predate on eggs of the other species. In the National Park of the Tuscan Islands, in the Tirrenian Sea, competition for nidification occurs among seagulls (\textit{Larus audouinii} and \textit{Larus cacchinnans}). Moreover, the latter predate upon newborns and adults of the former.

To model situations of this type, let the population sizes be denoted \( P(T) \) and \( Q(T) \). The classical competition model then reads

\[
\frac{dP}{dT} = P \left[ a - bP - cQ \right], \quad \frac{dQ}{dT} = Q \left[ d - eP - fQ \right].
\] (4.13)
Each equation states the logistic reproduction of the corresponding species, and its negative interaction with the other one is expressed by the negative sign of the mixed term. The equilibria analysis for (4.13) shows that the nullcline for $P$ intersects the coordinate axes at $P \equiv P_1 = \frac{a}{b}$ and $Q = \frac{c}{e}$, and the one for $Q$ intersects them at $P = \frac{a}{d}$ and $Q = \frac{d}{f}$.

Since for very small values of both populations the dominant terms in (4.13) are the positive constants, it is immediately found that the equilibrium located at the origin $E_0 \equiv (P_0, Q_0) \equiv (0, 0)$ is unstable, while the equilibrium $E_1 \equiv \left( \frac{a}{b}, 0 \right)$ is conditionally stable, namely, if the condition $\frac{bd}{c} < \frac{ae}{c}$ holds, i.e.,

$$bd < ae. \quad (4.14)$$

For $E_2 \equiv (0, \frac{d}{f})$ stability holds for $Q < \frac{d}{f}$, i.e.,

$$af < cd. \quad (4.15)$$

The nontrivial equilibrium $E_3 \equiv \left( \frac{af - cd}{bf - ce}, \frac{bd - ac}{bf - ce} \right)$ exists if the two nullclines intersect each other, which happens if the condition $bf \neq ce$ is satisfied. But this intersection has to lie in the first quadrant, and the condition under which this nontrivial equilibrium is feasible then becomes the requirement that $bf - ce, bd - ac, \text{ and } af - cd$ all possess the same sign, i.e., either one of the following alternative statements holds:

$$\begin{align*}
\frac{b}{e} &> \frac{a}{d} > \frac{c}{f}, \quad \text{i.e.} \quad \frac{\dot{P}}{Q} > P > Q > Q \quad \text{and} \quad \text{sl}(PQ) < \text{sl}(\dot{P}Q), \quad (4.16) \\
\frac{b}{e} &< \frac{a}{d} < \frac{c}{f}, \quad \text{i.e.} \quad \frac{\dot{P}}{Q} < P < Q < Q \quad \text{and} \quad \text{sl}(PQ) < \text{sl}(\dot{P}Q), \quad (4.17)
\end{align*}$$

where “sl” stands for the slope.

The Jacobian of the system is

$$\mathbf{J} = \begin{pmatrix}
    a - 2bP - cQ & -cP \\
    -eQ & d - eP - 2fQ
\end{pmatrix}. \quad (4.18)$$

The eigenvalues at $E_3$ are then easily found,

$$\lambda_{1,2} = \frac{1}{2} \frac{a \pm \sqrt{\Delta}}{bf - ce}, \quad \alpha = -b(af - cd) - f(bd - ac), \quad \Delta = \alpha^2 - 4(af - cd)(bd - ac)(bf - ce).$$

The feasibility conditions (4.16) imply that

$$\frac{\alpha}{bf - ce} = -bP_3 - fQ_3 < 0,$$

from which it follows that at least one of the eigenvalues is necessarily negative.

From (4.16) it follows that $\Delta < \alpha^2$, so that $\lambda_2 \equiv \frac{1}{bf - ce} \left( \alpha + \sqrt{\Delta} \right) < 0$. In
such a case, the equilibrium $\hat{E}_3$ is stable and the whole ecosystem thrives, with both populations at nonzero level. Since only one eigenvalue can be made positive, upon destabilization of $\hat{E}_3$ we would get a saddle. For this to occur, we need the conditions (4.17). It then follows that the saddle implies the existence through it of a separatrix in the phase plane, dividing it into basins of attraction, one each for the remaining equilibria $\hat{E}_1$ and $\hat{E}_2$, which are then mutually exclusive. Thus only one of the two species survives, while the other one becomes extinct. As to which is which, the answer depends on the initial condition of the system. Trajectories emanating from a point in the basin of the equilibrium $\hat{E}_1$ where only the $P$'s thrive tend asymptotically to this point, thus wiping out the $Q$ population, and vice versa.

The Russian biologist Gause in his famous laboratory experiments has determined the evolution of two species of yeast, the $P$ population of *Saccharomyces cerevisiae* and the $Q$ population of *Schizosaccharomyces kefyr*. When living independently from each other (cf. Renshaw, 1991), he found the following growth laws:

\[ P = \frac{13}{1 + e^{3.32816 - 0.21827 t}}, \quad Q = \frac{5.8}{1 + e^{2.47550 - 0.006069 t}}, \]

When allowed to interact, however, the two species became competitors and their evolution would follow the empirical findings

\[ \frac{dP}{dt} = r_P P (1 - s_{PP} P - s_{PQ} Q), \quad \frac{dQ}{dt} = r_Q Q (1 - s_{QQ} Q - s_{QP} P), \]

where

\[ r_P = 0.21827, \quad s_{PP} = 0.01679, \quad s_{PQ} = 3.15 \times s_{PP} = 0.05289 \]
\[ r_Q = 0.06069, \quad s_{QQ} = 0.01046, \quad s_{QP} = 0.439 \times s_{QQ} = 0.00459. \]

### 4.3.2 Symbiotic populations

Some scientists argue that symbiotic communities do not develop easily in ecological time, but rather represent evolutionary interactions. Therefore, population models to take this into account should contain different time scales. However, their remark may be easily questioned by considering one of the most elementary facts in nature, namely, the role that insects, in particular bees, have in the fecundation of flowers (e.g., see Boucher, 1985, p. 85). An entire chapter is devoted to this topic in Boucher (1985, p. 145), which contains a discussion of some aspects of evolution in a mutualistic environment.

Other well-known mutualistic interactions in nature are found in a wealth of different environments, such as in diatom mats in the ocean, between mangroves and root borers, spiders and parasitic wasps, invertebrates and their epibionts, and corals and fish. These as well as many other examples are
illustrated for instance in the book by Boucher (1985). Benefit models for symbiotic communities have been considered in Keeler (1985) for interactions involving myrmecochory, i.e., the phenomenon for which ants find and bury seeds while the plants producing the latter receive benefit by their dispersal. But more common examples include the pollination of plants by several insects, the mycorrhizal fungi, the fungus-gardening ants, the mixed feeding flocks of birds, and the other classical cases of the anemone-damselfish, and ant-plant interactions at extrafloral nectaries. Moths (of gene Tegeticula) pollinating yuccas have also been considered. Of importance is the activity of birds removing and disseminating in the environment the seeds of Casearia corymbosa in Costa Rica (cf. Jantzen et al., 1980). Algae-herbivore mutualistic interactions are studied in Porter (1976), Porter (1977). These are symbiotic communities as some algae survive the digestive process of the herbivore. While the herbivore feeds, the benefit for the undigested algae lies in the possibility of absorption of nutrients released by the herbivores.

The mutualistic model can elementarily be written by means of quadratic ordinary differential equations, as was done for the other interacting species models. We account for the logistic behavior of each independent population but then combine it with additional interaction terms, which are beneficial to both populations, i.e., improve the growth rate of each species and therefore carry a positive sign. Thus let \( P(T) \) and \( Q(T) \) denote the two population sizes. The model is then

\[
\frac{dP}{dT} = P \left( a - bP + cQ \right), \quad \frac{dP}{dT} = Q \left( d + cP - fQ \right).
\]  
(4.20)

Thus the equations express logistic growth for each individual population, and the positive sign for the mixed terms \( PQ \) reflects the remarks introducing the system, i.e., denote the common benefit from interactions among the different species.

The Jacobian of (4.20) is

\[
J = \begin{pmatrix}
 a - 2bP + cQ \\
 cP \\
 d + cP - 2fQ
\end{pmatrix},
\]
(4.21)

and it is easily seen to differ from (4.18) in the sign of the off-diagonal terms and part of the diagonal ones.

The equilibria of the system are four. We list them together with their eigenvalues, the signs of which allow their stability classification.

The origin, \( E_0 \equiv (0,0) \), has eigenvalues \( a \) and \( d \); thus it is always unstable, a repeller along both axes for the model trajectories. The point \( E_1 \equiv \left( \frac{a}{b}, 0 \right) \) is unstable as its eigenvalues are \(-a, \frac{1}{b}(ac + db)\). Also, \( E_2 \equiv \left( 0, \frac{a}{f} \right) \) is unstable, again in view of the positivity of the second eigenvalue, \(-d, \frac{1}{f}(af + cd)\).

The interior equilibrium, \( E_3 \equiv \left( \frac{af + cd}{bf - ce}, \frac{ae + bd}{bf - ce} \right) \), is feasible for \( bf - ce > 0 \),
and it has the eigenvalues
\[ \lambda_{d, \pm}^{(d)} = \frac{1}{2} \left\{ - (bP_3 + fQ_3) \pm \left[ (bP_3 + fQ_3)^2 - 4 (bf - ce) P_3Q_3 \right]^{1/2} \right\}. \] (4.22)

Hence, in view of the feasibility condition, the real parts of the eigenvalues are negative, thus always providing stability. As no other point is stable, the model dynamics must then be drawn to this equilibrium, which is then globally asymptotically stable. Furthermore, at the level provided by \( E_3 \), survival of both species is at a higher level than the one specified by the equilibrium for each single species, this being the heart of mutualism. Thus \( E_3 \) is more favorable than the equilibria provided by the carrying capacity of each species, and which each population would enjoy in the absence of its symbiotic counterpart. Finally, for \( bf < ce \), the \( \omega \)-limit set of the system is the point at \( \infty \), i.e., all trajectories would tend to larger and larger values for both \( P \) and \( Q \). We would thus have an “explosion” of the populations, when \( E_3 \) becomes infeasible. On the other hand, it is easy to show that the trajectories are bounded if the feasibility condition for the interior equilibrium is satisfied, namely, \( bf > ce \). It is enough to sketch the two nullclines in the phase plane and afterwards analyze the sign of the flow to show that it would point “inwards” toward the origin on any curve whose points \((P, Q)\) cut a set in the first quadrant containing \( O \) and \( E_3 \) and lie enough away from the origin.

4.3.3 Leslie–Gower model

As usual, we denote by \( U(T) \) and \( V(T) \) respectively the prey and predator sizes. The Leslie–Gower classical model assumes that when predation is not considered the prey population exhibits logistic growth, so that there are limited resources in the environment, leading to a carrying capacity \( K \) and net birth rate \( r \). The basic feature of the Leslie–Gower model is the assumption that the predator’s carrying capacity is variable, and in particular proportional to the prey size. The proportionality constant \( h \) indicates the prey amount needed to feed a predator in equilibrium conditions:
\[
\frac{dU}{dT} = rU \left( 1 - \frac{U}{K} \right) - cUV, \quad \frac{dV}{dT} = aV \left( 1 - \frac{hV}{U} \right). \] (4.23)

In adimensional form, it becomes
\[
\frac{du}{dt} = u(1 - u - v), \quad \frac{dv}{dt} = v \left( \delta - \beta \frac{v}{u} \right), \quad \delta = \frac{a}{r}, \quad \beta = \frac{ah}{cK^2r}. \] (4.24)

The full analysis of this system can be found in Hsu and Huang (1995). Note only that there are two equilibria, \( E_* \equiv (1, 0) \) and \( E^\dagger \equiv (u^\dagger, v^\dagger) \equiv \frac{\delta}{\beta + \delta}, \frac{\delta}{\beta + \delta} \). The Jacobian is
\[
J = \begin{pmatrix}
1 - 2u - v & -u \\
\beta \frac{v}{u} & \delta - 2\beta \frac{v}{u}
\end{pmatrix}. \] (4.25)
At $E_*$, the eigenvalues are easily seen to be $-1, \delta$ so that this equilibrium is always unstable. At $E^\dagger$, the characteristic equation is $\lambda^2 + (\delta + u^\dagger)\lambda + \delta = 0$ and gives the eigenvalues

$$\lambda_{\pm} = \frac{1}{2} \left[-\delta - u^\dagger \pm \sqrt{(\delta + u^\dagger)^2 - \delta}\right]$$

so that $\lambda_+ < 0$ always, thus $E^\dagger$ is a stable equilibrium. It is a focus if $\delta^2 + (2u^\dagger - 1)\delta + (u^\dagger)^2 < 0$, which then implies $1 - 2u^\dagger - \sqrt{1 - 4u^\dagger} < \delta < 1 - 2u^\dagger + \sqrt{1 - 4u^\dagger}$, and conversely it would be a node.

### 4.3.4 Classical Holling–Tanner model

The Holling–Tanner model for the prey–predator interaction assumes the same dynamics of the Leslie–Gower model for both species, except that the capturing term on prey is replaced by a saturation function $f(U, V)$, i.e., a function with a horizontal asymptote, May (1973), Renshaw (1991), and Tanner (1975). Models for it could be, for instance,

$$f(U, V) = \frac{m}{A + U}UV, \quad f(U, V) = mV(1 - e^{\alpha U}).$$

In this case we consider the Holling type II functional response. The model is then

$$\begin{align*}
\frac{dU}{dt} &= rU \left(1 - \frac{U}{K}\right) - \frac{m}{A + U}UV, \\
\frac{dV}{dt} &= V\gamma \left(1 - h\frac{U}{U}\right).
\end{align*} \tag{4.26}$$

A nondimensional form of system (4.26) can be obtained by setting

$$u \equiv \frac{1}{K}U, \quad v \equiv \frac{m}{\gamma rK}V, \quad t \equiv rT, \quad \delta \equiv \frac{\gamma}{v}, \quad \beta \equiv \frac{\gamma h}{m}, \quad a \equiv \frac{A}{K},$$

so that we get

$$\frac{du}{dt} = u(1 - u) - \frac{1}{a + u}uv, \quad \frac{dv}{dt} = v \left(\delta - \frac{\beta v}{u}\right). \tag{4.27}$$

These can be studied geometrically as follows. The isoclines give respectively a parabola and a straight line through the origin

$$\frac{du}{dt} = 0 : \quad v = (a + u)(1 - v); \quad \frac{dv}{dt} = 0 : \quad v = \frac{\delta}{\beta}u. \tag{4.28}$$

The parabola has the vertex at $(\hat{u}, \hat{v})$, $\hat{u} = \frac{1}{2}(1 - a)$, $\hat{v} = \frac{1}{4}r(1 + a)^2$, and it has positive height $\alpha$ at the origin and a feasible root at $u_0 = 1$. On geometrical grounds, an intersection in the first quadrant always exists, so that a feasible equilibrium is always guaranteed. In fact, for (4.27), we also find the boundary equilibria $O$ and $E_1(1, 0)$ in addition to the interior one $E_2(u^*, v^*)$ with

$$u^* = \frac{1}{2} \left[D + \sqrt{D^2 + 4a}\right], \quad D = 1 - a - \frac{\delta}{\beta}, \quad v^* = u^* \frac{\delta}{\beta}. \tag{4.29}$$
The unconditional existence of the latter is also clear from the algebraic representation, as the root contains a sum of positive quantities, that make it always larger than $D$ and render $u^*$ nonnegative. This model will be further analyzed for stability in Section 4.4.5.

4.3.5 Other growth models

Finally, we briefly mention a few other possible equations for the population growth. They are the Gompertz models:

$$\frac{dU}{dT} = \alpha U - \beta U \ln U, \quad \frac{dU}{dT} = \begin{cases} \alpha U, & U < K \\ \alpha U - \beta U \ln \frac{U}{K}, & U \geq K \end{cases}$$ (4.30)

$$\frac{dU}{dT} = \alpha U^\gamma - \beta U^\gamma \ln U, \quad \frac{dU}{dT} = \alpha U(\ln k - \ln U)^{1+p}. \quad (4.31)$$

The von Bertalanffy models are:

$$\frac{dU}{dT} = \alpha U^{\frac{2}{3}} - \beta U, \quad \frac{dU}{dT} = \begin{cases} \alpha U^\gamma - \beta U, & \gamma < 1 \\ \alpha U - \beta U \ln \frac{U}{K}, & \gamma = 1 \\ \alpha U - \beta U^\gamma, & \gamma > 1 \end{cases}$$ (4.32)

Also, generalized or hyper-logistic are in use:

$$\frac{dU}{dT} = \alpha U^\gamma - \beta U^4, \quad \frac{dU}{dT} = \alpha U^{1-p} - \frac{1}{k}(k - U)^{1+p}. \quad (4.33)$$

Analytical study of these models’ properties can be found in literature; otherwise we leave it to a reader as an excellent exercise to practice the mathematical technique described earlier in this book.

4.3.6 Models with prey switching

“Higher dimensional” interacting population models have also been considered, where the “dimensionality” here refers to the number of species that are considered. This is also in preparation for more complex models to be analyzed at the end of this chapter, and later on in Chapter 7.

Models for prey exhibiting group defense have also been proposed, (see, for instance, Freedman and Wolkowicz (1986)):

$$\frac{dU}{dT} = Ug(U, K) - Vp(U), \quad \frac{dV}{dT} = V(s - q(U)), \quad s, K \in \mathbb{R}^+, \quad (4.34)$$

where $g$, $p$, $q$ are continuously differentiable functions. The first function represents the prey growth rate in absence of predators, $p$ is the predators’ response function, while $q$ represents the predators’ conversion function. The
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assumptions they obey are summarized as follows:

\[ g(0, K) = g(K, K) = 0, \]
\[ \frac{\partial g}{\partial U}(K, K) < 0, \quad \frac{\partial g}{\partial U}(U, K) \leq 0, \quad \frac{\partial g}{\partial K}(U, K) > 0, \]
\[ p(0) = 0, \quad p(U) \geq 0, \]
\[ \frac{dp}{dU} > 0 \text{ for } 0 \leq U < M, \quad \frac{dp}{dU} < 0 \text{ for } U > M, \]
\[ q(0) = 0, \quad q(U) \geq 0, \quad q(M) > s, \]
\[ \frac{dq}{dU} > 0 \text{ for } 0 \leq U < M, \quad \frac{dq}{dU} < 0 \text{ for } U > M, \]

where \( M > 0 \) denotes a suitable constant.

A model incorporating group defense in a community dwelling in a fragmented habitat was developed by Khan et al. (1998):

\[
\begin{align*}
\frac{dU_1}{dT} &= (\alpha_1 - \epsilon_1)U_1 + \epsilon_2 p_{21}U_2 - \beta_1 U_2^2 V \left/ U_1 + U_2 \right., \quad (4.35) \\
\frac{dU_2}{dT} &= (\alpha_2 - \epsilon_2)U_2 + \epsilon_1 p_{12}U_1 - \beta_2 U_2^2 V \left/ U_1 + U_2 \right., \\
\frac{dV}{dT} &= V \left( -\nu + \frac{\delta_1 \beta_1 U_2}{U_1 + U_2} + \frac{\delta_2 \beta_2 U_1^2}{U_1 + U_2} \right),
\end{align*}
\]

where \( U_1 \) and \( U_2 \) denote the immature and adult populations, \( \tau \) representing the time from birth to reach maturity, the term \( \beta U_2(T - \tau)e^{-\mu_1 \tau} \) expressing the fraction of young individuals who reach the...
maturity stage, and $gU_1 U_2$ and $hU_1 U_2$ are interaction terms. The parameters have the following meanings: $\beta$ is the per capita birth rate of the prey species in stage 2, $\gamma$ is the maturation rate from stage 1 to stage 2, $\mu_i$ is the per capita death rate, $b_i$ is the capture rate of prey species of stage $i$, $c_i$ is the conversion factor for prey species of stage $i$, and $d$ is the per capita death rate of the predator.

Another switching similar model is presented in Tansky (1978):

$$\frac{dU_1}{dT} = U_1 \left( E_1 - \frac{aV}{1 + \left( \frac{U_2}{U_1} \right)^n} \right),$$

$$\frac{dU_2}{dT} = U_2 \left( E_2 - \frac{bV}{1 + \left( \frac{U_2}{U_1} \right)^n} \right),$$

$$\frac{dV}{dT} = V \left( -E_3 + \frac{aU_1}{1 + \left( \frac{U_2}{U_1} \right)^n} + \frac{bU_2}{1 + \left( \frac{U_2}{U_1} \right)^n} \right),$$

where the positive integer $n$ is a parameter.

A similar model was also considered by Khan et al. (2004):

$$\frac{dU_1}{dT} = g_1 U_1 \left( 1 - \frac{U_1}{K_1} \right) + e_2 p_{21} U_2 - \frac{\beta_1 U_1 U_2 V}{U_1 + U_2},$$

$$\frac{dU_2}{dT} = g_2 U_2 \left( 1 - \frac{U_2}{K_2} \right) + e_1 p_{12} U_1 - \frac{\beta_2 U_1 U_2 V}{U_1 + U_2},$$

$$\frac{dV}{dT} = V \left( -\mu + \frac{\delta_1 \beta_1 + \delta_2 \beta_2 U_1 U_2}{U_1 + U_2} \right),$$

where $K_i$ represents the carrying capacity of each prey; $g_i$ is the net effect of birth, death and migration rates; and the remaining parameters have interpretations as above, (4.36), namely, $e_i$ and $p_{ij}$ represent the strength of the barrier and the probability of moving to a different habitat, $\beta_i$ the predator responses to each prey, $\delta_i$ the conversion factors, and $\mu$ the per capita death rate of predators.

### 4.4 Global stability

Let us consider again the classical Lotka–Volterra system,

$$\frac{dU}{dT} = U(a - bV), \quad \frac{dV}{dT} = V(-m + cU).$$

(4.39)
In an attempt to find a closed form solution, we can calculate the slope of the trajectories in the $UV$ phase plane. Using the chain rule and exploiting (4.39), we get
\[ \frac{dV}{dU} = \frac{dV}{dT} \times \left[ \frac{dU}{dT} \right]^{-1} = \frac{V(-m + cU)}{U(a - bV)}, \]
so that variables can be separated to yield
\[ \int (a - bV) \frac{dV}{V} = \int (-m + cU) \frac{dU}{U}; \quad a \ln V - bV = -m \ln U + cU - h. \]

Therefore we can introduce the function
\[ z = L(U, V) \equiv -a \ln V + bV - m \ln U + cU - h, \quad (4.40) \]
which represents a surface defined on the phase space. The orbits of the model (4.39) are the loci of points $(U, V)$ in the phase space for which $L(U, V) = h$ for the constant $h \in \mathbb{R}$, i.e., the level curves $z = h$ of the surface $z = L(U, V)$; the larger $h$ is, the higher these levels are. In particular, given the initial condition $(U_0, V_0)$, the solution of the initial value problem is the level curve of $L = h_0$, where $h_0$ corresponds to
\[ h_0 = -a \ln V_0 + bV_0 - m \ln U_0 + cU_0. \quad (4.41) \]

To further study $L$, it is immediately seen that $\nabla L = 0$ at the equilibrium point of the system (4.39), $E_\infty \equiv (U_\infty, V_\infty)$ with $U_\infty = \frac{m}{c}$, $V_\infty = \frac{a}{b}$, and from this we define the value of the constant there as
\[ h_\infty = -a \ln V_\infty + bV_\infty - m \ln U_\infty + cU_\infty \quad (4.42) \]
\[ = a \left( 1 - \ln \frac{a}{b} \right) + m \left( 1 - \ln \frac{m}{c} \right). \]

The shape of the surface can be investigated by intersecting it with vertical planes. On $V = \tilde{V}$, we easily find that $L \to +\infty$ both when $U \to +\infty$ as well as $U \to 0^+$. Similarly, on $U = \tilde{U}$, we have $L \to +\infty$ for $V \to 0^+$ and $V \to +\infty$. It appears that the equilibrium $E_\infty$ is then a minimum. To further support this statement, we consider the intersection with a generic plane $\bar{V} = rU + s$. Then
\[ \bar{L}(U) \equiv L(U, \bar{V}) \equiv -m \ln U + cU - a \ln (rU + s) + b(rU + s) \]
and we must distinguish several cases. For $r, s > 0$, as $U \to +\infty$ we have
\[ \bar{L} = U \left[ c - m \ln \frac{U}{\bar{V}} \right] + (rU + s) \left[ b - a \ln \frac{rU + s}{rU + s} \right] \to +\infty. \]

The behavior $L \to +\infty$ can also be discovered for $U \to +\infty$ in the other cases. The function indeed needs to be investigated also on the remaining parts of
the boundary of its domain, which is $\mathbb{R}_+ \times \mathbb{R}_+ = \{(U, V) : U > 0, V > 0\}$. By taking the other suitable limits, namely, for $r, s > 0$ as $U \to 0^+$, while for $r > 0, s < 0$ as $V \equiv rU + s \to 0^+$, we find again $L \to \infty$. The case $r < 0, s > 0$ needs more care as the limits at infinity cannot be taken; rather, we must evaluate $L$ when both $U \to 0^+$ and $rU + s \to 0^+$, but again the conclusion is $L \to \infty$. The surface $L$ thus appears to be a bowl, going up vertically on the coordinate axes and also growing as the points in the domain drift toward infinity. $E_\infty$ then appears to be its absolute minimum. To mathematically substantiate this claim, we can compute the Hessian matrix,

$$H_L = \begin{vmatrix} mU^{-2} & 0 \\ 0 & aV^{-2} \end{vmatrix} > 0,$$

to discover that the curvature of the surface is always positive, i.e., the surface is everywhere convex. Therefore $E_\infty$ really represents an absolute minimum. By (4.40) and (4.42) we would then have $L(E_\infty) = 0$. This is the first property of the Lyapunov function. We have also shown that $L(U, V) \geq L(E_\infty) = 0$, which constitutes its second property. Below we will discover finally its third and fundamental property, namely, that the system trajectories enter into this bowl. In the present case, calculating the tangential derivative of $L$ we find that it is tangent to the solution trajectories,

$$\frac{dL}{dT} = \frac{\partial L}{\partial U} \frac{dU}{dT} + \frac{\partial L}{\partial V} \frac{dV}{dT} = -\frac{m}{U} U(a - bV) + cU(a - bV) - \frac{a}{V} V(-m + cU) = 0.$$

The function $L$ also allows the explicit analytical determination of the or-
bits. To this end, using the initial condition (4.41) coupled with the definition
(4.40) of $L$, we have

$$-m(\ln U - \ln U_0) + c(U - U_0) = a(\ln V - \ln V_0) - b(V - V_0)$$

from which

$$c(U - U_0) + b(V - V_0) = \ln \left( \frac{V}{V_0} \right)^a + \ln \left( \frac{U}{U_0} \right)^m$$

and finally the closed forms of the orbits

$$\left( \frac{U}{U_0} \right)^m \left( \frac{V}{V_0} \right)^a = e^{c(U - U_0) + b(V - V_0)}.$$

To investigate further the latter, let us study small perturbations around the
equilibrium, by defining $u = U - U_\infty = U - \frac{m}{c} \in \mathbb{R}$, $v = V - V_\infty = V - \frac{a}{b} \in \mathbb{R}$. Substitution into $L(U, V) = h$ brings

$$- m \left[ \ln \frac{m}{c} + \ln \left( 1 + \frac{cu}{m} \right) \right] + m \left( 1 + \frac{cu}{m} \right)$$

$$- a \left[ \ln \frac{a}{b} + \ln \left( 1 + \frac{bv}{a} \right) \right] + a \left( 1 + \frac{bv}{a} \right) = h.$$
Now, using Taylor’s formula and keeping only terms up to the second order, we are led to
\[-m \left[ \ln \frac{m}{c} + \frac{cu}{m} - \frac{1}{2} \left( \frac{cu}{m} \right)^2 \right] + m \left( 1 + \frac{cu}{m} \right)\]
\[-a \left[ \ln \frac{a}{b} + \frac{bv}{a} - \frac{1}{2} \left( \frac{bv}{a} \right)^2 \right] + a \left( 1 + \frac{bv}{a} \right) = h.\]

Linearization and further simplifications give
\[\frac{m}{2} \left( \frac{cu}{m} \right)^2 + a \left( \frac{bv}{a} \right)^2 = -m - a + h + m \ln \frac{m}{c} + a \ln \frac{a}{b}\]
and, finally, using (4.42) we arrive at
\[\frac{u^2}{A^2} + \frac{v^2}{B^2} = H, \quad A^2 = \frac{2m}{ct}, \quad B^2 = \frac{2a}{by}, \quad H = h - h_\infty \geq 0.\]
The trajectories around the equilibrium are therefore close to ellipses.

### 4.4.1 General quadratic prey–predator system

We consider now the prey–predator interactions with logistic growth in the prey, and competition among the predators, which do not have other food sources:
\[\frac{dU}{dT} = U(a - bV - \frac{U}{K_1}), \quad \frac{dV}{dT} = V(-m + cU - \frac{V}{K_2}),\]
with nonboundary equilibrium at \(E^* = (U^*, V^*)\), where
\[U^* \equiv K_1 \frac{a + mbK_2}{1 + bcK_1K_2}, \quad V^* \equiv K_2 \frac{-m + acK_1}{1 + bK_1K_2},\]
feasible for \(acK_1 > m\). Let us consider once again the perturbations \(u = U - U^*, \ v = V - V^*\) and proceed as follows:
\[\frac{du}{dv} = -\frac{u + U^*}{v + V^*} \frac{K_2 u + bvK_1}{cK_2u - v}, \quad \frac{du}{dv} = \frac{cK_2 u - v}{u + U^*} \frac{dv}{v + V^*} = -\frac{dv}{v + V^*} \frac{u + bvK_1}{v + V^*},\]
which is a separable equation,
\[cdv - cU^* \ln \frac{u + U^*}{u + U^*} = \frac{dv}{v + V^*} - \frac{bv + bV^*}{v + V^*},\]
which upon integration gives
\[cu - cU^* \ln \frac{u + U^*}{U^*} - \frac{v}{K_2} \ln (u + U^*) = -\frac{u}{K_1} \ln (v + V^*) - bv + bV^* \ln \frac{v + V^*}{V^*} + C,\]
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where the term \( C \) represents the constant of integration. This expression suggests to define the following Lyapunov function:

\[
L(u, v) \equiv c U^* \left[ \frac{u}{U^*} - \ln \left( 1 + \frac{u}{U^*} \right) \right] + b V^* \left[ \frac{v}{V^*} - \ln \left( 1 + \frac{v}{V^*} \right) \right].
\]

The property of the logarithm, \( x \geq \ln(1 + x) \) for any \( x > 0 \), implies that \( L(u, v) \geq 0 \), with \( L(0, 0) = 0 \). This of course corresponds to the vanishing of \( L \) at the equilibrium point in the original coordinates. Finally,

\[
\nabla L(u, v) \equiv \left( \frac{c}{u + U^*}, \frac{b}{v + V^*} \right)^T.
\]

It thus follows that the directional derivative along the solution trajectories of (4.43) is

\[
\frac{dL}{dT} = \nabla L \cdot (\frac{du}{dT}, \frac{dv}{dT})^T = \frac{cu}{u + U^*} (u + U^*) (-bv - \frac{u}{K_1}) + \frac{bv}{v + V^*} (v + V^*) (+cu - \frac{v}{K_2}) = -\frac{1}{K_1} cu^2 - \frac{1}{K_2} bv^2 < 0.
\]

This result thus shows that the trajectories make an obtuse angle with the (outward) normal to the level surfaces \( L(u, v) = h \), i.e., they enter into the bowl defined by the Lyapunov function independently of their initial values, i.e., the equilibrium \( E^* \) is indeed a global attractor of the system (4.43).

4.4.2 Mathematical tools for analyzing limit cycles

Here we want to mention some results in planar dynamical systems that are useful in the investigation of solutions’ sustained oscillations; see Hirsch and Smale (1974) and Strogatz (1994).

Poincaré–Bendixson theorem

A closed orbit \( \Gamma \) of a planar dynamical system is a nonempty subset of the phase plane, \( \Gamma \subset \mathbb{R}^2 \), not containing equilibrium points.

The Poincaré–Bendixson theorem states that if \( \Gamma \subset \mathbb{R}^2 \) is a closed and bounded set, and \( \frac{df}{dt} = f(x) \in C^1(\mathbb{R}^2) \) is a vector field defined on an open set containing \( \Gamma \), if \( \Gamma \) does not contain equilibrium points, and if there is a trajectory \( L \subset \Gamma \) contained in the orbit, then \( L \) is a closed orbit or tends to a closed orbit (i.e., a limit cycle). In other words, in such conditions \( \Gamma \) contains at least a limit cycle.
The theorem can be exploited by determining a positively invariant set Ω for the dynamical system, i.e., such that the system trajectories can only enter into Ω, and then if possible trying to destabilize the equilibria that are found inside Ω. The application of the above theorem would then show that at least a stable limit cycle exists around the equilibrium.

Dulac’s criterion

The next criterion is negative, in the sense that if satisfied it shows that limit cycles do not exist; see Strogatz (1994). It is essentially a consequence of Green’s theorem.

Let $A \subset \mathbb{R}^2$ be a simply connected region in the plane, i.e., it does not contain holes, and let $f$ and $g$ be continuously differentiable functions in $A$, i.e., mathematically $f, g \in C^1(A)$. Given the system

$$\frac{dx}{dT} = f(x, y), \quad \frac{dy}{dT} = g(x, y), \quad (4.44)$$

if a function $\beta(x, y) \in C^1(\mathbb{R}^2)$ exists for which the function

$$\frac{\partial}{\partial x} [\beta(x, y)f(x, y)] + \frac{\partial}{\partial y} [\beta(x, y)g(x, y)]$$

is of one sign in $A$, then in $A$ no closed orbit can exist.

In general this result is utilized by taking $A \equiv \mathbb{R}^2$ and $\beta(x, y) = (xy)^{-1}$.

As an example, let us consider the quadratic competition model

$$\frac{dU}{dT} = \alpha U \left(1 - \frac{U}{K}\right) - aUV, \quad \frac{dV}{dT} = rV \left(1 - \frac{V}{H}\right) - bUV.$$

With the above choice for $\beta$ we find

$$\frac{\partial}{\partial U} \left\{ \frac{1}{UV} \left[ aU \left(1 - \frac{U}{K}\right) - aUV \right] \right\} + \frac{\partial}{\partial V} \left\{ \frac{1}{UV} \left[ rV \left(1 - \frac{V}{H}\right) - bUV \right] \right\}$$

$$= -\frac{\alpha}{KV} - \frac{r}{HU} < 0.$$

It follows then that quadratic competition models do not possess limit cycles, and this fact clearly stems from the logistic terms in the equations, the only ones surviving after the above differentiation.

Potential systems

A related negative result (Strogatz, 1994) states that for potential systems, limit cycles do not exist; in other words, if a function $Z(x)$ exists for which the field $f(x)$ is the gradient of $Z$, $f(x) = \nabla Z(x)$, i.e., $Z$ is a potential function for the field, then no limit cycle for the dynamical system can exist.
As an example let us consider two logistically growing and not interacting populations,

\[
\frac{dU}{dT} = \alpha U \left(1 - \frac{U}{K}\right), \quad \frac{dV}{dT} = r V \left(1 - \frac{V}{H}\right).
\]

Considering

\[
Z(U, V) = \frac{\alpha}{2} U^2 - \frac{\alpha}{3K} U^3 + \frac{r}{2} V^2 - \frac{r}{3H} V^3,
\]

the above condition is immediately verified. Of course each population evolves naturally toward its own carrying capacity undisturbed by the other one, these equilibria being stable in view of the results on the single-population models, and in the two-dimensional phase plane the corresponding equilibrium must be stable. The corresponding Jacobian is a diagonal matrix that when evaluated at the equilibrium \((K, H)\), has negative entries, namely, \(J_{(K,H)} = \text{diag}(-\alpha, -r)\).

### 4.4.3 Routh–Hurwitz conditions

Stability of an equilibrium in dynamical systems as seen earlier is related to the negative sign of its eigenvalues, if real, or of their real part if complex. A criterion to determine the signs of the roots of a polynomial equation is the Routh–Hurwitz criterion. Namely, for the following polynomial equation the related determinants are defined:

\[
\sum_{i=0}^{n} a_i \lambda^i = 0, \quad D_1 = a_{n-1}, \quad D_2 = \begin{vmatrix} a_{n-1} & a_{n-3} \\ a_n & a_{n-2} \end{vmatrix}, \quad (4.45)
\]

\[
D_3 = \begin{vmatrix} a_{n-1} & a_{n-3} & a_{n-5} \\ a_n & a_{n-2} & a_{n-4} \\ 0 & a_{n-1} & a_{n-3} \end{vmatrix}, \quad \ldots, \quad D_n = \begin{vmatrix} a_{n-1} & a_{n-3} & a_{n-5} & \ldots & 0 & 0 \\ a_n & a_{n-2} & a_{n-4} & \ldots & 0 & 0 \\ 0 & a_{n-1} & a_{n-3} & \ldots & 0 & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots \\ 0 & 0 & 0 & \ldots & 0 & a_0 \end{vmatrix},
\]

with the clear understanding that \(a_j = 0\) for \(j < 0\) or \(j > n\). The criterion states that all the roots have negative real parts if and only if all the \(D_j\)'s are positive.

There is a variant of the above criterion, the Liénard–Chipart criterion, which essentially states that for stability if all coefficients are positive, \(a_i > 0\) for \(i = 0, \ldots, n\), only the determinants \(D_{n-1}, D_{n-3}, D_{n-5}, \ldots\) need to be positive.

It is useful to see what the above statements amount to for polynomials up to degree 4. Observe that by a change of sign of the whole polynomial, the assumption \(a_n > 0\) can always be made. Then, for a linear polynomial with root \(\lambda_1\), trivially we need only

\[
D_1 = a_0 > 0, \quad \lambda = -\frac{a_0}{a_1} < 0. \quad (4.46)
\]
The most useful cases, also for the subsequent chapters, are the low degree polynomials. For a quadratic, the conditions reduce to
\[ a_0 > 0, \quad a_1 > 0. \tag{4.47} \]
Geometrically, they imply that the height and the slope at the origin are both positive, so that the vertex lies to its left, and the possible roots occur only on the left-half plane. For a cubic they are
\[ a_0 > 0, \quad a_2 > 0, \quad a_2a_1 > a_0a_3, \tag{4.48} \]
and for a quartic they are
\[ a_0 > 0, \quad a_3 > 0, \quad a_2a_3 > a_1a_4, \quad a_1(a_2a_3 - a_1a_4) > a_0a_3^2. \tag{4.49} \]

### 4.4.4 Criterion for Hopf bifurcation

In general, the Routh–Hurwitz conditions are needed to determine the presence of negative roots for an equation. When the latter is the characteristic equation of an equilibrium point, the criterion tells us the stability of the associated equilibrium. If the latter depends on one parameter \( \mu \), it is possible that its stability changes with modifications of this parameter value. To determine this, without explicitly calculating the zeros of the characteristic equation, another criterion is available; see Liu (1994). Let this characteristic polynomial be
\[ P_\mu(\lambda) = \sum_{i=0}^{n} p_i(\mu)\lambda^i \]
and define, with the same conventions \( p_j(\mu) \equiv 0 \) for \( j < 0 \) or \( j > n \) used above,
\[ L_n(\mu) = \begin{pmatrix} p_1(\mu) & p_0(\mu) & \ldots & 0 \\ p_2(\mu) & p_2(\mu) & \ldots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ p_{2n-1}(\mu) & p_{2n-2}(\mu) & \ldots & p_n(\mu) \end{pmatrix}, \tag{4.50} \]
and denote by \( D_j \) the principal minors of order \( j \) of the above matrix. If there is a locus of equilibria \((x(\mu), \mu)\) emanating from \( x(\mu_0) = x_0 \) for the dynamical system \( \frac{dx}{dT} = f(x, \mu) \), the sufficient conditions for a simple Hopf bifurcation are that the Jacobian of the system at the equilibrium, \( J_x[f_{\mu_0}(x_0)] \), possesses two purely imaginary eigenvalues, \( \lambda(\mu) \), while all the other roots have negative real parts. Moreover,
\[ \frac{d\text{Re}(\lambda(\mu_0))}{d\mu} \neq 0. \tag{4.51} \]
The criterion then states that the above sufficient conditions are equivalent to
\[ p_0(\mu_0) > 0, \quad D_1(\mu_0) > 0, \quad \ldots, \quad D_{n-2}(\mu_0) > 0, \quad D_{n-1}(\mu_0) = 0 \quad \text{and} \quad \frac{dD_{n-1}(\mu_0)}{d\mu} \neq 0. \tag{4.52} \]
4.4.5 Instructive example

As an example of the above techniques, we consider again the Holling–Tanner model introduced in Section 4.3.4. The Jacobian in this case is

\[
J = \begin{pmatrix}
1 - 2u - au \frac{1}{(a + u)^2} & -u \frac{1}{a + u} \\
\frac{\beta \mathrm{e}^2}{u^2} & \delta - 2\beta \frac{\mathrm{e}}{u}
\end{pmatrix}.
\]

(4.53)

The origin and \( \hat{E}_1 \) are easily seen to be always unstable, having at least a positive eigenvalue. Using (4.29), the Jacobian at the equilibrium \( \hat{E}_2 = (u^*, v^*) \) can be rewritten as

\[
J^* = \begin{pmatrix}
u^* \left( \frac{\delta}{\beta} - \sqrt{D^2 + 4a} \right) - u^* \frac{1}{u + u^*} \\
\frac{\beta \mathrm{e}^2}{u^2} & -\delta
\end{pmatrix}.
\]

(4.54)

The Routh–Hurwitz criterion for the quadratic characteristic equation (4.47) in this case requires \(-\text{Tr}(J^*) > 0\) and \(\det(J^*) > 0\). Easily, we find

\[
\det(J^*) = \frac{u^* \delta \sqrt{D^2 + 4a}}{a + u^*} > 0.
\]

Moreover, for the trace of the above matrix, using again (4.29), we must have

\[
-\text{Tr}(J^*) = \frac{u^* \delta \sqrt{D^2 + 4a}}{a + u^*} + \delta > 0.
\]

(4.55)

If the abscissa of the equilibrium lies to the right of the abscissa of the vertex of the parabola (4.28), namely, \( u^* > \hat{u} \), i.e. \( \sqrt{D^2 + 4a} - \delta > 2a \), then (4.55) holds and \( E_2 \) is then stable. For \( u^* < \hat{u} \) instead the condition (4.55) must be checked for stability.

The equilibrium \( \hat{E}_2 \) can be further analyzed on geometrical grounds. Let us consider a rectangle \( R \) with vertex at the origin and the opposite one \((u^*, v^*)\) with \( u^* > u_0 = 1 \), where we recall that \( u_0 \) is the positive root of the isocline \( \frac{du}{dT} = 0 \), which as we know is a parabola (4.28), and \( v^* > \hat{v} \), the height of its vertex. It is easily seen that the flow of the dynamical system enters it on the two sides that do not lie on the coordinate axes. Therefore, \( R \) is a positively invariant set. All its equilibria are unstable if (4.55) is violated. In this case, by applying the Poincaré–Bendixson theorem, a limit cycle must arise around \( \hat{E}_2 \).

Figure 4.1 reports some results for chosen parameter sets. In the first column \( E_2 \) is stable since \( u^* > \hat{u} \). The remaining columns show some cases for \( u^* < \hat{u} \) and in particular two limit cycles.

To complete the discussion, we mention two more recent results for the Holling–Tanner model. It has been shown to possess two bifurcation points (Braza, 2003); when the latter nearly coincide, the stable branches of the periodic solution connect, but by changing the growth rate, the Hopf-bifurcation
FIGURE 4.1: Interior equilibrium and limit cycles for the Holling–Tanner model. Top: populations vs time; center: isocline diagram; bottom: phase plane plots. Parameter values for first column are $a = 0.2$, $\beta = 0.1$, and $\delta = 0.05$. Parameter values for second column are $a = 0.2$, $\beta = 0.1$, and $\delta = 0.12$. Parameter values for third column are $a = 0.2$, $\beta = 0.1$, and $\delta = 1.3$. Parameter values for fourth column: $a = 0.12$, $\beta = 0.1$, and $\delta = 0.23$.

points separate, the limit cycle becomes unstable and this may possibly lead to population outbreaks.

Global stability can be shown by constructing a suitable Lyapunov function; for details see Hsu and Huang (1995). The analysis leads to the following conclusions. Both species can be shown to persist globally in the ecosystem. Moreover, if the prey carrying capacity is small, then both populations tend to constant values, and no periodic behavior is possible. For the predator net birth rate larger than the prey one, there is no limit cycle. A limit cycle exists if the maximal consumption is neither too small nor too large.

4.4.6 Poincaré map

We outline now another mathematical technique useful for analyzing the stability of limit cycles, but on a rather sophisticated and artificial biological
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example. Essentially we consider a prey–predator model that includes the Allee effect and migration.

With the usual notation let us consider the model

\[ \frac{dU}{dT} = A + BU + CU^2 - U^3 + f(U, V), \tag{4.56} \]
\[ \frac{dV}{dT} = H + KV + LV^2 - V^3 + g(U, V), \]

where the interaction terms are assumed to be

\[ f(U, V) = -V[1 + (U - U_*)(V - 2V_*)], \] \tag{4.57}
\[ g(U, V) = U[1 + (V - V_*)(U - 2U_*)] \]

(a generalized populations dependence on the predation rate and the conversion factor), and the coefficients are chosen in the following particular way:

\[ A = V_* - U_* + U_*^3 + U_*V_*^2, \quad B = 1 - 3U_*^2 - V_*^2, \tag{4.58} \]
\[ C = 3U_*(> 0), \quad H = U_*^2V_* + V_*^3 - U_* + V_*, \]
\[ K = -1 - U_*^2 - 3V_*^2, \quad L = 3V_*(> 0). \]

One of course can ask what the quantities \( U_* \) and \( V_* \) are, and here is the second artificial aspect of the model. They are supposed to represent an equilibrium value for the two populations. Substituting (4.58)–(4.57) into (4.56) and expanding we find

\[ \frac{dU}{dT} = -(V - V_*) + (U - U_*) - (U - U_*)^3 - (U - U_*)(V - V_*), \tag{4.59} \]
\[ \frac{dV}{dT} = U - U_* + V_ - V - (U - U_*)^2(V - V_*) - (V - V_*)^3. \]

We now introduce \( x = U - U_* \) and \( y = V - V_* \) as perturbations around the equilibrium value, so that (4.59) turns to

\[ \frac{dx}{dT} = -y + x - x^3 - xy^2 = -y + x(1 - x^2 - y^2), \tag{4.60} \]
\[ \frac{dy}{dT} = x + y - x^2y - y^3 = x + y(1 - x^2 - y^2). \]

At this point, the introduction of polar coordinates becomes natural. On differentiating \( r^2 = x^2 + y^2 \) and \( \theta = \arctan(y/x) \), we find

\[ \frac{dr}{dT} = \frac{x}{r} \frac{dx}{dT} + \frac{y}{r} \frac{dy}{dT}, \]
\[ \frac{d\theta}{dT} = \frac{x}{x^2 + y^2} \frac{dy}{dT} - \frac{y}{x^2 + y^2} \frac{dx}{dT} = \frac{x}{r^2} \frac{dy}{dT} - \frac{y}{r^2} \frac{dx}{dT}. \]
On using (4.60), in our case we find
\[
\frac{dr}{dT} = r \left[ -y + x(1 - r^2) \right] + \frac{y}{r} \left[ x + y(1 - r^2) \right]
\]
(4.61)
\[= \frac{x^2}{r} - \frac{x^2 r^2}{r} + \frac{y^2}{r} - \frac{y^2 r^2}{r} = r(1 - r^2),\]
\[
\frac{d\theta}{dT} = \frac{x}{r^2} \left[ x + y(1 - r^2) \right] - \frac{y}{r^2} \left[ -y + x(1 - r^2) \right] = \frac{1}{r^2} (x^2 + y^2) = 1.
\]

On integrating the second one, clearly \(\theta = T + T_0\), thus the phase is tied to the flow of time, this already suggesting some kind of oscillatory behavior. Using partial fractions on the former,
\[
\frac{dr}{r} + \frac{1}{2} \frac{dr}{1 - r} - \frac{1}{2} \frac{dr}{1 + r} = dT,
\]
so that, upon integration,
\[
\ln \frac{r}{\sqrt{1 - r^2}} = T + C, \quad \frac{r}{\sqrt{1 - r^2}} = \tilde{K} e^T, \quad (4.62)
\]
\[
\frac{r^2}{1 - r^2} = K e^{2T}, \quad r(T) = \frac{\tilde{K} e^T}{\sqrt{1 + K e^{2T}}},
\]
with \(\tilde{K}^2 = K\). Notice that for \(T \to \infty\) starting from the initial condition inside the unit circle, we have
\[
r(T_0) = r(0) = \frac{\tilde{K}}{\sqrt{1 + K}} < 1, \quad r(T) \to \frac{\tilde{K}}{\sqrt{K}} = 1. \quad (4.63)
\]

To better understand this behavior, let us consider a time interval \(T_1 - T_0 = 2\pi\). The phase then returns to its original value \(\theta(T_1) = \theta(T_0) + 2\pi \equiv \theta(T_0)\), but the point in the plane moves away from the origin, because its distance from the origin is now larger:
\[
r(T_1) = r(2\pi) = \frac{\tilde{K}}{\sqrt{1 + K + e^{-4\pi}}} > r(T_0) = r(0),
\]
which is a true inequality, reducing itself to \(1 + K > \exp(-4\pi) + K\). Now, the map \(P : \mathbb{R} \to \mathbb{R}\) defined by \(P(r_k) = r_{k+1}\) is the Poincaré map, which can be constructed on any arbitrary line with a given phase \(\theta_\ast\). Another important remark that we make at this point concerns the unit circle. It is easily seen from the first equation of (4.61) that \(r = 1\) is an equilibrium solution of the dynamical system. Therefore, because trajectories cannot be crossed by the existence and uniqueness theorem, the above reasoning with the Poincaré map shows that the unit circle can only be approached from the inside. To examine also the behavior from the outside, since (4.63) implies \(r(0) < 1\), we need to rewrite the solution of (4.62) in another form for \(r_0 > 1\):
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so that, finally,

\[ r^2(T) = \frac{Ke^{2T}}{1 + Ke^{2T}} = \frac{1}{1 + e^{-2T} \left( \frac{1}{r_0} - 1 \right)} \tag{4.64} \]

\[ r(T) = \left[ 1 + e^{-2T} \left( \frac{1}{r_0} - 1 \right) \right]^{-\frac{1}{2}}. \]

Now it is easily seen that the Poincaré map satisfies \( P(r_0) > 1 \), and also \( r_1 = P(r_0) < r_0 \). Indeed, the two conditions respectively become

\[ r_1 = \left[ 1 + e^{-4\pi} \left( \frac{1}{r_0} - 1 \right) \right]^{-\frac{1}{2}} > 1^{-\frac{1}{2}} \equiv 1; \quad \left[ 1 + e^{-4\pi} \left( \frac{1}{r_0} - 1 \right) \right]^{-1} < r_0^2, \]

\[ 1 + e^{-4\pi} \left( \frac{1}{r_0} - 1 \right) > r_0^2; \quad (1 - e^{-4\pi}) \left( 1 - \frac{1}{r_0^2} \right) > 0, \]

which are always verified. One final consideration. While the above computations have been carried out in almost every detail to double check the assertions, a simple consideration could have led to the same conclusions. Indeed, observe that the system rotates in view of the second equation of (4.61), while the right-hand side of the first one is a cubic polynomial \( p_3(r) \) with a negative biologically infeasible root \( r = -1 \), the second one located in the origin and the positive one giving the unit circle as already remarked above, \( r = 1 \). On plotting it, we find that \( p_3(r) > 0 \) for \( 0 < r < 1 \), so that the origin must be an unstable equilibrium, while \( r = 1 \) in polar coordinates is a stable point. But that means, in cartesian coordinates, that the unit circle is stable. Thus we have found a stable limit cycle.

As a final biological interpretation of this result, the equilibrium \((U_*, V_*)\) of the original system is unstable and around it there is a stable limit cycle \((U_* - U)^2 + (V_* - V)^2 = 1\). In these considerations note that the feasibility of both has not been analyzed, but just assumed.

4.5 Food web

Natural environments are not confined to contain only two populations, rather in general many species intermingle. Their interactions may be of all the kinds previously discussed, i.e., of competitive nature, or mutually beneficial. It turns out that in some circumstances a population that is a predator of another one is itself the prey of a higher trophic level predator. In this section we describe a very general model that attempts a more complex description of reality. It is meant as a brief introduction to food chains. We address a closed ecosystem, in which trophic levels from a top predator down to the mineral.
Interacting populations

nutrients on which the plants feed are accounted for. The analysis should assess its possible sustainable equilibria and their stability. The mineral substrate and homogeneously mixed populations on three trophic levels are the populations in the environment, representing vegetation, herbivore, and carnivore animals. The homogeneous mixing mathematically implies that the predation terms are taken to be bilinear, i.e., proportional to both interacting populations. This also holds for the interactions among plants and minerals, as the growth of plants depends with direct proportionality on the abundance of the mineral substrate, while the latter is depleted in a way directly proportional to the plants population. It is important to also model the mineral level in a closed system, as it represents the bottom level to which organic matter is recycled. Questions of time scales are disregarded in this presentation.

Let $M$ represent the minerals, $P$ the plants, $H$ the herbivores, and $C$ the carnivores. Keeping on following the general convention of the book that all the parameters are nonnegative unless otherwise stated, the model then is given by

$$\frac{dM}{dT} = m_1 P + m_2 H + m_3 C - b_1 PM, \quad (4.65)$$
$$\frac{dP}{dT} = b_2 PM - m_1 P - c_1 PH,$$
$$\frac{dH}{dT} = c_2 PH - m_2 H - d_1 CH,$$
$$\frac{dC}{dT} = d_2 CH - m_3 C.$$

From the bottom, i.e., the first equation, decaying organic matter is converted into minerals and they in turn are depleted by the plants’ uptake at rate $b_1$ for their own growth. The second equation describes this uptake from the plants’ viewpoint, at rate $b_2 < b_1$. Plants are also grazed by herbivores at rate $c_1$ and have an intrinsic mortality $m_1$. Similar considerations hold for the herbivores, feeding at rate $c_2$ on plants, and for carnivores feeding on the former at rate $d_2$. Notice that we do not assume logistic growth, in part to keep the mathematics tractable, in view of the nine parameters the model already contains, as well as to investigate the system’s behavior assuming it to be closed, i.e., when the possible carrying capacities are dictated by the availability of the nutrients at the lower trophic level. Indeed, as the only food source is given by the trophic level just below the one considered, as long as the latter supports the former, the assumption of “unlimited” growth is sound.

To reduce the parameters, let us introduce new dependent variables as follows: $M = \tilde{\alpha} x_1(t)$, $P = \beta x_2(t)$, $H = \gamma x_3(t)$, $C = \delta x_4(t)$, $T = \pi t$, and
define the new coefficients as
\[ p_1 = \frac{b_2}{b_1}, \quad p_2 = \frac{m_1 c_1}{m_2 b_2}, \quad p_3 = \frac{c_2}{b_1}, \quad p_4 = \frac{m_1 d_1}{m_2 b_2}, \quad p_5 = \frac{m_2}{m_1}, \quad p_6 = \frac{m_1 d_2}{m_2 b_2}, \quad p_7 = \frac{m_3}{m_1}. \] (4.66)

The simplified model containing only seven parameters can be stated as follows:
\[
\begin{align*}
\frac{dx_1}{dt} &= -x_1 x_2 + p_1 x_2 + x_3 + x_4, \\
\frac{dx_2}{dt} &= x_2 (x_1 - p_2 x_3 - 1), \quad (4.67) \\
\frac{dx_3}{dt} &= x_3 (p_3 x_2 - p_4 x_4 - p_5), \\
\frac{dx_4}{dt} &= x_4 (p_6 x_3 - p_7),
\end{align*}
\]
with its Jacobian matrix
\[
V = \begin{pmatrix}
-x_2^{(i)} & p_1 - x_1^{(i)} & 1 & 1 \\
x_1^{(i)} - p_2 x_3^{(i)} - 1 & -p_2 x_2^{(i)} & 0 \\
0 & p_3 x_3^{(i)} - p_4 x_4^{(i)} - p_5 - p_4 x_3^{(i)} & p_6 x_3^{(i)} - p_7 \\
0 & 0 & 0 & 0
\end{pmatrix}, \quad (4.68)
\]
which we have evaluated at the equilibrium \( E_i \equiv (x_1^{(i)}, x_2^{(i)}, x_3^{(i)}, x_4^{(i)}). \)

The model (4.67) admits five such feasible equilibria. The first three of these are boundary equilibria, namely: the origin \( E_0 = (0, 0, 0, 0) \), the point \( E_1 \equiv (x_1^{(1)}, x_2^{(1)}, 0, 0) \) with arbitrary values of \( x_1^{(1)}, x_2^{(1)} \), an equilibrium which however is very sensitive to environmental fluctuations, since it exists only for \( p_1 = 1 \), and \( E_2 = (x_1^{(2)}, 0, 0, 0) \) also with \( x_1^{(2)} \) arbitrary. All of them are always feasible.

We then find another boundary equilibrium
\[
E_3 \equiv \left( \frac{p_1 p_2 p_5 - p_3}{p_2 p_5 - p_3}, \frac{p_5 p_1 p_5 - p_5}{p_3}, \frac{p_1 p_5 - p_5}{p_3} \right), \quad (4.69)
\]
which is feasible if all its components are nonnegative, i.e., for either of
\[
p_1 < 1 < \frac{p_3}{p_2 p_5}, \quad p_1 > 1 > \frac{p_3}{p_2 p_5} \quad (70)
\]
conditions that also imply \( x_1^{(3)} \geq 0 \). Thus (4.70) are necessary and sufficient for the feasibility of \( E_3 \).

The inner equilibrium is the most important for the sustainability of the whole ecosystem. Let us define \( \alpha \equiv (p_2 p_5 - p_3) r_7 - (p_1 - 1) p_5 p_6 \) and \( \delta \equiv (p_1 - 1) p_4 p_6 - p_2 p_4 p_7 + p_3 p_6 \). Then the equilibrium is
\[
E_4 \equiv \left( \frac{p_2 p_7 + p_6}{\delta}, \frac{p_5 p_6 - p_4 p_7}{\delta}, \frac{p_7}{\delta} \right), \quad (4.71)
\]
The feasibility conditions for $E_4$ require that $x_2^{(4)} \geq 0$, $x_4^{(4)} \geq 0$, as the remaining components are obviously nonnegative. These conditions reduce to either
\[ \alpha \geq 0, \quad \delta \geq 0, \quad p_5p_6 \geq p_4p_7 \tag{4.72} \]
or
\[ \alpha \leq 0, \quad \delta \leq 0, \quad p_5p_6 \leq p_4p_7. \tag{4.73} \]

Model properties

To establish the behavior of the system near each equilibrium point, we need to determine the signs of the eigenvalues of the Jacobian (4.68).

The first equilibrium $E_0$ has one zero eigenvalue $\lambda_1 = 0$, and the other ones negative, $\lambda_2 = -1, \lambda_3 = -p_5$ and $\lambda_4 = -p_7$. The origin is thus a stable equilibrium, neutral along the $x_1$-axis. Also, for $E_2$ there is a vanishing eigenvalue, $\lambda_1 = 0$, the other ones being $\lambda_2 = x_1^{(2)} - 1, \lambda_3 = -p_3$, and $\lambda_4 = -p_7$. Thus, if $\dot{x}_1^{(2)} \leq 1$, the equilibrium is stable, else it is unstable.

The first eigenvalue of $E_3$ is $p_0x_3^{(3)} - p_7$. The signs of the other ones can be obtained from the Routh–Hurwitz conditions applied to the characteristic equation. They give $x_2^{(3)}x_3^{(3)}(p_2p_3x_2^{(3)} + 1) > 0$, which is obviously true, and $p_3x_2^{(3)}x_3^{(3)}(p_2x_2^{(3)} - 1) > 0$. Thus $E_3$ is stable if and only if the first eigenvalue is negative, i.e., $p_0x_3^{(3)} < p_7$ and the further condition $p_2p_3 > p_3$ is satisfied, which falsifies the first equation in (4.70). Hence feasibility must be given by the second condition (4.70), which yields $p_1 > 1$.

Let $N(\lambda) \equiv x_2^{(4)}[\lambda^2(p_1 - x_1^{(4)}) + \lambda p_3x_3^{(4)} + p_4p_6x_3^{(4)}x_4^{(4)}(1 + p_1 - x_1^{(4)})]$ and $D(\lambda) \equiv x_3^{(4)} + \lambda$. The root-finding problem for the characteristic equation at $E_4$ can be restated as the intersection of the two curves

\[
K(\lambda) = \lambda^3 + \lambda(p_4p_6x_3^{(4)}x_4^{(4)} + p_2p_3x_2^{(4)}x_3^{(4)}), \quad R(\lambda) \equiv \frac{N(\lambda)}{D(\lambda)}.
\]

Now $K$ has only a real root at the origin and $K'(\lambda) > 0$ for every $\lambda$, so that $K(\lambda) \sim \lambda^3$ as $\lambda \to \infty$. Instead, $R(\lambda) \sim x_2^{(4)}(p_1 - x_1^{(4)})\lambda + p_3x_2^{(4)}x_3^{(4)}$, and also as $\lambda \to \infty$. The function $R(\lambda)$ has a vertical asymptote located at the zero $-x_2^{(4)}$ of $D(\lambda)$, and two zeros at the roots of $N(\lambda)$:

\[
\lambda_{\pm} = \frac{-p_3x_3^{(4)} \pm \sqrt{p_3^2(x_3^{(4)})^2 - 4(p_1 - x_1^{(4)})p_3p_6x_3^{(4)}x_4^{(4)}(1 + p_1 - x_1^{(4)})}}{2(p_1 - x_1^{(4)})}.
\]

Now it is easy to verify that $\lambda_{\pm} < 0$ if $p_1 > x_1^{(4)}$ and $1 + p_1 - x_1^{(4)} > 0$. On the other hand, for $p_1 < x_1^{(4)}$ and $1 + p_1 - x_1^{(4)} > 0$, $\lambda_+ < 0 < \lambda_-$. Finally, for $p_1 < x_1^{(4)}$ and $1 + p_1 - x_1^{(4)} < 0$, $\lambda_{\pm} > 0$. Also, notice that $R(0) = 1 + p_1 - x_1^{(4)} \equiv p_1 - p_2x_3^{(4)}$. 


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From this analysis, there is always an intersection between \( K \) and \( R \),
\[ K(\lambda^*) = R(\lambda^*) \text{ occurring for } \lambda^* > 0, \text{ unless } p_1 < x_1^{(4)} \text{ and } 1 + p_1 - x_1^{(4)} < 0. \]
We have thus determined easily verifiable sufficient instability conditions for \( E_4 \). The necessary and sufficient conditions for stability are instead given by the following three Routh–Hurwitz conditions:
\[ p_6(p_4 + p_1 p_4) < p_4(p_2 p_7 + 1), \]
\[ p_6 x_4^{(4)}(x_2^{(4)} - p_4 x_3^{(4)}) + (1 - p_2)[p_3 x_3^{(4)} + x_2^{(4)}(p_1 - x_1^{(4)})] > 0. \]

Observe further that letting
\[ A = x_2^{(4)} > 0, \quad B = p_1 - x_1^{(4)} = p_1 - p_2 x_3^{(4)} - 1, \]
\[ D = x_3^{(4)}, \quad F = \frac{1}{p_4}[p_6 p_3 x_2^{(4)} - p_5 p_6], \]
the characteristic equation can be written as
\[ \varphi(\lambda) = \sum_{i=0}^{4} a_i \lambda^i, \quad a_4 = 1, \quad m_3 = x_2^{(4)}, \quad m_2 = -AB + p_2 p_3 A D + p_4 D F, \]
\[ m_1 = -p_3 A D + p_2 p_3 A^2 D + p_4 A D F, \quad a_0 = -ADF(p_3 + p_4 B). \]

Since \( \varphi \) is a quartic, \( \varphi(\lambda) \to +\infty \text{ as } \lambda \to \pm \infty \). Its inflection points \( \varphi''(\lambda_\pm) = 0 \) are real if \( \Delta \equiv 9m_3^2 - 24a_4 > 0 \), in which case it is easily seen
that they lie in the negative left half-plane, as their abscissae are
\[ \lambda_\pm = \frac{1}{12}(-3m_3 \pm \sqrt{\Delta}) \leq 0. \]
(4.75)

In such a case,
\[ a_0 = \varphi(0) > 0, \quad m_1 = \varphi'(0) > 0 \]
(4.76)
are sufficient conditions for stability, ensuring that no positive eigenvalue exists. For \( \Delta < 0 \) instead, no inflection point exists, but again satisfying both (4.76) ensures once more the stability of \( E_4 \).

Results and discussion

The equilibrium \( E_4 \) can also be investigated numerically, to find features that are analytically harder to show. Indeed for the parameters, \( p_1 = 1.84 \) (Figure 4.2) and \( p_1 = 1.89 \) (Figure 4.3), \( p_2 = 0.6, p_3 = 0.4, p_4 = 4.25, p_5 = 0.9, p_6 = 0.555, p_7 = 0.9 \) exhibit limit cycles. We then numerically try to modify the parameters one at a time to investigate the effects of this change.
Changes toward smaller values of \( p_1 \) are generally reflected in oscillations of smaller amplitude around a smaller reference value. The value \( p_1 = 0.5 \) brings a collapse of the limit cycle first toward the corresponding underlying equilibrium value \( E_4 \), then a further decrease of \( p_1 \) leads to the equilibrium.
FIGURE 4.2: Persistent oscillations around the inner equilibrium $E_4$ shown in standard and semilogarithmic plots.

$E_3$ where only one population is wiped out, and then finally induces a further change also of this other equilibrium to the equilibrium $E_2$. Changes toward higher values of $p_2$ damp out the oscillations so as to get back to $E_4$. Smaller values instead lead one of the species to grow toward very high values while still oscillating. Increasing $p_7$ instead gives us again the stable equilibrium $E_3$.

Decreasing $p_1$ from the reference value produces limit cycles, again around lower values of the equilibrium, and the smaller the value of $p_3$, the lower the equilibrium is. Increasing it instead to $p_3 = 1$, the oscillations disappear and at least two trophic levels prevail.

The parameters $p_4$ and $p_5$ finally affect much less the model behavior, although a bifurcation value at $p_5 = 8.33122$ exists, this time from the reference values $p_1 = 1.86$, $p_2 = 0.6$, $p_3 = 0.415$, $p_4 = 4.25$, $p_5 = 0.555$, $p_6 = 0.9$. Also from these latter values a bifurcation value $p_6 = 0.807$ is found driving the system back to $E_3$.

In summary, the results obtained are meaningful. Indeed, the neutral stability of the origin is easily explained since an empty closed system “by definition” remains empty as long as migrations into it are not allowed. At $E_2$ the ecosystem is lifeless and stable at an arbitrary level $x_1^{(2)}$. This is possible, however, only if the amount of the matter is below a threshold level. Thus the nutrients need to be at a minimum level, to avoid collapse of the plants and eventually of the ecosystem as a whole. An equilibrium environment is shown to exist, in which plants and herbivores can coexist in absence of carnivores, by the range of parameters leading to a stable $E_3$, namely, if $p_1 > 1$.
FIGURE 4.3: Change in the oscillations around the inner equilibrium $E_4$ due to a change in $p_1$, the cycles become wider, around a higher reference value; note that the vertical scale differs from the one in Figure 4.2.

The point where the whole ecosystem thrives is $E_4$. We have found conditions on its parameters ensuring feasibility, (4.72) or (4.73), necessary and sufficient stability conditions (4.74), and easier to verify sufficient conditions (4.76). However, the numerical simulations show that when $E_4$ is unstable, the model first exhibits sustained oscillations around this equilibrium and then trajectories may collapse into one of the former equilibria. When the dynamics is forced toward boundary equilibria, some of the populations disappear; in biological terms, we have loss of biodiversity. The ultimate failure of the system is thus represented by $E_1$, i.e., disappearance of life in some or all of its forms. This may very well be provoked by tampering with the model parameters by external factors, which may be due to “natural” causes, as weather, sunlight, and so on, or man-made.

Although this study is far too elementary to draw any serious forecasts on the global environment, it nevertheless shows that sustained oscillations in the ecosystem are feasible and compatible with human activities, provided the latter do not induce too drastic changes in the parameters. A simple model like the one presented here thus shows that it is very important to assess our present stand, to understand whether the changes nowadays occurring on a worldwide scale are going to be permanent or are only part of a temporary and thus still reversible process.
4.6 More about chaos

In the previous sections, we have been dealing mostly with either a system’s relaxation to a stable steady state or with periodic solutions due to a stable limit cycle. In a time-continuous population community with the number of species more than two, however, there can be a more complicated and more exotic type of dynamics which is known as (deterministic) chaos.

As an instructive example, following the work by Hastings and Powell (1991), let us consider a simple food web (or, rather, a food chain) that includes three trophic levels, i.e., a prey, its predator, and a top predator:

\[
\begin{align*}
\frac{du}{dt} &= u(1 - u) - \frac{a_1 uv}{1 + b_1 u}, \\
\frac{dv}{dt} &= \frac{a_1 uv}{1 + b_1 u} - \frac{a_2 vw}{1 + b_2 v} - d_1 v, \\
\frac{dw}{dt} &= \frac{a_2 vw}{1 + b_2 v} - d_2 w
\end{align*}
\] (4.77)

(in dimensionless variables), where \( u, v, \) and \( w \) are the densities of prey, predator, and top predator, respectively, and \( a_{1,2}, b_{1,2} \) and \( d_{1,2} \) are parameters.

The system (4.77) is difficult to study analytically, but it is more or less straightforward to reveal its main properties by means of numerical simulations. A problem arising on this way is that it depends on as many as six parameters. Therefore, a comprehensive study would require thousands of simulation runs accomplished for different parameter sets. Alternatively, however, since our goal here is to give an example of chaotic dynamics rather than to fulfill a detailed study of the system properties, we can fix most of the parameters at a certain hypothetical value and study the system’s behavior as a response to variations in the remaining “controlling parameter.”

It is well-known (e.g., see Murdoch and Oaten, 1975, and also Section 10.1 of Chapter 10) that stability of a prey–predator system with Holling type II response of predator essentially depends on the half-saturation constant, and hence on parameter \( b_1 \). We can thus assume that \( b_1 \) is likely to control the properties of the tritrophic system (4.77) as well and choose it as the controlling parameter.

Numerical simulations show that for small values of \( b_1 \) the system normally possesses a stable steady state. With an increase in \( b_1 \), its stability is eventually broken through the Hopf bifurcation and a stable limit cycle appears; see the left-hand panel of Figure 4.4. With a further increase in \( b_1 \), the system undergoes a series of period-doubling bifurcations and the shape of the limit cycle becomes more and more complicated (cf. Figure 4.4b) until it turns into a strangely looking attractor that no longer bears any resemblance to a limit cycle; see Figure 4.5. Correspondingly, the oscillations of the population densities versus time become irregular, too.
A visually different shape of attractor, however, is surely not enough to conclude that the system dynamics has acquired any qualitatively new features. Indeed, theoretically speaking, the attractor shown in Figure 4.5 could still be a multiple cycle. What actually distinguishes chaos from a multiperiodic dynamics, however complicated the latter may be, is a different response to variations of the initial conditions. While a (multi)periodic limit cycle is stable with regards to such variations, a fundamental property of chaotic dynamics is a special type of sensitivity so that distance $d$ between any two initially closed trajectories grows with time exponentially. This property is usually quantified by the dominant Lyapunov exponent:

$$
\lambda_D = \lim_{d(0) \to 0, t \to \infty} \frac{1}{t} \log \left[ \frac{d(t)}{d(0)} \right]
$$

(4.78)

(cf. Nayfeh and Balachandran, 1995).

It is very difficult to prove chaotic behavior analytically, but it is relatively easy to demonstrate it numerically. Figure 4.6 shows the density of the top predator versus time calculated for two very close sets of initial conditions; $u_1(0) = u_2(0)$, $v_1(0) = v_2(0)$ and $w_1(0) - w_2(0) = 0.001$. It is readily seen that, while at an earlier stage of the system dynamics the trajectories remain close, the discrepancy between the solutions grows steadily so that for $t \approx 2000$ it becomes on the same order as that of the solutions themselves. The corresponding value of $\lambda_D$ can then be roughly estimated as 0.01.

Runaways of system trajectories due to the system’s sensitivity to the initial conditions make intermediate-term prediction of the system state impossible, and that may have profound implications. This is one of the reasons why the issue of chaos – in particular, in population dynamics and ecology – has been attracting a lot of attention over the last three decades; e.g., see May (1974),

Another example of a population dynamics model exhibiting chaos is given by a Lotka–Volterra-type system with one predator and two preys (cf. Gilpin, 1979; Arneodo et al., 1980):

\[
\begin{align*}
\frac{du_1}{dt} &= (1 - r_{11}u_1 - r_{12}u_2 - r_{13}v)u_1, \\
\frac{du_2}{dt} &= (1 - r_{21}u_1 - r_{22}u_2 - r_{23}v)u_2, \\
\frac{dv}{dt} &= (-1 + r_{31}u_1 + r_{32}u_2 - r_{33}v) v,
\end{align*}
\]  

(4.79)

where \(u_1, u_2\), and \(v\) are the (dimensionless) densities of prey 1, prey 2, and predator, respectively, and all coefficients \(r_{ij}\) are assumed to be nonnegative. Having chosen the predation rate \(r_{31}\) as a controlling parameter and other parameters fixed at some hypothetical values, Schaffer and Kot (1986) showed that the system (4.79) exhibits onset of chaos through the period-doubling bifurcation scenario when \(r_{31}\) increases to a certain critical value.

A further discussion of prerequisites and implications of chaos in “multi-
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FIGURE 4.6: (See color insert.) Top predator density vs time: there is a growing discrepancy between two initially close trajectories \( (w_1(0) - w_2(0) = 0.001) \) in the chaotic regime of system (4.77). Parameters are the same as in Figure 4.5.

species systems\(^1\) can be found in Rinaldi and Feo (1999).

Note that, when looking for chaotic dynamics in a time-continuous model described by a system of ordinary differential equations, the condition that the number of interacting species must not be less than three is essential. Indeed, the solution uniqueness theorem, which is valid in a general case of \( n \) species system (2.1) under some unrestrictive and biologically reasonable assumptions about the properties of the interaction terms \( F_i \), obviously rules out a possibility of trajectories intersection. In the case of two-species system, where the phase space is reduced to a plane, it thus excludes existence of an attractor more complicated than a limit cycle. The situation is thus very much different from time-discrete systems where chaos can occur even in a single species model; see Section 3.4.

It should be mentioned here that, although there are many theoretical arguments showing that chaos should be a common phenomenon in population dynamics (and chaos has indeed been observed for at least two different laboratory populations; see Costantino et al., 1997; Becks et al., 2005), a conclusive evidence of chaos in ecological data is still lacking. This disagreement between theory and available data has been a subject of continuing intensive discussion and controversy; the two reasons that are usually mentioned as a

\(^1\)In this context, systems with the number of species greater than two.
probable explanation why chaos is not immediately seen in ecological dynamics are the impact of noise and an insufficient length of ecological time series. The latter also raises a more fundamental concern that chaos in ecosystems dynamics cannot be seen at all (even if it may be an intrinsic property of a given system) because of the impact of transients (Hastings, 2001, 2004). As a whole, however, the question of whether mother nature is chaotic and if not, why not (cf. Berryman and Millstein, 1989; Hastings et al., 1993) yet remains largely open.

4.7 Age-dependent populations

This is probably the most theoretical section of the whole book. We start by illustrating a simple example in some of its fine details to describe the analytical technique used. The purpose of the later sections is to present the well-known contraction mapping principle on a nontrivial, rather sophisticated application. We have already encountered fixed points in the earlier chapter on a single discrete population. In this one the continuous case is discussed, allowing interactions among two populations. Excellent and thorough references on this topic for the interested reader are the books by Cushing (1998), Iannelli (1995), and Webb (1985).

Lotka (1956) and von Foerster (1959) in population theory and McKendrick (1926) in the context of epidemics introduced an age-dependent description of the population in which instead of the total population size as a function of time, they considered as the dependent variable a density function, which gives at every instant the number of individuals in the same age group, technically called a cohort, and looks at its evolution in time. The model is formulated as a hyperbolic partial differential equation of the first order.

Leslie (1945; 1948) considered a discrete system, in which each component of a vector describes the cohort in a certain age interval, while the evolution of the population is described by a matrix, the Leslie matrix, whose entries contain the mortality of each cohort.

We will consider only the continuous-time version, and describe now how to obtain the governing equations.

If \( u(a, T) \) represents the density of the population, the total population is in this case provided by summing the density over all possible ages. Technically, two possibilities arise, namely, to assume that there is a maximum allowed age \( A \), so that \( u(a, T) \equiv 0 \) for all \( a \geq A \) at all times \( T \), or simply to allow all possible ages up to \( \infty \), in which case mathematically we need to assume the maternity to have compact support, so as to be able to meaningfully define the integral that follows. This second possibility will be used later in Section 6.3.
Thus

\[ U(T) = \int_0^A u(a, T) \, da. \quad (4.80) \]

The above equation represents then the relationship of this model with the previous ones, which are independent of age.

If we now let a time \( \delta T \) go by, we find that all individuals will be aged \( a + \delta T \), apart from those who in the meantime passed away. This remark shows that the differentials of age and time are the same, \( dT \equiv da \), or, integrating, \( a = T - T_0 \), where \( T_0 \) denotes the “birthdate” of the cohort. Thus, introducing the individual mortality rate \( m(a, T) \), we find the balance equation:

\begin{align*}
\frac{u(a + \delta T, T + \delta T) - u(a, T)}{\delta T} &\approx \frac{u(a + \delta T, T + \delta T) - u(a + \delta T, T)}{\delta T} \\
&+ \frac{u(a + \delta T, T) - u(a, T)}{\delta T} = m(a, T)u(a, T). \quad (4.81)
\end{align*}

In general, the mortality function is independent of time, or better, it depends on time implicitly via the density \( u(a, T) \) itself, or rather said, through the population \( m = m(a, U(T)) \). As a first approach, however, for the next considerations, let us take it to be only age dependent, namely, \( m \equiv m(a) \).

Letting \( \delta T \rightarrow 0 \), we find the basic equation governing the age-dependent population models:

\[ \frac{\partial u(a, T)}{\partial T} + \frac{\partial u(a, T)}{\partial a} + m(a)u(a, T) = 0. \quad (4.82) \]

The model is still incomplete, as an initial condition is needed, which in this case is provided by an initial density \( u(a, 0) = \phi(a) \), but above all because we need to describe the process by which new individuals are added to the population. To this end, let us introduce the maternity function \( \beta(a, T) \equiv \beta(a, U(T)) \), again implicitly a function of time via the population size. It represents the reproduction of the cohort in the population aged \( a \) at time \( T \).

Now the newborns can be calculated by summing the contributions of each individual reproduction function over all possible ages. The newborns \( B(T) \) at time \( T \) are thus given by

\[ B(T) = \int_0^A \beta(a, U(T))u(a, T) \, da. \quad (4.83) \]

The model thus formulated is an initial and boundary value problem, in the sense that the “boundary” is expressed by the fact that age can never be negative, and the new individuals coming into the population are the newborns of age \( 0 \); they are accounted for by the boundary term \( (4.83) \).

The characteristics of \( (4.82) \) are straight lines, as \( dT = da \), which in parametric form are written \( T = s + T_0, \ a = s \). Notice that the tangent unit
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vector to these lines is \( t = \frac{1}{\sqrt{2}}(1,1)^T \). Remembering the meanings of age and
time differentials, observe that

\[
ds^2 = dT^2 + da^2, \quad ds = \sqrt{2}da.
\] (4.84)

Consider \( \dot{u}(s) \equiv u(s, s + T_0) \), i.e., the density along the characteristics. In
general, the partial differential operator on the left of (4.82) represents the
derivative of the restriction of the function \( u \) to the characteristics,

\[
Du(s, T) \equiv \lim_{\delta a \to 0} \frac{u(s + \delta a, T + \delta a) - u(s, T)}{\delta a} = \frac{d\dot{u}(s)}{ds}.
\] (4.85)

Notice on the other hand that the partial differential operator on the left-
hand side of (4.82) is related to the directional derivative of \( u \) along the
characteristics \( D_\nu u \). Indeed, since

\[
Du = \frac{\partial u}{\partial T} + \frac{\partial u}{\partial a} = \nabla u \cdot (1,1)^T = \sqrt{2} \nabla u \cdot t \equiv \sqrt{2} D_\nu u.
\] (4.86)

Equations (4.86) and (4.85) are related, representing the same concept, the
difference being the use of the arclength.

To better motivate the use of the latter, let us ignore dependence on time
in the mortality function, which is then only dependent on age. Thus \( u(a, T) \)
will be subject to \( m(a) \), and \( u(a + \delta a, T + \delta T) \) will be subject to \( m(a +
\delta T) = m(a + \delta a) \). But letting \( \delta T \) go by, from \( u(a_0, t_0) = u(s_0) \) measured
along one of the characteristics, subject to the mortality \( m(a_0) \) we arrive
at \( u(a_0 + \delta a, T_0 + \delta T) = u(s_0 + \delta s) \). Recalling (4.84), the density at the
point \( (a_0 + \delta a, T_0 + \delta T) \) would be subject to the mortality \( m(a_0 + \sqrt{2}\delta a) \)
rather than to the correct one \( m(a_0 + \delta a) \). This consideration tells us that
along the characteristics we need to use as a parameter the arclength, i.e., the
normalized \( s \) via the factor \( \sqrt{2} \), namely, \( \sigma = \frac{1}{\sqrt{2}} s \).

With this position, we can then describe the evolution of the density \( u \) as follows:

\[
\left[ u \left( \frac{s + \delta s}{\sqrt{2}} \right) - u \left( \frac{s}{\sqrt{2}} \right) \right] \frac{\sqrt{2}}{\delta s} = -m \left( \frac{s}{\sqrt{2}} \right) u \left( \frac{s}{\sqrt{2}} \right).
\]

Upon taking the limit as \( \delta \sigma \to 0 \),

\[
\frac{du}{d\sigma} (\sigma) = m(\sigma) u(\sigma), \quad \frac{du}{u} = m(\sigma) d\sigma,
\]

and integration setting \( s = \sqrt{2} \alpha \) then finally gives

\[
\ln u(\sigma) - \ln u(0) = -\int_0^\sigma m(\alpha) d\alpha, \quad u(\sigma) = u(0) \exp \left( -\int_0^\sigma m(\alpha) d\alpha \right).
\]

This is the process known as integration along the characteristics of the first
order partial differential equation; for a geometric interpretation see Zach-
manoglou and Thoe (1976).
We now need to distinguish two cases. When the characteristic meets the initial times, i.e., $T_0 > 0$, notice that when we start counting time the cohort is already aged $a$. In such a case, the value of $u(s)$ is provided by the value of the initial distribution, $\psi(a)$, so that $\tilde{u}(0) = u(a, 0) = \psi(a)$. For $T_0 < 0$, instead, when $T_0$ denotes the birthtime of the cohort, $\tilde{u}(0) = u(0, T_0) = B(T_0)$, and the latter is determined from (4.83). Thus, for $a > T$ and $a < T$, we respectively have

$$u(a, T) = \psi(a - T) \exp \left[- \int_0^{a-T} m(\alpha) d\alpha \right],$$

(4.87)

$$u(a, T) = B(T - a) \exp \left[- \int_0^{T-a} m(\alpha) d\alpha \right].$$

(4.88)

Upon substitution into the population equation, we respectively find for $\xi = a - T$ and $\eta = T - a$,

$$U(T) = \int_0^T u(a, T) da + \int_0^T u(a, T) da$$

$$= \int_0^{A-T} u(\xi, T) d\xi + \int_0^T u(a, T) da.$$

The problem is then finally completely described by the pair of integral equations (4.83) and (4.89):

$$U(T) = \int_0^{A-T} \psi(\xi) \exp \left(- \int_0^\xi m(\alpha) d\alpha \right) d\xi$$

$$+ \int_0^T B(\eta) \exp \left(- \int_0^\eta m(\alpha) d\alpha \right) d\eta.$$ 

By instead multiplying by the maternity function (4.87) and using again (4.83), with the very same steps we find

$$B(T) = \int_0^{A-T} \beta(\xi, U(T))u(\xi, T) d\xi + \int_0^T \beta(a, U(T))u(a, T) da,$$ 

(4.90)

which leads to the single integral equation

$$B(T) = \int_0^{A-T} \psi(\xi)\beta(\xi, U(T)) \exp \left(- \int_0^\xi m(\alpha) d\alpha \right) d\xi$$

$$+ \int_0^T B(\eta)\beta(T + \eta, U(T)) \exp \left(- \int_0^\eta m(\alpha) d\alpha \right) d\eta.$$ 

(4.91)

It is also possible to introduce survival probabilities and relate this equation to the renewal equation of probability theory; for details see Cushing (1998).
4.7.1 Prey–predator, age-dependent populations

Let the nonnegative densities for the predator and prey be denoted by \( v(a, T) \) and \( u(a, T) \), assumed to have compact support with respect to age at every time. Then the populations are easily found by integration:

\[
U(T) = \int_0^\infty u(a, T) \, da, \quad V(T) = \int_0^\infty v(a, T) \, da.
\]

(4.92)

The growth of each population would obey (4.82), in the absence of the other one, with \( \mu \) and \( \nu \) representing the mortality functions for each species. The mortality function should in general be higher for young individuals, then decreasing to a minimum at some intermediate age, and finally increasing steadily as the individual becomes older.

To describe mutual relations, an extra mortality term has to be added to the equation of the prey. The “reward” that the predators obtain is in general assumed to be new population members, i.e., it should be accounted for in the newborn term. With respect to the classical age-independent prey–predator models, we replace here the proportionality constants in the interaction terms by functions of age. This allows us to distinguish encounters of a particular age group within one population and the individuals of the other species, considered as a whole:

\[
\begin{align*}
\frac{\partial u}{\partial a} + \frac{\partial u}{\partial t} + \mu(a, U) u + \alpha_0(a) u V &= 0, \\
\frac{\partial v}{\partial a} + \frac{\partial v}{\partial t} + \nu(a, V) v &= 0.
\end{align*}
\]

(4.93)

Letting \( \beta_u \) and \( \beta_v \) represent the maternity functions for each species, the birth rates are then given by

\[
\begin{align*}
B_u(T) &= u(0, T) = \int_0^\infty \beta_u(a, U(T)) u(a, T) \, da, \\
B_v(T) &= v(0, T) = \int_0^\infty [\beta_v(a, V(T)) + \gamma_0(a) U(T)] v(a, T) \, da.
\end{align*}
\]

(4.94)

Here \( \alpha_0(a) \) expresses the hunting function on a prey aged \( a \), while \( \gamma_0(a) \) is the return predators aged \( a \) get from hunting. Since young and old individuals have less defenses, \( \alpha_0(a) \) could be modeled with a minimum at some maturity age \( M \) and then tending asymptotically to a limiting value, while \( \gamma_0(a) \), since it represents the hunting skills of predators, should have a maximum at some intermediate age.

Suitable initial conditions are as follows:

\[
\begin{align*}
u(0, a) &= g(a), & u(0, a) &= \eta(a).
\end{align*}
\]

(4.95)

For mathematical tractability, as functions of \( U \) and \( V \), all the mortalities and maternities are assumed to have continuous partial derivatives and together with them to be bounded functions. Instead of pursuing analytically this model, we will now instead formulate and investigate a more general one.
4.7.2 More about age-dependent populations

We extend the model formulated in the previous section by considering an environment in which several population are present and interact, accounting for species that either live in symbiosis or compete for common resources, or such that some species are hunted by others. The approach follows closely Gurtin and McCamy (1974), and the results are related to those obtained via nonlinear semigroups in Webb (1985). Here we also allow nonlinear interactions, by letting both the maternity and mortality functions for each species be influenced by all the other species. The necessary assumption will of course be that these modified mortalities retain positive signs so that they will never generate newborns of some positive age. The modifications increase or decrease each species density according to the type of interaction with other species. Similar remarks hold for the maternities as well, which are always positive, i.e., never generating a negative number of newborns.

Let \( x_i(a, t), i = 1, ..., N \) represent the densities of the \( N \) populations, assumed to be of compact support with respect to age at any given time \( t \). This ensures that the population sizes are well defined:

\[
X_i(t) = \int_0^\infty x_i(a, t) \, da.
\]

Letting \( X \equiv (X_1, ..., X_N)^T \), the birth rates can then be defined as

\[
B_i(t) = x_i(0, t) = \int_0^\infty \beta_i(a, X(t)) \, x_i(a, t) \, da.
\]

Here \( \beta_i \) represents the maternity function for the \( i \)th species. The model then becomes

\[
\frac{\partial x_i}{\partial t} + \frac{\partial x_i}{\partial a} + \mu_i(a, X(t)) \, x_i(a, t) = 0,
\]

with initial conditions

\[
x_i(a, 0) = \phi_i(a).
\]

The maternity and mortality functions are assumed to be continuous and nonnegative on \( \mathbb{R}^+ \times (\mathbb{R}^+)^N \). Moreover, for every multiindex \( \alpha \), \( 0 \leq |\alpha| \leq P \) for some \( P \), the maternity and mortality are bounded above, as functions of \( X \in (\mathbb{R}^+)^N \), together with their mixed partial derivatives:

\[
\frac{\partial^\alpha \beta_i}{\partial X^\alpha}, \frac{\partial^\alpha \mu_i}{\partial X^\alpha}, \quad i = 1, ..., N.
\]

As functions of age, for the maternity and mortality functions, the considerations of the previous section hold. Thus the following quantities are well
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defined:

\[ \mu^0 = \sup (\mu_i (a, X) : (a, X) \in \Omega; \ i = 1, ..., N) ; \] (4.100)

\[ \mu' = \sup \left( \frac{\partial \mu_i}{\partial X_j} (a, X) : (a, X) \in \Omega; \ i, j = 1, ..., N \right) ; \] (4.101)

\[ \beta^0 = \sup (\beta_i (a, X) : (a, X) \in \Omega; \ i = 1, ..., N) ; \] (4.102)

\[ \beta' = \sup \left( \frac{\partial \beta_i}{\partial X_j} (a, X) : (a, X) \in \Omega; \ i, j = 1, ..., N \right) . \] (4.103)

Moreover, assume

\[ b^* = \sup \{ \beta_i (a, X) : a \geq 0, X_j \geq 0, \ i, j = 1, ..., N \} \] (4.104)

\[ m^* = \inf \{ \mu_i (a, X) : a \geq 0, X_j \geq 0, \ i, j = 1, ..., N \} . \] (4.105)

**Integral reformulation – local existence**

For \( i = 1, ..., N \), let \( x_i \) denote a solution of the problem up to some time \( T > 0 \). Let us then set

\[ \tilde{x}_i (h) = x_i (a^0 + h, t^0 + h) , \quad \tilde{X}_i = X_i (t^0 + h) , \quad \tilde{\mu}_i (h) = \mu_i \left( a^0 + h, \tilde{X} (h) \right) . \]

As it was shown at the beginning of this section, since

\[ \frac{d \tilde{x}_i (h)}{dh} = \frac{\partial x_i}{\partial a} + \frac{\partial x_i}{\partial t} , \]

the model becomes the following system of ordinary differential equations:

\[ \frac{dx_i (h)}{dh} + \tilde{\mu}_i (h) \tilde{x}_i (h) = 0 . \]

Integrating and rephrasing,

\[ \tilde{x}_i (h) = \tilde{x}_i (0) \exp \left( - \int_0^h \tilde{\mu}_i (s) ds \right) , \]

\[ x_i \left( a^0 + h, t^0 + h \right) = x_i \left( a^0, t^0 \right) \exp \left( - \int_0^h \tilde{\mu}_i (s) ds \right) . \]

For \( a \geq t \), choose \( a^0 = a - t \), \( t^0 = 0 \), and for \( a \leq t \), let \( a^0 = 0 \), \( t^0 = t - a \), \( h = a \), to find the densities in terms of the initial and boundary conditions

\[ x_i (a, t) = \phi_i (a - t) \exp \left( - \int_0^t \mu_i \left( a - t + s, X (s) \right) ds \right) \] (4.106)

for \( a \geq t \), and

\[ x_i (a, t) = B_i (t - a) \exp \left( - \int_0^a \mu_i \left( s, X (t - a + s) \right) ds \right) \] (4.107)
for \( a \leq t \).

Let

\[
K_i(\alpha, t, X) = \exp \left( -\int_{t-a}^t \mu_i(\tau + t - \alpha, X(\tau)) \, d\tau \right),
\]

\[
L_i(v, t, X) = \exp \left( -\int_0^t \mu_i(v + \tau, X(\tau)) \, d\tau \right).
\]

Integrating (4.106) and (4.107) gives the total population sizes and repeating the operation on the same formulas multiplied by the maternities provides the total birth rates. Mathematically, then,

\[
X_i(t) = \int_0^t B_i(u) K_i(t - u, t, X) \, du + \int_0^\infty \phi_i(v) L_i(v, t, X) \, dv,
\]

\[
B_i(t) = \int_0^t B_i(u) \beta_i(t - u, X) K_i(t - u, t, X) \, du + \int_0^\infty \phi_i(v) \beta_i(t + v, X) L_i(v, t, X) \, dv.
\]

Thus our model is reduced to the system of integral equations (4.108)–(4.109).

Given instead \( X_i(t) \) and \( B_i(t) \) continuous and positive, satisfying equations (4.108)–(4.109) on \([0, T]\), \( x_i(a, t) \) can be defined on \( \mathbb{R}^+ \times [0, T] \) following (4.96). Then \( x_i(a, t) \) is nonnegative since \( \phi \) and \( \beta \) are; also, \( x_i(0, t) = B_i(t) \) and \( x_i(a, 0) = \phi_i(a) \). Moreover, \( x_i(a, t) \in L_1(\mathbb{R}^+) \) since \( \mu_i, B_i \) and \( X_i \) are continuous and the initial condition belongs to \( L_1(\mathbb{R}^+) \). Thus (4.96) and (4.97) hold, by using the definition of \( x_i(a, t) \) and (4.108)–(4.109). Finally, from the definition of \( x_i(a, t) \), the partial derivatives exist so that (4.98) is satisfied.

Thus the original model is equivalent to the system of Volterra integral equations (4.108)–(4.109).

**Application of the fixed point method to local existence**

A nontrivial application of an important result of functional analysis, the contraction mapping principle, or fixed point method, provides the local existence result. Let us fix once again \( T > 0 \) and define

\[
\mathcal{C}^+[0, T] = \{ f \in \mathcal{C}[0, T] : f \geq 0 \}.
\]

Formally solving the linear uncoupled part of (4.108)–(4.109) we get the birth rates \( B_i \):

\[
B_i(t) = \langle \mathcal{B}_i(T, X)(t) \rangle.
\]
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Here the operator $\mathbf{B} (T, \mathbf{X})$ depends on $\mathbf{X}$. Define $\mathcal{X} (T, \mathbf{X})$ by substituting the $i$th component of $\mathbf{B} (T, \mathbf{X})$ into the right-hand side of the nonlinear part of the system,

$$
(\mathcal{X}_i (T, \mathbf{X}) (t)) = \int_0^t (\mathcal{B}_i (T, \mathbf{X}) (u)) K_i (t - u, t, \mathbf{X}) \, du + \int_0^\infty \phi_i (v) L_i (v, t, \mathbf{X}) \, dv.
$$

Now $\mathcal{B} (T, \mathbf{X})$ and $\mathcal{X} (T, \mathbf{X})$ map the Banach space $(C^+ [0, T])^N$ equipped with the supremum norm $\| \cdot \|_T$ onto itself. To get a solution of (4.108)–(4.109) we need to show that they have a fixed point, i.e., that they are contractions. Let $r > 0$ be given and define

$$
\Phi = \left[ \int_0^\infty \phi_1 (a) \, da, \ldots, \int_0^\infty \phi_N (a) \, da \right],
$$

$$
\Sigma_T = \left\{ f : f \in (C^+ [0, T])^N \to \mathbb{R}^N, \| f - \Phi \|_T < r \right\}.
$$

We now prove that $\mathcal{X} (T, \mathbf{X})$ maps $\Sigma_T$ onto itself and is a contraction.

For the first step consider the strip in $\mathbb{R}^+ \times \mathbb{R}^+$ given by

$$
\Omega = \{ (a, \mathbf{X}) : a \geq 0, X_k \geq 0, \| \mathbf{X} - \Phi \| \leq r, \ k = 1, \ldots, N \}.
$$

Let $\mathbf{X} \in \Omega$. Recalling (4.100) from (4.109), it follows,

$$
(\mathcal{B}_i (T, \mathbf{X}) (t)) \leq \beta^0 \int_0^t (\mathcal{B}_i (T, \mathbf{X}) (u)) \, du + \beta^0 \Phi_i,
$$

and then Gronwall’s inequality gives

$$
(\mathcal{B}_i (T, \mathbf{X}) (t)) \leq \beta^0 \Phi_i \exp (\beta^0 t).
$$

Moreover, because

$$
\sup |L_i (v, t, \mathbf{X}) - 1| \leq \mu^0 T \exp (\mu^0 T),
$$

for $T$ small enough we have

$$
| (\mathcal{X}_i (T, \mathbf{X}) (t)) - \phi_i | \leq \Phi_i \beta^0 T \exp (\beta^0 T) + \Phi_i \mu^0 T \exp (\mu^0 T) \leq r,
$$

and combining the components also $\| \mathcal{X} (T, \mathbf{X}) (t) - \Phi \| \leq r$. Thus $\mathcal{X}_i (T, \mathbf{X})$ maps $\Sigma_T$ onto itself.
To show that $\mathcal{X}(T, X)$ is a contraction, take now $X', X'' \in \Sigma_T$ and estimate as follows:

\[ |X_1 - X_2| \equiv |(X_i(T, X')(t)) - (X_i(T, X'')(t))| \leq \int_0^t |K_i(t-u, t, X') - K_i(t-u, t, X'')| (B_i(T, X') (u)) \, du \]

\[ + \int_0^t |K_i(t-u, t, X'')| |(B_i(T, X') (u)) - (B_i(T, X'') (u))| \, du \]

\[ + \int_0^\infty \phi_i(v) |L_i(v, t, X') - L_i(v, t, X'')| \, dv. \]

Taylor’s formula gives

\[ |L_i(v, t, X') - L_i(v, t, X'')| \leq N \mu |X' - X''|_{\Sigma_T} \exp (2\mu T) \]

\[ |K_i(t-u, t, X') - K_i(t-u, t, X'')| \leq N \mu' |X' - X''|_{\Sigma_T} \exp (2\mu' T). \]

The definition of $B(T, X)$, letting $m_i(t) = B_i(T, X')(t) - B_i(T, X'')(t)$, gives the estimate

\[ |m_i(t)| = \left| \int_0^\infty m_i(u) \beta_i(t-u, X') K_i(t-u, t, X'') \, du \right| + |f_i(t)| \leq \beta^0 \int_0^\infty |m_i(u)| \, du + |f_i(t)|, \]

and from Gronwall’s inequality we conclude that

\[ |m_i(t)| \leq |f_i(t)| + \beta^0 \int_0^t |f_i(u)| \exp \left[\beta^0 (t-u)\right] \, du. \]  

(4.114)

Now

\[ |f_i(t)| \leq \int_0^\infty \phi_i(v) |\beta_i(X') L_i(X') - \beta_i(X'') L_i(X'')| \, dv \]

\[ + \int_0^t (\beta_i(T, X')(v)) |\beta_i(X') K_i(X') - \beta_i(X'') K_i(X'')| \, dv. \]

Using (4.113), the last term can be estimated to give the upper bound:

\[ \Phi_i N \beta^0 T \exp (2\beta^0 T) \left[ |\beta' + \beta^0 \mu' T | \right] |X' - X''|_{\Sigma_T}. \]

Using instead (4.112), an upper bound for the first term in (4.115) is obtained:

\[ \Phi_i N \mu' T \exp (2\mu_0^0 T) \left[ |\beta^0 + \left( \beta^0 T \right) | \right] |X' - X''|_{\Sigma_T}. \]

From (4.114) we have

\[ |m_i(t)| \leq Tk(T) |X' - X''|_{\Sigma_T}, \]  

(4.116)
and for $T$ small enough, the right-hand sides of (4.112), (4.113), and (4.116) are small and combining them with (4.111) we find
\[
|(X(t), X'(t)) - (X(T), X''(t))| \leq T \bar{k}(T) \|X' - X''\|_T < \|X' - X''\|_T.
\]
Thus, as claimed, $X(T, X)$ is a contraction for $T$ small enough.

**Boundedness**

Integrate Equation (4.98) with respect to age,
\[
\frac{dX_i}{dt} = \int_0^\infty \beta_i(a, X)x_i(a, t) \, da - \int_0^\infty \mu_i(a, X)x_i(a, t) \, da. \tag{4.117}
\]
From (4.104)–(4.105) we have $K_i(a, t, X)L_i(v, t, X) \leq \exp(-m^*t)$ and using then (4.106), (4.107), (4.108), (4.117), and Gronwall’s inequality for $0 \leq t \leq T$, the population and density growths are bounded above by
\[
X_i(t) \leq \Phi_t \exp[(b^* - m^*)t], \quad \beta_i(t) \leq b^* \Phi_t \exp[(b^* - m^*)t],
\]
\[
x_i(a, t) \leq b^* \Phi_t \exp[-m^*a + (b^* - m^*)t], \quad a < t,
\]
\[
x_i(a, t) \leq \|\Phi_t\| \exp(-m^*a), \quad a > t.
\]

We now consider the Taylor’s expansions of the maternities and mortalities $\beta_i$ and $\mu_i$ up to order $P$. These expansions hold in the sphere $S(R)$ of radius $R = \max \{\|X(0)\|, \|X^0\|\}$, whose radius $X^0$ will be determined later.

Let $B^0 = \sup \left\{ \left| \gamma_i^{(\alpha)}(a) \right| : a \geq 0, X \in S(R) \right\}$, $\gamma_i^{(\alpha)}(a) = \frac{\partial^{\alpha} \gamma_i}{\partial X^\alpha}(a)$, and $\gamma_i^{(\alpha)}(a) = \frac{\partial^{\alpha} \gamma_i}{\partial X^\alpha}(a, \alpha X)$, where $0 < c < 1$, for $|\alpha| = P$, and $\gamma_i = \beta_i - \mu_i$. Taking the sum over the multiindex $\alpha = (k_1, \ldots, k_N)$, the system then becomes
\[
\frac{dX_i}{dt} = \sum r_{i, \alpha}(t) X_i^{k_1} \cdots X_N^{k_N}, \quad r_{i, \alpha}(t) = \int_0^\infty \frac{\gamma_i^{(\alpha)}(a)}{R^N X_i(t) B^0} \, da.
\]

The main result of this section shows that for (4.117), the unit hypercube $X^0$ is a positive invariant set.

Let $J(i) = \{\alpha = (k_1, \ldots, k_N) : r_{i, \alpha}(t) > 0\}$ and observe that taking the sum over the multiindex $\alpha$, for every $t$ we have
\[
0 < |r_{i, \alpha}(t)| < \frac{1}{R^N}, \quad \sum r_{i, \alpha}(t) < 1.
\]
Define the vectors
\[
(w_{i, \alpha}(X))_j = \delta_{ij} \left( X_i^{k_1} \cdots X_i^{k_{i'-2}} \cdots X_N^{k_N} \right) (1 - X_i) X_i, \quad j = 1, \ldots, N,
\]
starting from $|\alpha| = P$ and then iterating the procedure up to $|\alpha| = 0$. At every such step the coefficients also get modified; for $|\alpha'| \leq |\alpha|$ we have
\[
r_{i, \alpha'}(t) = r_{i, \alpha'}(t) + r_{i, \alpha}(t).
\]
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The solution \( X(t) \) of \( \frac{dX}{dt} = \text{sgn} \left( r^{i,0}(t) \right) w_{i,0}(X) \) satisfies \( X_j(t) = X_j(0) \) for \( j \neq 1 \), and \( X_1(t) \to 0 \) if \( \text{sgn} \left( r^{i,0}(t) \right) < 0 \) while \( X_i(t) \to 1 = X_i^0 \) conversely. Then, in a neighborhood of \( t = 0 \), \( X_i(t) < \max \left( X_i(0), X_i^0 \right) \). We can now replace system (4.117) by

\[
\frac{dX_i}{dt} = \sum w_{i,\alpha}(t) r^{i,\alpha}(t). \tag{4.122}
\]

We finally observe that \( r^{i,0}(t) < 0 \) since this condition is equivalent to

\[
\sum r_{i,\alpha}(t) \leq \sum r_{i,\alpha}(t),
\]

where the first sum is for \( \alpha \in J(i) \), while in the second one \( \alpha \not\in J(i) \), and this condition holds by assumption. Hence, as time flows, the solution of

\[
\frac{dX_i}{dt} = \text{sgn} \left( r^{i,0}(t) \right) w_{i,0}(X) X_i
\]

always tends to zero. We can rewrite our system with \( v \in \mathbb{R}^N \) as

\[
\frac{dX}{dt} = u_1(t) X_1(X) + \ldots + u_k(t) X_N(X) \quad X(0) = X_0, \quad 0 \leq u_i(t) \leq \frac{1}{N}.
\]

Let \( \phi_t(X) \) be its actual solution. Let \( \psi_{it}(x, y) \) be the solution trajectories in the population space of the individual systems,

\[
\frac{dX}{dt} = X_i, \quad X(0) = X_0 \quad i = 1, \ldots, N.
\]

Rewrite the system as

\[
\sum \left( \frac{dX}{dt} - X_t(X) \right) u_i(t) + \left( 1 - \sum u_i(t) \right) \left( \frac{dX}{dt} - 0 \right) = 0, \quad X(0) = X_0.
\]

Then, by the very definition of \( \phi_t \) and \( \psi_{it} \), at every instant

\[
\phi_t(X) = \sum u_i(t) \psi_{it}(X) + \left( 1 - \sum u_i(t) \right) X_0,
\]

thus showing that the trajectories are ultimately confined to the unit hypercube.

Global existence and uniqueness

The result of the previous section allows us to show now that the problem has a unique solution for all time.

Indeed, by the local existence result, given an initial distribution \( \ldots, X_i(0), \ldots \), the solution exists up to time \( T \), where \( T \) depends on the initial data. Boundedness implies then that \( X_i(T) < \max(g_0, X^*) \). Now repeat the argument with \( \ldots, X_i(T), \ldots \) as the new initial condition. Since local uniqueness holds, any two solutions agree on an interval \([0, T)\), and by their continuity necessarily \( T = \infty \).
Stability of equilibrium distributions

We have seen in the previous chapters how to analyze the stability of equilibria of models governed by ordinary differential equations. Here we study the same question for structured models formulated by means of hyperbolic partial differential equations.

The equilibrium distributions of the system are in this case once again the time independent solutions, which thus must be functions dependent only on age, i.e.,

\[ x_i (a, t) = x_i (a), \quad X_i (t) = X_i. \]

The system (4.98) thus simplifies to give

\[ \frac{dx_i}{da} + \mu_i (a, X) x_i (a) = 0 \quad (4.123) \]
\[ X_i = \int_0^{\infty} x_i (a) \, da \quad (4.124) \]
\[ B_i = x_i (0) = \int_0^{\infty} \beta_i (a, X) x_i (a) \, da. \quad (4.125) \]

The formal solution of this system is

\[ x_i (a) = x_i (0) \exp \left( - \int_0^a \mu_i (s, X) \, ds \right) = x_i (0) M_i (a, X), \quad (4.126) \]

where \( M_i (a, X) \) denotes the survival function up to age \( a \) of the \( i \)th population. The total population sizes are obtained by integration over age of (4.126):

\[ X_i = B_i \int_0^{\infty} M_i (a, X) \, da. \quad (4.127) \]

The average number of offsprings of an individual is then

\[ R_i (X) = \int_0^{\infty} \beta_i (a, X) M_i (a, X) \, da, \]

and together with (4.125) and (4.126) we have

\[ R_i (X) = 1, \quad i = 1, \ldots, N, \quad (4.128) \]

and in turn (4.128) implies (4.125), so that the necessary and sufficient condition for existence of the equilibrium age distribution is represented by the fact that each individual in the population has exactly one offspring. At equilibrium, then,

\[ x_i^* (a) = B_i^* M_i (a, X), \quad B_i^* = \frac{1}{\int_0^{\infty} M_i (a, X^*) \, da} X_i^*. \]
We investigate the stability of this equilibrium by considering now the following perturbation. Let

\[
x_i(a,t) = x_i^*(a) + y_i(a,t), \quad X_i(t) = X_i^* + Y_i(t).
\]

From now on, the sums are always meant for \(j, k = 1, \ldots, N\). Letting

\[
K_{ij} = \int_0^\infty V(i,j)(a) x_i^*(a) \, da, \\
Z_i(t) = Y_i(t) \sum_j \int_0^\infty V(i,j)(a) y_i(a,t) \, da \\
+ \sum_{j,k} \int_0^\infty W(i,j,k)(a, X^* + Y q) [x_i^*(a) + y_i(a,t)] \, da, \\
A_i(a,t) = \sum_{j,k} Q(i,j,k)(a, X^* + x Y) Y_j Y_k x_i^* \\
+ \sum_j E(i,j)(a) Y_j y_i(a,t), \\
Q(i,j,k)(a,p) = \frac{\partial^2 \mu_i}{\partial X_j \partial X_k}(a,p), \\
E(i,j)(a) = \frac{\partial \mu_i}{\partial X_j}(a, X^*), \\
V(i,j)(a) = \frac{\partial \beta_i}{\partial X_j}(a, X^*), \\
W(i,j,k)(a,p) = \frac{\partial^2 \beta_i}{\partial X_j \partial X_k}(a,p), \quad 0 < q, z < 1.
\]

From Equations (4.98) and (4.99) it follows that

\[
\frac{\partial y_i}{\partial a} + \frac{\partial y_i}{\partial t} + \mu_i(a, X^*) y_i(a,t) + A_i(a,t)(x_i^* + y_i) = 0, \quad (4.129)
\]

\[
Y_i(t) = \int_0^\infty y_i(a,t) \, da, \quad (4.130)
\]

\[
y_i(0,t) = \int_0^\infty \beta_i(a, X^*) y_i(a,t) \, da + \sum_j Y_j(t) K_{ij} + Z_i(t), \quad (4.131)
\]

\[
\phi_i(a,0) - x_i^*(a) = n_i(a).
\]

Observe that \(A_i(a,t)\) and \(Z_i\) are quantities of second order in \(y_i, Y_i\), and \(W(i,j,k)(a,p)\) and \(Q(i,j,k)(a,p)\) are \(o\left(\|p\|^{-1}\right)\) uniformly as \(a \to 0\). Set
Interacting populations

\( D_i (a) = \mu_i (a, X^*) \). We consider the linearized system

\[
0 = \frac{\partial y_i}{\partial t} + \frac{\partial y_i}{\partial a} + D_i (a) y_i (a, t) + \sum_j E_{ij} (a) x_j^* (a) Y_j (t),
\]

(4.132)

\[
Y_i (t) = \int_0^{\infty} y_i (a, t) da,
\]

\[
y_i (0, t) = \int_0^{\infty} \beta_i (a, X^*) y_i (a, t) da + \sum_j Y_j (t) K_{ij},
\]

and for \( g_i, c \) both complexes look for solutions in the form

\[
y_i (a, t) = g_i (a) e^{ct}, \quad i = 1, ..., N.
\]

Now (4.132) gives

\[
0 = \frac{dg_i}{da} + [c + D_i (a)] g_i (a) + \sum_j E_{ij} (a) x_j^* (a) G_j,
\]

(4.133)

\[
G_i = \int_0^{\infty} g_i (a) da,
\]

\[
g_i (0) = \int_0^{\infty} \beta_i (a, X^*) g_i (a) da + \sum_j G_j K_{ij}.
\]

Letting

\[
M_i^0 (a) = M_i (a, X^*), \quad M_i^0 (s, a) = \frac{M_i^0 (a)}{M_i^0 (s)},
\]

by integration we get the homogeneous system in the \( 2N \) unknowns \( G_i \) and \( g_i (0) \):

\[
0 = g_i (0) \left[ 1 - \int_0^{\infty} \beta_i (a, X^*) \exp (-ca) M_i^0 (a) da \right] - \sum_j G_j K_{ij}
\]

(4.134)

\[
+ \sum_j G_j \int_0^{\int_0^{\infty}} E_{ij} (w) x_j^* (w) \exp - [(a - w) c] M_i^0 (w, a) dwdx,
\]

\[
0 = -g_i (0) \int_0^{\int_0^{\infty}} \exp (-ca) M_i^0 (a) da + G_i
\]

\[
- \sum_j G_j \int_0^{\int_0^{\infty}} E_{ij} (w) x_j^* (w) \exp - [(a - w) c] M_i^0 (w, a) dwdx.
\]

Now the above system gives the criterion to determine whether the equilibrium distribution is stable, namely, if the determinant \( \Delta (c) \) of the system (4.134) has no roots with positive real part, then for suitable \( m^0 \) and initial condition \( \Phi \), the solution as \( t \rightarrow \infty \) satisfies

\[
\| x (t) - X^* \| = O \left( \exp - (m^0 t) \right),
\]

(4.135)

\[
\| x (a, t) - X^* \| = O \left( \exp - (m^0 t) \right).
\]
Letting \( b_i (t) = y_i (0, t) \), the formal solution of Equations (4.129)–(4.131) gives for \( a < t \),

\[
y_i (a, t) = M^{-1}_i (a) b_i (t - a) + \int_{t - a}^{t} M^{-1}_i (z + a - t, a) A_i (z + a - t, z) dz
\]

\[
- \sum_j \int_{t - a}^{t} M^{-1}_i (z + a - t, a) E_{ij} (z + a - t) x^*_j (z + a - t) Y_j (t) dz,
\]

and for \( a > t \),

\[
y_i (a, t) = M^{-1}_i (a - t, a) n_i (a) + \int_0^{t} M^{-1}_i (a - t + s, a) A_i (a - t + s, s) ds
\]

\[
- \sum_j \int_0^{t} M^{-1}_i (a - t + s, s) E_{ij} (a - t + s) x^*_j (a - t + s) Y_j (s) ds.
\]

Setting \( \mathbf{v} (t) = (Y_1 (t), \ldots, Y_N (t), b_1 (t), \ldots, b_N (t))^T \), an age integration then gives the system

\[
\mathbf{H} \mathbf{v} (t) + \int_0^{t} \mathbf{K} (t - s) \mathbf{v} (s) ds = \mathbf{f} (t),
\]

where, for \( i = 1, \ldots, N \),

\[
f_i (t) = \int_0^{t} M^{-1}_i (s, s + t) n_i (s + t) ds + \int_0^{t} \int_0^{t} M^{-1}_i (s, s + t - z) A_i (s, s + t - z) ds dz,
\]

\[
f_{N+i} (t) = Z_i (t) + \int_0^{t} \beta_i (s + t, \mathbf{X}^+) M^{-1}_i (s, s + t) n_i (s + t) ds + \int_0^{t} \int_0^{t} M^{-1}_i (s, s + t - z) A_i (s, z) \beta_i (s + t - z, \mathbf{X}^+) ds dz,
\]

while, for \( i, j = 1, \ldots, N \),

\[
H_{ij} = \delta_{ij},
\]

\[
H_{N+i, j} = -K_j + \delta_{i+N,N+j},
\]

\[
K_{ij} (t) = \int_0^\infty M^{-1}_i (s, s + t) E_{ij} (s) x^*_j (s) ds,
\]

\[
K_{i,N+j} (t) = -\delta_{ij} M^{-1}_i (t),
\]

\[
K_{N+i,j} (t) = \int_0^\infty \beta_i (s + t, \mathbf{X}^+) M^{-1}_i (s, s + t) E_{ij} (s) x^*_j (s) ds,
\]

\[
K_{N+i,N+j} (t) = -\delta_{ij} \beta_i (t, \mathbf{X}^+) M^{-1}_i (t).
\]
Now $M^0 (a)$ has at most an exponential growth; since $E_{ij}$ and the maternity function are bounded above, the Laplace transform of the system of integral equations exists for $Re (p) > -D^* \equiv \inf \{ D_i (a) : a \geq 0, \ i = 1, \ldots, N \}$. Also, the condition $\det \left( H + \hat{K} (p) \right) = 0$ is equivalent to system (4.134), and thus it has no solutions with $Re (p) > 0$. For $p \to \infty$ and $Re (p) > -D^*$, $\det \left( H + \hat{K} (p) \right) \to 1$. A positive constant $m$ exists then such that $\det \left( H + \hat{K} (p) \right)$ does not vanish for $Re (p) > -m$. Hence $H + \hat{K}$ is analytically invertible in $Re (p) > -m$ and in view of $\hat{K}_{ij} (p) \to 0$ as $p \to \infty$, the inverse is of the form $H^{-1} + \hat{J} (p)$, with $\hat{J} (p)$ analytic in $Re (p) > -m$ and $\hat{J}_{ij} (p) \to 0$ as $p \to \infty$ and $\hat{J} (p) = J^0 / p + O \left( p^{-2} \right)$. $J^0$ is an invertible constant matrix so that with the bounds $\| J_{ij} (t) \| \leq \text{const} \ \exp \left( -mt \right)$, we have

$$J (t) = \frac{1}{2\pi} \int_{-\infty}^{\infty} \exp [t (iz - m)] \hat{J} (iz - m) \, dz.$$ 

The solution of system (4.138) is

$$v (t) = H^{-1} f (t) + \int_0^t J (t - s) f (s) \, ds.$$ 

We finally need an estimate of $\| v \|_2$ and $\| y (\cdot, t) \|_1$. Easily,

$$\| f \|_2 \leq C_1 \left[ \| n \|_1 \exp (-tD^*) + \int_0^t \| A_i (\cdot, z) \|_1 \exp [-D^* (t - z)] \, dz + \| Z \|_2 \right].$$

From the convolution properties, then,

$$\| v \|_2 \leq C_2 \left[ \| n \|_1 e^{-tD^*} + \| Z \|_2 + \int_0^t e^{-m(t - s)} \left[ \| A_i (\cdot, s) \|_1 + \| Z \|_2 \right] ds \right],$$

$$\int_0^\infty | y_i (a, t) | \, ds \leq C_3 \| n \|_1 e^{-tD^*} + C_4 \int_0^\infty e^{-D^* (t - z)} \left[ \| Z \|_2 + \| A_i (\cdot, z) \|_1 \right] \, dz.$$ 

For $1 > \epsilon > 0$, we can find a $\delta' (\epsilon) < \epsilon$ such that if $\| v \|_2 < \delta'$, we have $| W (i, j, k) |, | Q (i, j, k) | \leq \epsilon \| v \|_2$. For $\sigma (t) = \| y (\cdot, t) \|_1 + \| v \|_2$, it follows that

$$\| A_i (\cdot, t) \|_1, \| Z \|_2 \leq C_5 \| v \|_2, \| y (\cdot, t) \|_1 + \epsilon \| v \|_2 \leq C_5 \sigma (t),$$

$$\sigma (t) \leq C_6 \left[ \exp (-D^* t) \| n \|_1 + 2\epsilon \int_0^t \exp [-D^* (t - s)] \sigma (s) \, ds \right],$$

and Gronwall’s inequality for $0 \leq t < T$ implies

$$\sigma (t) \leq K \exp (-D^* t + 2K\epsilon t) \| n \|_1 = K \| n \|_1 \exp (-m^0 t),$$

which in turn gives

$$\| Y (0) \|_2 \leq \| v (0) \|_2 \leq \delta, \quad \delta = \min (\delta' (\epsilon), \delta' (\epsilon) / K).$$

If the initial data satisfy $\| n \|_1 < \delta$, iterating on intervals of length $T$ we get $\| v (t) \|_2 < \delta$ for every $t > 0$. Then (4.135) follows from the above estimates using (4.137) and (4.136).
4.7.3 Simulations and brief discussion

The model presented is clearly very difficult to study analytically. Numerical experiments are then needed to assess its behavior. The program uses a finite difference approximation scheme to discretize directly the system (4.92)–(4.95), adapting to the present model the Wendroff implicit approximation scheme (cf. Smith, 1979, p. 155). Given a rectangle in the at phase space, the computational cell to approximate the value of the function at its center is made by its four vertices. Assuming that we know the information at the current time level and the left upper vertex, i.e., the previous age point at the next time, then it becomes an explicit scheme for the upper left vertex, i.e., the next age point at the next time. The problem is that at the next time level, it needs the first “upper left” vertex, which corresponds to the newborns at the next time, and this information analytically comes from the integral of the maternity at the same time level of the newborns, i.e., at the next time. This remark makes the algorithm once more implicit. However, in our implementation we bypassed this point, by simply evaluating the integral (4.83) at the current time step. This corresponds to artificially adding a numerical delay the size of which is the time step.

FIGURE 4.7: The two figures show the persistent oscillating behaviors of the solutions of the model: top, the prey; bottom, the predators.
Figure 4.7 shows results of numerical simulations obtained for parameters chosen in such a way that each species treated independently of the other one evolves toward a stable equilibrium. When the two interfere with each other by the predation mechanism, then the solution trajectory in the phase space shows a limit cycle; see Figure 4.7.

Thus the age-dependent prey–predator model exhibits radically different characteristics than the classical quadratic prey–predator model, as the latter, as shown in Section 4.4.1, always possesses asymptotically stable equilibria and limit cycles can never arise.
Chapter 5

Case study: biological pest control in vineyards

Vineyards are agroecosystems that strongly reflect man’s historical relationship with the land. In Piedmont (NW Italy), as in other countries, this kind of agroecosystem is facing land abandonment and agricultural intensification. The latter process of transformation results in the progressive removal of natural landscape elements, in particular loss of woods in vineyard landscapes, thus contributing to loss of heterogeneity affecting biotic communities (Agger and Brandt, 1988). Wise (1993) stresses the importance of the spider assemblages in limiting insect pests in agroecosystems and remarks on the need for research on prey limitation in natural communities and agroecosystems. The landscape structure then needs to be considered both in spatial and temporal terms (Merriam, 1988). Spatial diversity is given by the number of spider habitat types available and landscape heterogeneity by how intermingled they are. Temporally, the landscape can also be described with respect to disturbance synchronization, e.g., by season and by how large a proportion of the fields are at the same time affected by management, crop rotation, land use, and husbandry (Thomas et al., 1990). Thus the landscape structure is not static. Spider communities are significantly affected by these factors, and different agroecosystem dynamics can show different spider assemblages (Isaia et al., 2006b).

The spiders’ role in agroecosystems is not completely understood, but their impact is shown by several studies, e.g., see Wise (1993), Furuta (1977), and Nyffeler and Benz (1987). In more general terms, Nyffeler and Benz (1987), in reviewing several recent studies, support the conclusion that wandering spiders contribute significantly to the control of insect populations in soil/litter systems. The role of web weaving spiders as biological controllers is studied much more frequently by ecologists than wanderer spiders, since prey can be easily supplemented to web spinners, not moving their web sites frequently. Thus feeding behavior and interactions with conspecific and fecundity can be observed and measured directly and easily.

Here we consider first two mathematical models for understanding wanderer spiders as controllers of vineyard-infesting insects, expressly modeling the residual wood and green patches as the spiders’ habitat in the otherwise homogeneous landscape of the Langa Astigiana. These predators can move around among vineyards and woods, in which two different prey, i.e., two
insect populations, live. Since vineyards predominate on woods, the insects affecting the cultures will be more abundant than the wood ones, so that in the more complex model spiders experience feeding satiation. Since they are able to move around and seek other food sources, they will then predate on the insect population living in the nearby green patches. In both models we then investigate the effect of human activities in this ecosystem, by also considering insecticide spraying and its effects.

We then consider a transport phenomenon named ballooning for which young spiders are carried by the wind from vineyards into the surrounding woods, and vice versa, since this appears to be one of the key factors for the evolutionary success of spiders (Greenstone et al., 1987). It appears to be an example of an evolved behavior to avoid competition (Wise, 1993). Most ballooners are immature instars under 1.0 mg, but the most frequent long-range dispersers are the smaller adult Linyphiids. In view of the landscape not being a static environment, this immigration process becomes important as the spider population in a single field may need to recover several times every year. Experiments to understand population dynamics of Linyphiids are very labor intensive and expensive to carry out on a landscape scale (Wiens et al., 1993). From these considerations the need of modeling this phenomenon arises. The third mathematical model we propose takes into account this transport phenomenon. A relevant feature consists in not taking space explicitly into account in the governing equations, in contrast to what is done elsewhere (Halley et al., 1996).

5.1 First model

Since we are considering wanderer spiders, we denote their population by \( s(T) \) independently of the location in which they live. On the other hand, to model the differences in the landscape, the insects living in open fields and woods are denoted by \( w(T) \), and by \( v(T) \) we denote the population of parasites living in vineyards. Note that both are prey for these types of spiders, as they can move from one type of landscape to the other. The model is then given by a single predator population that feeds on two types of prey, not interacting with each other. It reads

\[
\frac{ds}{dT} = s(-a + kbv + kcw),
\]

\[
\frac{dw}{dT} = r w \left( 1 - \frac{w}{W} \right) - cs w,
\]

\[
\frac{dv}{dT} = v(e - bs).
\]

The first equation states as mentioned that \( w \) and \( v \) are prey for the spiders,
but, moreover, since the latter die exponentially fast when the former lack, these two types of insects are then the only food source for spiders. The second equation models the wood-living insects, reproducing logistically. Notice that we assume a small carrying capacity $W$, in view of the reduced extension of their environment with respect to the vineyard. The third equation describes the Malthus growth of the parasites in the vineyard, since the latter is so large that we can take it as having unlimited resources. Predation is modeled by mass action law in each equation. Prey are turned into new spiders via the “efficiency” constant $0 < k < 1$.

We as usual investigate the feasible equilibria of (5.1) to find the origin $E_0 \equiv O$ and the points

\[ E_1 \equiv (0, W, 0), \quad E_2 \equiv \left( \frac{e}{b}, 0, \frac{a}{bk} \right), \]
\[ E_3 \equiv (s_3, w_3, 0) = \left( \frac{c}{k} \left( 1 - \frac{a}{ckW} \right), \frac{a}{ck}, 0 \right), \]
\[ E_4 \equiv \left( \frac{e}{b}, W \left( 1 - \frac{ce}{br} \right), \frac{1}{bk} \left[ a - ckW \left( 1 - \frac{ce}{br} \right) \right] \right). \]

The feasibility condition for $E_3$ is $a < ckW$, while those for $E_4$ are instead $br > ce$ and $abr > ckW(br - ce)$.

The local stability analysis shows that $E_0$ and $E_1$ are always unstable, their eigenvalues being respectively $r, -a, e$ and $-r, -a + ckW, e$. Those for $E_2$ are given by $b^{-1}(br - ce), \pm i\sqrt{ac}$, thus giving a stable or unstable center in the $s, v$ plane, depending on whether $br < ce$. The eigenvalues of $E_3$ are $\lambda_3(1) = e - bs_3 < 0$ and the roots of the quadratic equation $\Psi(\lambda) \equiv ckW\lambda^2 + ra\lambda + ar(ckW - a) = 0$. Since $\Psi(0) > 0$ and $\Psi'(0) > 0$, we obtain two roots with negative real parts. Hence $E_3$ is stable if and only if $c^2kW^2 < \frac{e}{br}(ckW - a)$. Finally, the Routh–Hurwitz criterion applied to the characteristic equation of $E_1$,

\[ b^2r\lambda^3 + br(br - ce)\lambda^2 + \lambda e \left[ ab^2r + ckW(br - ce)(c - b) \right] + e(br - ce)[abr - ckW(br - ce)] = 0, \]

coincide with the two feasibility conditions of the same equilibrium $E_4$. Thus the latter is inconditionally stable when feasible.

Out of the five possible equilibria, the origin has two positive eigenvalues, so that the populations $w$ and $v$ increase. It is then always unstable. Similar results hold for $E_4$, in this case one of its three eigenvalue is positive, implying the growth of $v$.

The behavior of the system is thus determined by the remaining three equilibria. Notice that $E_4$ is always stable when feasible, $E_3$ is conditionally stable, and it is if and only if $E_4$ is infeasible. The point $E_2$ has one real and a pair of pure imaginary eigenvalues, so that it behaves like a center in the $s, v$ plane. If $br < ce$, the latter is stable, i.e., spiders and vineyard parasites tend to a Lotka–Volterra behavior in the $s, v$ plane around the projected point $(\xi, \frac{a}{ce})$. 
Otherwise the system shows an oscillatory behavior moving away from the $s,v$ plane along the $w$ direction. These features can be tracked by the numerical simulations, which are not reported here. Notice also that there are strict interdependencies among the feasibility and stability of some of these nontrivial equilibria. More specifically, $E_2$ stable implies $E_4$ infeasible and $E_3$ unstable. Also, $E_4$ feasible implies the instability of $E_2$.

Summarizing, if $E_4$ is feasible, it is the only possible stable equilibrium. When it is infeasible because of $br < ce$ and $abr > ckW(br - ce)$, the equilibrium $E_2$ is a stable center. When it is infeasible because of $abr < ckW(br - ce)$ but $br > ce$, the point $E_3$ is feasible and stable. Note that the two infeasibility conditions for $E_4$, namely, $br < ce$ and $abr < ckW(br - ce)$, cannot occur at the same time. In conclusion, only one of the three equilibria at the time determines the final behavior of the system.

Numerical experiments further substantiate the theoretical findings, revealing the existence of oscillations in some cases. This indicates that the system seems to be persistent, although not at a steady state (see Figure 5.1), for the following parameter values: $a = 3.1$, $b = 2.87$, $c = 8$, $e = 3.5$, $r = 1$, $W = 5$, $k = 0.8$. However, for these values the equilibria are $E_2 \equiv (1.2195, 0, 1.3502)$ with real eigenvalue $\lambda_1^{[2]} = 0.0244$ and $E_4 \equiv (1.2195, 0.1220,1.3162)$ with real eigenvalue $\lambda_1^{[4]} = -0.0242$. Thus, $E_2$ is unstable and repels the trajectories, which oscillate neutrally around it as we have theoretically seen they should do. The close stable equilibrium $E_4$ attracts them, but the eigenvalues are both small so that the dynamics is very slow, apparently giving rise to limit cycles, which, however, on a very much longer time scale should result in being damped toward $E_4$. In Figure 5.2 we report the findings on the spiders controlling the pests. This occurs for the parameter values $a = 3.1$, $b = 0.001$, $c = 0.2$, $e = 25$, $r = 1$, $W = 5$, $k = 0.8$, but the populations periodically rise to very high levels.

### 5.1.1 Modeling the human activity

The intervention in the ecosystem caused by man consists here in insecticide spraying, modeled by an impulse function, at particular instances in time. Let $\delta(T_i)$ denote Dirac’s delta function and $T_i$, $i = 1, 2, ...$ be the spraying instants. Equations (5.1) are modified as follows:

\[
\begin{align*}
\frac{ds}{dT} &= s(-a + kbv + kcw) - hKq\delta(T_i) \\
\frac{dw}{dT} &= rw \left(1 - \frac{w}{W}\right) - csw - h(1 - q)\delta(T_i) \\
\frac{dv}{dT} &= v(e - bs) - hq\delta(T_i).
\end{align*}
\]

We allow for a part $1 - q$ of insecticide sprayed ending accidentally in the woods, while the fraction $q$ lands on the vineyards as aimed. Also, $h$ denotes
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FIGURE 5.1: Wanderer spiders model: apparently persistent oscillations around the interior equilibrium.

FIGURE 5.2: Wanderer spiders model: the spiders control the pests, although at very high levels.
the effectiveness against the parasites, the parameter $0 < K < 1$ models instead the smaller effect it supposedly should have on the spiders as the poison is meant to act on the pests and the former may be less affected.

The simulations are reported in Figures 5.3–5.6. Figure 5.3 is a reference picture, when no spraying is used. We see that the ecosystem thrives at least for the time shown. The effects of the spraying are then added. For a small fraction of insecticide landing in the woods, i.e., high $q = 0.9$, Figure 5.4, the wood insects survive, while the whole vineyard ecosystem is wiped away. In Figure 5.5, more adverse conditions are modeled where the whole ecosystem collapses, for a high fraction $q = 0.2$ of poison ending in the wood insects’ habitat. The discontinuities are hardly seen in these cases as $K$ is small. Finally, Figure 5.6 shows that in some particularly nasty situations, even though the spraying may be aimed at controlling the vineyard pests, it is actually the spiders and the wood insects that are affected, as their populations vanish, while the pests remain uncontrolled and their growth explodes.

The results of our simulations show that spraying seems to destabilize the vineyards’ pests explosions. These considerations are useful in the Langa Astigiana, where the small woods constitute a refuge for many spiders and parasites, other than the vineyards’ own. Under natural conditions the ecosystem persists globally.

5.2 More sophisticated model

Wanderer spiders frequently confronted with a shortage of prey search for more productive microhabitats on the basis of prey abundance (Edgar, 1971; Kronk and Riechert, 1979; Morse and Fritz, 1982). Laboratory-field comparisons (Edgar, 1969; Hagstrum, 1970; Anderson, 1974) show that an increase in prey availability favors their growth rate and fecundity. If increases in these parameters tend to increase the average population, we can assume that wandering spiders are theoretically food limited. An increase of the population increases competition for territoriality among adults and thus favors their dispersal behavior. Researchers’ tests show that spiders are not constantly food limited, but intrinsic limitations in their experimental design mean that food limitation cannot be ruled out for most of the species. More direct and indirect evidence points out the fact that spiders are frequently hungry, even to exhibit growth and reproduction rates below what is thought to be physiologically possible (Wise, 1993). There is evidence that spiders, for normal growth and reproduction, need to actively select prey so as to optimize the diet proportion of essential amino acids (Miyashita, 1968; Greenstone, 1979). The proportion of different types of prey in the diet and thus the need for
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FIGURE 5.3: Wanderer spiders model-spraying effects: reference figure with no spray, persistent populations in ecosystem.

FIGURE 5.4: Wanderer spiders model-spraying effects: the wood insects survive, the vineyard ecosystem is wiped away.
FIGURE 5.5: Wanderer spiders model-spraying effects: entire ecosystem collapse.

FIGURE 5.6: Wanderer spiders model-spraying effects: vineyard pests explosion, all spiders are wiped away.
searching for new microhabitats could then be due to the degree of satiation of spiders and to their rejection of certain types of prey.

We keep here the same notation previously used that led to (5.1). In the first equation we find again the dynamics of spiders, in which, as before, a negative Malthus growth describes their mortality, and the two nonnegative terms account for the growth due to predation on the two insect populations. But in contrast to (5.1), here we model the saturation effect the spiders experience when the vineyard insects are too numerous. In such a case, since the wanderers move around and search for other prey in the surroundings of the cultivated land, in the green niches around the vineyards inhabited by the $v$ insects, a suitable alternative prey source is found. Thus, basing our considerations on the field data analysis of Rypstra (1995) and Marc et al. (1999), we use Holling type II responses to model this feeding saturation effect. These terms of course reappear in the appropriate insect evolution equation, but for the rest the dynamics of all the insect populations considered is the same as in (5.1):

$$
\frac{ds}{dT} = s \left(-a + \frac{kbw}{H + v + kw}\right),
$$

$$
\frac{dw}{dT} = rw \left(1 - \frac{w}{W}\right) - csw,
$$

$$
\frac{dv}{dT} = v \left[e - \frac{bs}{H + v}\right].
$$

Analysis of the boundary equilibria

The first two feasible equilibria $E_0$ and $E_1$ of (5.1) are found again here, with the same inconditional instability due to the occurrence of the very same eigenvalues. The equilibrium point $E_2$ instead gets modified as follows:

$\hat{E}_2 \equiv (\hat{s}_2, 0, \hat{v}_2) = \left(\frac{ekH}{bk - a}, 0, \frac{aH}{bk - a}\right)$. In this case, it exists only if $a < bk$, while for $E_2$ no such restriction arises. The last equilibrium point lying on the coordinate planes $\hat{E}_3$ is exactly $E_3$ of (5.1), with of course the same existence condition $a < ckW$.

Stability conditions of the last two equilibria differ however from those derived for (5.1). Notice indeed that for $\hat{E}_3$, although derived similarly to the former model, one of the eigenvalues is now $\frac{1}{H}(cH - bs_3)$ while the other two are the roots of the quadratic $W k c \omega^2 + r(cWk - 2a)\omega + W k^2 c s_3 = 0$. Hence $E_3$ is stable if and only if $br(ckW - a) > c^2 ekHW$ and $2a < ckW$. Notice that the presence of the half-saturation constant $H$ renders the satisfaction of the first condition more strict than for (5.1).

The eigenvalues of $\hat{E}_2$ are $r - \frac{1}{a}ce\hat{v}_2$ and those of the matrix

$$
J = \begin{pmatrix}
\frac{1}{a(H + \hat{v}_2)^2} \left(ab\hat{v}_2(H + \hat{v}_2) - a^2(H + \hat{v}_2)^2\right) & \frac{bekH\hat{v}_2}{a(H + \hat{v}_2)^2} \\
-\frac{ab\hat{v}_2(H + \hat{v}_2)}{a(H + \hat{v}_2)^2} & \frac{ae(H + \hat{v}_2)^2 - beH\hat{v}_2}{a(H + \hat{v}_2)^2}
\end{pmatrix}.
$$
This gives an upper bound on the location of this root.

where

Here, the Routh–Hurwitz criterion for \( \hat{\lambda} \) is

The coefficients are unique and feasible if and only if the following conditions are satisfied:

\[
bkW(H + \hat{\lambda})^2 > a(\hat{\lambda} + H)^2, \quad bkW(\hat{\lambda} + H)^2 > a \hat{\lambda}.
\]

Nontrivial equilibrium \( E_4 \)

Now, from the ecological standpoint, of particular interest is the system equilibrium preserving biodiversity, i.e., ensuring the coexistence of all species.

The equilibrium point at which the ecosystem thrives is

\[
\hat{E}_4 \equiv (\hat{\omega}_4, \hat{s}_4, \hat{\lambda}_4) = \left( \frac{1}{b} e(H + \hat{\lambda}_4), \frac{1}{br} W(br - c\hat{\lambda}_4)[e(H + \hat{\lambda}_4)], \hat{\lambda}_4 \right),
\]

and \( \hat{\lambda}_4 > 0 \) solves the following quadratic equation:

\[
kc^2We^2 + (abr - kb^2r + 2kc^2WHe - kcWB)u
+ aHbr - kcWbr + kc^2WH^2c = 0.
\]

Two positive roots arise if \( bcbrW < abr + 2c^2ekWH < bcbrW + kb^2r \), while there is only one if \( abr + kc^2WHe < kcWbr \). Thus the interior equilibrium is unique and feasible if and only if the latter condition holds and \( br > c_0(H + \hat{\lambda}_4) \).

This gives an upper bound on the location of this root.

For stability, let us analyze once again the characteristic equation at \( \hat{E}_4 \),

\[
\lambda^3 + Q_1\lambda^2 + Q_2\lambda + Q_3 = 0.
\]

The coefficients are

\[
Q_1 = -bkW\hat{\omega}_4 + rH^2\hat{\omega}_4 + 2rH\hat{\omega}_4\hat{\lambda}_4 + r\hat{\omega}_4\hat{\lambda}_4^2,
Q_2 = \frac{s}{W(H + \hat{\lambda}_4)^2} \left[ b^2kHW\hat{\omega}_4 - brH\hat{\omega}_4\hat{\lambda}_4 - br\hat{\omega}_4(\hat{\lambda}_4)^2 + kc^2W(H + \hat{\lambda}_4)^3\hat{\omega}_4 \right],
Q_3 = \frac{bk\hat{\omega}_4\hat{s}_4\hat{\lambda}_4}{W(H + \hat{\lambda}_4)^3} \left[ brH - c^2W\hat{\omega}_4 - c^2W\hat{s}_4\hat{\lambda}_4 \right].
\]

The Routh–Hurwitz criterion for \( \hat{E}_4 \) gives \( Q_1 > 0, Q_2 > 0 \) and \( Q_1Q_2 - Q_3 > 0 \). Here,

\[
Q_1Q_2 - Q_3 = \frac{\hat{\omega}_4}{W^2(H + \hat{\lambda}_4)^3} \cdot \left[ r(kc^2HW\hat{\omega}_4 + A)\hat{\omega}_4^2\hat{\lambda}_4 + (b^2rW\hat{\omega}_4 + rB\hat{\omega}_4)\hat{\omega}_4^2\hat{\lambda}_4^2 + D \right],
\]

where \( A = 5c^2kWH - br, B = 10Hc^2kW - 3br, C = rBH^2\hat{\omega}_4 + b^2HW\hat{\omega}_4(r\hat{\omega}_4 - bkW), \) and \( D = \hat{\omega}_4^2C + rAH^3\hat{\omega}_4^2\hat{\lambda}_4 + rc^2kH^3W\hat{\omega}_4^3 \).
We now verify the transversality condition (ii) of (5.7) at
however, the roots are functions of
The three roots of (5.6) are
sality condition must hold. Thus a value
which entails the equality
Bifurcation analysis
We now investigate the stability behavior of the coexistence equilibrium as
and
Sufficient conditions for these conditions to hold are 10c²KH > 3br and
r w  > bkW. The latter should be satisfied together with the above feasibility
conditions.

\begin{align*}
\text{Case study: biological pest control in vineyards} &  \\
We then find that Q_1 > 0 if and only if \\
\quad rH^2\dot{w}_4 + 2rH\dot{w}_4\dot{v}_4 + r\dot{w}_4\dot{v}_4^2 > bW\dot{s}_4\dot{v}_4, \\
\quad Q_3 > 0 if and only if \\
\quad bHr > c^2HW\dot{s}_4 + c^3W\dot{s}_4\dot{v}_4, \\
\text{and } Q_1Q_2 - Q_3 > 0 if and only if \\
\quad Q_1Q_2 - Q_3 \equiv r(c^2kW\dot{v}_4 + A)\dot{w}_4\dot{v}_4^2 + [b^2rW\dot{s}_4 + \dot{w}_4rB]\dot{w}_4\dot{v}_4^3 + D > 0. \quad (5.5)
\end{align*}

Sufficient conditions for these conditions to hold are 10c²KH > 3br and
r w  > bkW. The latter should be satisfied together with the above feasibility
conditions.

\begin{align*}
\text{Bifurcation analysis} &  \\
\text{We now investigate the stability behavior of the coexistence equilibrium as}
\text{a function of the parameter } c \text{ using a relatively simple method. To ensure}
\text{that the characteristic polynomial (5.4) has purely imaginary roots, it must}
\text{have the form}
\quad (\lambda^2 + Q_2)(\lambda + Q_1) = 0, \quad (5.6)
\text{which entails the equality } Q_1Q_2 = Q_3 \text{ associated to (5.5). Also, a transver-
\text{sality condition must hold. Thus a value } c = c^* \text{ must exist such that}
\quad (i) \ g(c^*) = Q_1(c^*)Q_2(c^*) - Q_3(c^*) = 0, \quad (ii) \ \frac{d}{dc}\text{Re}(\lambda(c))|_{c=c^*} \neq 0. \quad (5.7)
\text{The three roots of (5.6) are } \eta_1 = +i\sqrt{Q_2}, \ \eta_2 = -i\sqrt{Q_2}, \ \eta_3 = -Q_1. \ \text{In general,}
\text{however, the roots are functions of } c:
\quad \eta_1(c) = \beta_1(c) + i\beta_2(c), \quad \eta_2(c) = \beta_1(c) - i\beta_2(c), \quad \eta_3(c) = -Q_1(c).
\text{We now verify the transversality condition (ii) of (5.7) at } \eta_1 \text{ and } \eta_2. \ \text{Substitut-
\text{ing the general form of the root } \eta_j(c) = \beta_1(c) + i\beta_2(c) \text{ into (5.6) and taking}
\text{the derivative, we find}
\quad K(c)\beta_1(c) - L(C)\beta_2(c) + A(c) = 0, \quad L(c)\beta_1(c) + K(c)\beta_2(c) + B(c) = 0,
\text{with}
\quad K(c) = 3\beta^2_1(c) + 2Q_1(c)\beta_1(c) + Q_2(c) - 3\beta^2_2(c),
\quad L(c) = 6\beta_1(c)\beta_2(c) + 2Q_1(c)\beta_2(c),
\quad A(c) = \beta^2_1(c)Q_1(c) + Q^*_2(c)\beta_1(c) + Q^*_3(c) - Q_1(c)\beta^2_2(c),
\quad B(c) = 2\beta_1(c)\beta_2(c)Q_1(c) + Q^*_2(c)\beta_2(c).
Since $L(c^\star)B(c^\star) + K(c^\star)A(c^\star) \neq 0$, we have

$$
\frac{d}{dc}(\text{Re}(\eta_j(c)))\bigg|_{c=c^\star} = \left[ \frac{LB + KA}{K^2 + L^2} \right]_{c=c^\star} \neq 0
$$

and $\eta_3(c^\star) = -Q_1(c^\star) \neq 0$ so that the transversality condition holds. Thus a nondegenerate Hopf bifurcation occurs at $c = c^\star$.

**Action of spraying and new simulations**

Again human intervention in the ecosystem is modeled via insecticide spraying, which in the previous case mathematically corresponded to the addition of Dirac’s delta functions at suitable instances in time to the system (5.1). It is modified here to take into account the fact that poisonous effects of the insecticide last in time. We assume then an exponential decay of these effects $\psi(T_i) = e^{-\alpha(t-T_i)}$, where $T_i \leq t$, $i = 1, 2, ..., N$ denote the spraying instances for (5.3). We then get

$$
\frac{ds}{dT} = s(-a + \frac{kbv}{H + v} + kcw) - hKq\Sigma_{i=1}^{N}\psi(T_i),
$$

$$
\frac{dw}{dT} = rw(1 - \frac{w}{W}) - cs(1 - q)\Sigma_{i=1}^{N}\psi(T_i),
$$

$$
\frac{dv}{dT} = v(e - \frac{bs}{H + v}) - hq\Sigma_{i=1}^{N}\psi(T_i).
$$

The insecticide efficacy is modeled by the parameter $\alpha$, with the parameters $q, K, h$ retaining their meaning as for (5.2).

The behavior near the equilibria determined by the theoretical analysis can also be checked by means of suitable situations. In the absence of spraying, we show in Figure 5.7 the limit cycles obtained with the Hopf bifurcation described earlier. As far as the spraying is involved, general considerations on the collapsing of the ecosystem or some of its parts entirely similar to the ones of the previous model without saturation (5.1) also hold in this case, despite the different kind of modeling for the poison’s effects used in this case. But for this more realistic situation (5.8) with insecticide effects lasting in time, the insects in the cultivated areas as well as the whole spider population become extinct even for very low parameter values.

**5.2.1 Models comparison**

A major difference between the two proposed models (5.1) and (5.3) is found in the cycles that can arise in the model introduced here, while they do arise in the simpler model. This is in agreement with recently collected vineyard field data, showing persistent oscillations for the spider population (Isaia et al., 2006a).
Case study: biological pest control in vineyards

FIGURE 5.7: Limit cycles obtained by the Hopf bifurcation for the Holling type II saturation model (5.3).

From the numerical simulations, to maintain stability of the coexistence equilibrium point, the predation rates must be in an interval with lower and upper bounds respectively depending on the prey in the woods and the prey in the vineyards, i.e., the gap between the parameters $b$ and $c$ should not be too large. Should this difference be so large that the system oscillates, the stable equilibrium can be reobtained by an increase in the specific growth rate of the prey in the woods, i.e., the parameter $r$.

The method of spider augmentation led to 60% reduction of pesticide use, Maloney et al. (2003) and the effectiveness of biological control through spiders in rice fields and fruit orchards has been documented (Riechert and Bishop, 1990; Marc and Canard, 1997; Nyffeler and Benz, 1987). On the other hand, some insecticides are highly toxic for spiders, for instance, dimethoate gives 100% mortality for the lycosid *Trochosa ruricola* (Birnie et al., 1998) even when applied at lower concentrations than recommended, and methyl paranthion and pyrethroid cypermethrng are poisons for the genus *Erigone* (Linyphiidae) (Brown et al., 1994; Huusela-Veistola, 1998).

These field observations should be coupled with our numerical experiments. In the simpler model (5.2) simulations showed that populations can recover after spraying if the atmospheric and environmental conditions are not too harsh. In the ecosystem (5.8), instead, even very low parameter values related to human intervention can permanently destabilize the ecosystem. Thus the results of these models agree with field data. Halley et al. (1996) discovered that if spraying is low and less frequently used, spiders can survive, but for
larger quantities of the insecticide or if frequently used, chances are that
the spider population will be wiped out. This is also documented by the
mathematical models presented here.

5.3 Modeling the ballooning effect

The model we introduce here differs from the previous ones since in this
context spiders are essentially stantial, so that we need to also distinguish the
two spider populations living in the woods and in the vineyard, respectively,
$s_w(t)$ and $s_v(t)$. We keep the same notation used earlier, but this time we
also assume a vineyard insects carrying capacity $V$, with $V >> W$, to model
the Langhe environment. The model then reads

\[
\frac{dw}{dT} = bw(1 - \frac{w}{W}) - \ell ws_w,  \tag{5.9}
\]

\[
\frac{ds_w}{dT} = -cs_w + s_w[\tilde{\ell}w - \alpha v\tilde{k} - \frac{V}{V + W}] + s_w\alpha v\tilde{k} - \frac{W}{V + W},
\]

\[
\frac{dv}{dT} = av(1 - \frac{v}{V}) - kus_v,
\]

\[
\frac{ds_v}{dT} = -es_v + s_v[\tilde{k}v - \alpha v\tilde{k} - \frac{W}{V + W}] + s_w\alpha v\tilde{k} - \frac{V}{V + W}.
\]

Here the first two equations express the fact that insects reproduce logistically
and are subject to predation by the spiders living in the same environment.
The two ecosystems are separate entities, as the web-making spiders tend to
live in the same place, if there is no ballooning effect. Wind transport of
young spiders is taken care of in the two equations modeling the growth of
the spider populations. A fraction $\alpha$ of newborns is carried in the air and
may land either in woods or surrounding vineyards with probability that is
assumed proportional to the surface of the two patches, which in turn is related
to their respective carrying capacities. The last two terms in the spiders’
evolution equations describe this migration effect. The last one represents
the immigration of newborns into the woods from the vineyards in the third
equation and conversely in the fourth equation, while the third terms represent
the emigration of wood-born spiders into the surrounding landscape in the
third equation and the opposite way in the dynamics of the vineyard spiders.
The predation efficiency of the latter is $\tilde{k}$. Notice that the transport effect of
the wind is clearly the same for both species, while it is neglected for insects,
as they are able to fly on their own and move toward the environment they
prefer.
Case study: biological pest control in vineyards

Boundedness of the solutions

Now we are going to have a look at the properties of the model (5.9). The first step is to check solution boundedness.

The first two equations of (5.9) show that the two insect populations are bounded above:

\[ w(t) \leq \max\{w(0), W\}, \quad \limsup_{t \to \infty} w(t) \leq W; \quad (5.10) \]

\[ v(t) \leq \max\{v(0), V\}, \quad \limsup_{t \to \infty} v(t) \leq V. \]

Let us also define \( \phi(T) = w + v + s_w + s_v \). Summing the equations (5.9) for any arbitrary \( \kappa \leq \min\{c, e, \ell - \bar{\ell}, k - \bar{k}\} \), we have

\[
\frac{d\phi}{dT} + \kappa \phi \leq bw \left(1 - \frac{w}{W}\right) + av \left(1 - \frac{v}{V}\right) \\
- (c - \kappa)s_w - (e - \kappa)s_v - (\ell - \bar{\ell} - \kappa)s_w - (k - \bar{k} - \kappa)s_v \\
\leq bw \left(1 - \frac{w}{W}\right) + av \left(1 - \frac{v}{V}\right) \leq \frac{1}{4}[bW + aV] \equiv \bar{M}.
\]

Then \( \frac{d\phi}{dT} \leq -\kappa \phi + \bar{M} \), from which Gronwall’s inequality shows that every population is bounded for all time:

\[ \phi(t) \leq \exp(-\kappa t) + \frac{\bar{M}}{\kappa} [1 - \exp(-\kappa t)] \leq M. \quad (5.11) \]

Long-term behavior

Now, we need to analyze the system’s equilibria \( E^{(i)} \equiv (w^{(i)}, v^{(i)}, s_w^{(i)}, s_v^{(i)}) \).

The first three, the origin \( E^{(0)} \) and the axial points with \( w^{(1)} = W \), \( w^{(2)} = V \), where the other components are zero, are easily ruled out, since they are unstable. Respectively, we find the eigenvalues

\[ a, b, -c, -e; \]
\[ a, -b, -c + \bar{\ell} \frac{W}{V+W} (V+W-\alpha V); \]
\[ b, -a, -c - e + \bar{k} \frac{V}{V+W} (V+W-\alpha W). \]

The first interesting equilibrium is \( E^{(3)} \equiv (W, V, 0, 0) \), whose eigenvalues are \(-a, -b, \) and the roots of the quadratic equation

\[
\lambda^2 + \lambda \left[ c - W \bar{\ell} + e - V \bar{k} + \alpha VW(\bar{\ell} + \bar{k}) \right] \\
+ \left( c - W \bar{\ell} + \alpha VW \bar{\ell} \right) \left( c - V \bar{k} + \alpha VW \bar{k} \right) - \frac{V^2 W^2 \bar{\ell} \bar{k} \alpha^2}{(V+W)^2} = 0.
\]
Sufficient conditions for which the Routh–Hurwitz criterion holds, so that \( \mathcal{E}^{(3)} \) is stable, are

\[
    c > W\bar{\ell}, \quad e > V\bar{k}.
\]

(5.12)

The next two are still boundary equilibria, with only one zero component, and in them one of the two insect populations goes extinct. We find

\[
    \mathcal{E}^{(4)} \equiv \left( 0, \frac{c(V + W)}{k(V + W - \alpha W)} \right),
\]

\[
    \frac{a}{k} \left[ 1 - \frac{e(V + W)}{kV(V + W - \alpha W)} \right],
\]

(5.13)

\[
    \mathcal{E}^{(5)} \equiv \left( \frac{c(V + W)}{\ell(V + W - \alpha V)}, 0 \right), \quad \frac{bV\alpha}{\ell} - e \left[ \frac{c(V + W)}{\ell W(V + W - \alpha V)} \right].
\]

(5.14)

An easy verification based on the fact that \( 0 \leq \alpha \leq 1 \) shows that the feasibility conditions reduce respectively to

\[
    \tilde{k}V(V + W - \alpha W) > c(V + W), \quad \tilde{\ell}W(V + W - \alpha V) > c(V + W).
\]

(5.15)

For the stability analysis, let us calculate the Jacobian of the system \( J^{(i)} \equiv J(\mathcal{E}^{(i)}) \):

\[
   \begin{pmatrix}
   b(1 - 2\bar{w}^{(i)}) - \ell\bar{s}^w^{(i)} & 0 & -\ell\bar{w}^{(i)} & 0 \\
   0 & a(1 - 2\bar{v}^{(i)}) - k\bar{s}^v^{(i)} & 0 & -k\bar{v}^{(i)} \\
   (1 - \alpha \frac{V}{V + W})\ell\bar{s}^w^{(i)} & \alpha \frac{W}{V + W}\ell\bar{s}^v^{(i)} & (1 - \alpha \frac{V}{V + W})\ell\bar{w}^{(i)} - c & \alpha \frac{W}{V + W}\ell\bar{v}^{(i)} \\
   \alpha \frac{V}{V + W}\ell\bar{s}^w^{(i)} & \bar{s}^v^{(i)}(1 - \alpha \frac{W}{V + W}) & \bar{w}^{(i)}(1 - \alpha \frac{W}{V + W}) & \bar{v}^{(i)}(1 - \alpha \frac{W}{V + W}) - e
   \end{pmatrix}
\]

Thus, for equilibrium \( \mathcal{E}^{(4)} \), the eigenvalues are \(-c, b - \ell\bar{s}_w^{(4)}\), and the roots of the quadratic

\[
   \lambda^2 - \text{tr}(\tilde{J}(\mathcal{E}^{(4)}))\lambda + \det(\tilde{J}(\mathcal{E}^{(4)})) = 0,
\]

(5.16)

where

\[
   \tilde{J}(\mathcal{E}^{(4)}) \equiv \begin{pmatrix}
   a(1 - 2\bar{v}^{(4)}) - k\bar{s}^v^{(4)} & -k\bar{v}^{(4)} \\
   k\bar{s}^v^{(4)}(1 - \alpha \frac{W}{V + W}) & -k\bar{v}^{(4)}(1 - \alpha \frac{W}{V + W}) - e
   \end{pmatrix}
\]

(5.17)
The Routh–Hurwitz conditions for stability are $-\text{tr}(J^{(4)}) > 0$ and $\det(J^{(4)}) > 0$, and in view of $0 \leq \alpha \leq 1$ we find

$$-\text{tr}(J^{(4)}) \equiv k s_v^{(4)} - a + 2 \frac{a}{V} v^{(4)} = \alpha c \frac{V + W}{k V (V + W - \alpha W)},$$

$$\det(J^{(4)}) \equiv \tilde{k} v^{(4)} s_v^{(4)} \frac{V + W - \alpha W}{V + W} > 0.$$

Stability of $E^{(4)}$ follows by just requiring

$$b < \ell s_w^{(4)}.$$

(5.18)

For $E^{(5)}$, the analysis is similar. We find the two eigenvalues, $-e$, $a - ks_v^{(5)}$, and then the eigenvalues of

$$\tilde{J}(E^{(5)}) \equiv \begin{pmatrix} b(1 - 2 \frac{w^{(5)}}{W}) - \ell s_w^{(5)} & -\ell w^{(5)} \\ \ell s_w^{(5)} (1 - \alpha \frac{V}{V + W}) & \ell w^{(5)} (1 - \alpha \frac{V}{V + W}) - c \end{pmatrix}$$

(5.19)

The Routh–Hurwitz criterion yields again

$$-\text{tr}(\tilde{J}(E^{(5)})) = \frac{bc(V + W)}{\ell W (V + W - \alpha V)} > 0,$$

$$\det(\tilde{J}(E^{(5)})) = \ell \tilde{w}^{(5)} s_w^{(5)} \frac{V + W - \alpha V}{V + W} > 0,$$

and stability of $E^{(5)}$ is ensured by

$$a < ks_v^{(5)}.$$

(5.20)

We analyze sufficient conditions for the existence of the coexistence equilibrium $E^{(6)} \equiv E^* = (w^*, v^*, s_w^*, s_v^*)$. Solving the first two equations of (5.9) in terms of $w$ and $v$, we find

$$w^* = \frac{W}{b} (b - \ell s_w^*), \quad v^* = \frac{V}{a} (a - ks_v^*).$$

(5.21)

For feasibility of $E^*$ the opposite conditions of (5.18) and (5.20), but both evaluated at $E^*$, must hold:

$$a > ks_v^*, \quad b > \ell s_w^*.$$

(5.22)
Let

\[ A \equiv \frac{\ell}{b} \frac{W}{V + W} (V + W - \alpha V) > 0, \]

\[ \tilde{B} \equiv \frac{1}{2} \frac{\ell}{b} \frac{W}{V + W} (V + W - \alpha V) - e \in \mathbb{R}, \]

\[ C \equiv \frac{1}{2} \tilde{k} \frac{V W}{V + W} > 0, \quad D \equiv \frac{k}{a} \frac{V W}{V + W} \tilde{k} \alpha > 0, \]

\[ E \equiv \frac{\ell}{b} \frac{V W}{V + W} \alpha > 0, \quad F \equiv \frac{1}{2} \tilde{k} \alpha \frac{V W}{V + W} > 0, \]

\[ \tilde{G} \equiv \frac{1}{2} \frac{\ell}{b} \frac{V}{V + W} (V + W - \alpha W) - e \in \mathbb{R}, \]

\[ H \equiv \frac{k}{a} \frac{V}{V + W} \tilde{k} (V + W - \alpha W) > 0. \]

Notice that in this context the feasibility of \( E(4) \) is equivalent to \( \tilde{G} \geq 0 \), and the one of \( E(5) \) corresponds to \( \tilde{B} \geq 0 \). Substitute now (5.21) into the last two equations of (5.9), to get two equations in \( s_w \) and \( s_v \),

\[ -A s_w^2 + 2\tilde{B} s_w + 2C s_v - D s_v^2 = 0, \quad (5.23) \]

\[ -E s_w^2 + 2F s_w + 2\tilde{G} s_v - H s_v^2 = 0. \quad (5.24) \]

Both (5.23) and (5.24) are ellipses in the \((s_w, s_v) \equiv (x, y)\) plane, both crossing the origin. Notice that the origin here in \( \mathbb{R}^2 \) gives for the whole model the three equilibria we named \( E^{(0)} \), \( E^{(1)} \), and \( E^{(2)} \). We now study the possible further intersections of the two ellipses.

The first ellipse with center \( \left( \frac{\tilde{B}}{A}, \frac{C}{D} \right) \) and vertices

\[ \left( \frac{\tilde{B}}{A} \pm \frac{1}{AD} \sqrt{B^2 D^2 + AC^2 D}, \frac{C}{D} \right) \]

\[ \left( \frac{\tilde{B}}{A} \frac{C}{D} \pm \frac{1}{AD} \sqrt{A^2 C^2 + AB^2 D} \right) \]

intersects the positive vertical semi-axis at \( P \equiv (x_P, y_P) = (0, 2\frac{C}{D}) \) and the horizontal axis at \( Q \equiv (x_Q, y_Q) = (2\frac{\tilde{B}}{A}, 0) \). Its derivative at the point \( P \) is

\[ y'_1(P) = \frac{\tilde{B}}{\frac{C}{D}}, \]

and at the origin it is \( y'_1(O) = -\frac{\tilde{B}}{\frac{C}{D}} \). It is then easily seen to have the same sign as the abscissa \( x_Q \) at \( P \), while at the origin it always has its opposite sign.
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The second one has center at \((\frac{F}{E}, \frac{G}{H})\), vertices
\[
\left( \frac{F}{E} + \frac{1}{EH} \sqrt{G^2E^2 + EF^2H^2}, \frac{G}{H} \right)
\]
and a further intersection with the positive vertical semiaxis at \(R \equiv (x_R, y_R) = (0, 2\frac{\tilde{G}}{\tilde{H}})\), with the horizontal axis at \(S \equiv (x_S, y_S) = (2\frac{\tilde{F}}{\tilde{E}}, 0)\). The derivative at the point \(R\) is \(y_2'(R) = \frac{F}{E}\tilde{G}\), and at the origin it is \(y_2'(O) = -\frac{F}{E}\tilde{G}\). It thus has the same sign as the height \(y_R\) when evaluated at \(R\) and opposite sign when evaluated at the origin. Thus both ellipses have axes parallel to the coordinate axes.

Four cases of sufficient conditions for the existence of \(E^*\) are obtained from the above results:

1A) \(\tilde{B} > 0, \tilde{G} > 0\); here existence and uniqueness of an intersection between \(y_1\) and \(y_2\) in the first quadrant, which gives a feasible \(E^*\) if conditions (5.22) also hold, are guaranteed by either
\[
CH > D\tilde{G} \quad \text{and} \quad FA > E\tilde{B}
\]
(5.27)
or, alternatively,
\[
CH < D\tilde{G} \quad \text{and} \quad FA < E\tilde{B}.
\]
(5.28)

1B) \(\tilde{B} > 0, \tilde{G} < 0\); we obtain only one condition,
\[
FA > E\tilde{B}.
\]
(5.29)

2A) For \(\tilde{B} < 0, \tilde{G} > 0\), the condition now is
\[
CH > D\tilde{G}.
\]
(5.30)

2B) For \(\tilde{B} < 0, \tilde{G} < 0\) comparing the derivatives of \(y_1\) and \(y_2\) at the origin, \(y_1'(O) < y_2'(O)\), we impose an intersection in the first quadrant, in other words,
\[
FC < \tilde{G}\tilde{B}.
\]
(5.31)

Conditions (5.27) are always satisfied upon substituting the values of \(A, \tilde{B}, C, D, E, F, \tilde{G}\). Indeed, they correspond to requiring \(e > 0\) and \(e > 0\) and geometrically to \(x_S \geq x_Q\) and \(y_P \geq y_R\), in which equality holds only for the cases \(e = 0\) and \(e = 0\), respectively. We find also that (5.29) and (5.30) hold always, together with (5.22). The latter imply that the intersection \(E^*\) of the ellipses must lie within the square with vertices \(OPUS\), where \(U \equiv (x_S, y_P) \equiv (2\frac{\tilde{E}}{\tilde{F}}, 2\frac{\tilde{F}}{\tilde{E}}) \equiv \left(\frac{\tilde{G}}{\tilde{H}}, \frac{2\tilde{F}}{\tilde{E}}\right)\). But this square is inscribed into the
first ellipse in the cases 1A and 1B corresponding to \( x_Q > 0 \), i.e., \( \bar{G} > 0 \), so that in such cases \( E^* \) is infeasible since one of the first two coordinates, \( v, w \), is negative. In cases 2A and 2B the arc \( OP \) of the first ellipse not containing \( Q \) lies entirely in the square \( OPUS \) and (5.30) and (5.31) guarantee an intersection. It follows that \( E^* \) is always feasible. Figure 5.8 depicts such situations.

FIGURE 5.8: Illustration of the possible ellipses intersections; on the left, a blowup of the right picture.

Let us look now at some particular cases, starting from \( e = 0 \). Two new equilibria then arise, \( \bar{E}^{(0)} \equiv (0, 0, 0, \bar{s}_v) \) for \( \bar{s}_v \equiv y_P \equiv \frac{a}{b} \), or also \( \bar{E}^{(1)} \equiv (W, 0, 0, \bar{s}_v) \). For \( c = 0 \) we find \( \bar{E}^{(2)} \equiv (0, 0, \bar{s}_w, 0) \) and \( \bar{E}^{(3)} \equiv (0, V, \bar{s}_w, 0) \), with \( \bar{s}_w \equiv \frac{b}{\ell} \). Substituting into the Jacobian, we find the eigenvalues \( a, b, -c, 0 \) giving instability for \( \bar{E}^{(0)} \) and \( -b, a - k\bar{s}_v \equiv 0, -c + \bar{G} \), \( \bar{E}^{(1)} \). The stability condition for the latter follows if \( \bar{G} < 0 \), i.e., \( \bar{B} < 0 \), or also \( x_Q < 0 \). Case 2A makes \( \bar{E}^{(1)} \) stable. The eigenvalues of \( \bar{E}^{(2)} \) are \( b - \ell\bar{s}_w \equiv 0, -a, 0, \bar{G} \), \( \bar{E}^{(3)} \) is stable if \( \bar{G} < 0 \), i.e., for \( y_R < 0 \), so that case 2A makes it unstable, while case 2B
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renders it stable. The equilibrium $E^{(3)}$ is always unstable, as the eigenvalues are $0, a, 0, -e$.

Now as $e$ grows, the axis intersection between the ellipses moves away from the $s_v$-axis. It retains its stability and thus gives a stable manifold, the arc $OP$ of the ellipse not containing the point $Q$, where $E^*$ is located. It can be shown that for $e < 0$ this is the stable branch of a saddle-node bifurcation of an infeasible equilibrium. Similar remarks hold exchanging $e$ with $c$, $P$ with $S$, and $Q$ with $R$ and reversing the stability concepts.

Condition (5.31) is equivalent to the restriction $y'_1(O) = -\frac{\bar{B}}{C} < -\frac{F}{\bar{G}} = y'_2(O)$, i.e., to $FACH > E\bar{B}\bar{G}D > EFCD$, which yields $A > \frac{D}{H}$. Substituting the parameter values and simplifying, we obtain $(V+W-\alpha V)(V+W-\alpha W) > VW$. From this the true inequality $(1-\alpha)(V^2+W^2)+VW(1-\alpha)^2 > 0$ follows, so that (5.31) always holds. Case 2B always gives a feasible intersection.

**Hopf bifurcation**

We also analyze in detail this case although the method is similar to the one used for the wanderer spiders models, since it applies here to a fourth degree equation, and the bifurcation condition (5.35) below differs from the one previously derived for a cubic equation. The characteristic polynomial in this case is the quartic in $\lambda$,

$$\sum_{i=0}^{4} b_i \lambda^i,$$  \hspace{1cm} (5.32)

and the following condition ensures that it has purely imaginary eigenvalues:

$$\sum_{i=0}^{4} b_i \lambda^i = (\lambda^2 + \eta^2)(\lambda^2 + \beta \lambda + \zeta) = 0.$$  \hspace{1cm} (5.33)

Expanding and equating coefficients of the same powers gives

$$b_3 = \beta, \quad b_2 = \zeta + \eta^2, \quad b_1 = \beta \eta^2, \quad b_0 = \eta^2 \zeta,$$  \hspace{1cm} (5.34)

which in turn are equivalent to the following relationship for the coefficients of (5.32):

$$b_3 b_2 b_1 = b_0^2 + b_1^2.$$  \hspace{1cm} (5.35)

Let us denote the principal minors of order $k$ of the Jacobian by $M_k(J)$. Assuming that the following sums are taken over all possible principal minors of the given order, we then find $b_3 = -\text{tr}(J)$, $b_2 = \sum M_2(J)$, $b_1 = -\sum M_3(J)$, $b_0 = \text{det}(J)$. As a bifurcation parameter we can take $e$, since it appears only in the element $J_{44}$ with a negative sign, or $c$ appearing only in $J_{33}$ also with negative sign. It turns out then that (5.35) is a cubic equation in $e$:

$$b_3 b_2 b_1 - b_0^2 - b_1^2 \equiv \sum_{i=0}^{3} c_i e^i.$$  \hspace{1cm} (5.36)
To guarantee the existence of a real positive root \( e^1 \), the signs of the constant term and of the coefficient of \( e^3 \) must be opposite. The condition (5.35) can be rewritten as

\[
\text{tr}(J) \sum M_2(J) \sum M_3(J) = (\text{tr}(J))^2 \det(J) + (\sum M_3(J))^2.
\]  
(5.37)

Let \( M_2[J_{(1:3,1:3)}] \) denote the principal minors of order two of the submatrix \( J(1 : 3, 1 : 3) \) of the Jacobian, formed by its first three rows and columns, and \( \det[J_{(1:3,1:3)}] \) be the corresponding determinant. From (5.37) we extract the highest order terms containing \( J_{44} \), obtaining

\[
J_{44} \cdot J_{44}[J_{11} + J_{22} + J_{33}] \cdot J_{44} \left( \sum M_2[J_{(1:3,1:3)}] \right) = [J_{44}]^2 \det[J_{(1:3,1:3)}].
\]

The last term \( (\sum M_3(J))^2 \) gives only a contribution to the coefficient of \( J_{44}^2 \) and therefore has not been considered. The coefficient of \( J_{44}^2 \) is thus

\[
c_3 = [J_{11} + J_{22} + J_{33}] \left( \sum \tilde{M}_2 \right) - \det[J_{(1:3,1:3)}].
\]  
(5.38)

The term not containing \( e \) is

\[
c_0 = \sum_i M_2[J_{(1:3,1:3)}] \det[J_{(1:3,1:3)}] - [J_{11} + J_{22} + J_{33}] \left( \sum (-1)^i \tilde{M}_3[J_{i4}] \right),
\]

\( \tilde{M}_3[J_{i4}] \) being the minor of the element \( J_{i4} \) in \( J \). The existence condition of \( e^1 > 0 \) for which (5.33) holds is then

\[
c_0 c_3 < 0.
\]  
(5.40)

To detect a Hopf bifurcation we need then only worry about the transversality condition. The roots of (5.33) are in general of the form

\[
\lambda_1(e) = \xi_1(e) + i\xi_2(e), \quad \lambda_2(e) = \xi_1(e) - i\xi_2(e),
\]  
(5.41)

\[
\lambda_{\pm}(e) = \frac{1}{2} \left[ -b_3 \pm \sqrt{b_3^2 - \frac{4}{b_1} b_0 b_3} \right].
\]

Differentiating the characteristic equation with respect to \( e \) and denoting this by a prime, we get

\[
\gamma_{11} \xi_1' - \gamma_{12} \xi_2' = \gamma_{10}, \quad \gamma_{12} \xi_1' + \gamma_{12} \xi_2' = \gamma_{20},
\]

where

\[
\gamma_{11} = 4\xi_1^2 - 12\xi_1\xi_2^2 + 3\beta(\xi_1^3 - \xi_2^3) + 2(\eta^2 + \zeta)\xi_1 + \eta^2 \beta
\]

\[
\gamma_{12} = 12\xi_1^2\xi_2 - 4\xi_2^2 + 6\beta\xi_1\xi_2 + 2(\eta^2 + \zeta)\xi_2
\]

\[
\gamma_{10} = \beta'(3\xi_1^2 - \xi_1^4) + [(\eta^2)' + \zeta'](\xi_2^2 - \xi_1^2)
\]

\[
- \xi_1[(\eta^2)' + \zeta'] - (\eta^2)'\zeta + \eta^2 \zeta'
\]

\[
\gamma_{20} = \beta'(\xi_2^2 - 3\xi_1^2\xi_2) - \xi_1\xi_2((\eta^2)' + \zeta') - \xi_2((\eta^2)' + \eta^2 \beta').
\]
On solving for $\xi'_1$ and $\xi'_2$ in the above system, we find

$$\xi'_1 = \frac{\gamma_{10} \gamma_{11} + \gamma_{20} \gamma_{12}}{\gamma_{11} + \gamma_{22}}, \quad \xi'_2 = \frac{\gamma_{20} \gamma_{11} - \gamma_{10} \gamma_{12}}{\gamma_{11} + \gamma_{22}}.$$ 

Letting

$$m^\dagger_2 = \sum M_2[J(1:3,1:3)(e^\dagger)], \quad m^\dagger_3 = \sum M_3[J(e^\dagger)]$$

$$t^\dagger = \text{tr}[J(e^\dagger)] \quad d^\dagger = \text{det}[J(e^\dagger)],$$

the transversality condition $\xi'_1(e^\dagger) \neq 0$ in terms of the original entries in the Jacobian matrix is

$$(d^\dagger + m^\dagger_2)(m^\dagger_2 t^\dagger - m^\dagger_3)(t^\dagger)^4 + 2(m^\dagger_3)^4[(t^\dagger)^2 + (m^\dagger_3)^2] \neq 0.$$ (5.42)

Numerical simulations show that the Hopf bifurcation does indeed arise, so that sustained oscillations are possible. Figure 5.9 supports our statement.

**FIGURE 5.9:** Sustained oscillations for the ballooning model (5.9); ecosystem survival.
5.3.1 Spraying effects and human intervention

Again we can investigate the results of insecticide spraying, similarly as done before. Observe that in general the vineyard is assumed to be sprayed from planes flying not too high above the ground and aiming at the vineyards, but due to the wind effect the insecticide may land on the woods as well. Also as already mentioned, the poison is meant to act on the insects and pests of the vineyards, but it may very well affect the spiders, too. Let $T_i$ denote the spraying instants, focusing on the “instant killing effect” of the poison; we thus obtain, as for the wanderer spiders models, the modified model

$$\frac{dw}{dT} = w \left[ b \left( 1 - \frac{w}{W} \right) - \ell s_w \right] - h (1-q) \delta (T_i),$$

$$\frac{dv}{dT} = v \left[ a \left( 1 - \frac{v}{V} \right) - ks_v \right] - hq \delta (T_i),$$

$$\frac{ds_w}{dT} = s_w \left[ -c + \tilde{\ell} w \left( 1 + \alpha \left( -\frac{V}{V+W} \right) \right) \right]$$
$$+ \frac{W}{V+W} \tilde{k} v s_w \alpha - K (1-q) \delta (T_i),$$

$$\frac{ds_v}{dT} = s_v \left[ -e + \tilde{\ell} v \left( 1 + \alpha \left( -\frac{V}{V+W} \right) \right) \right]$$
$$+ \frac{V}{V+W} \tilde{\ell} w s_w \alpha - K q \delta (T_i),$$

with parameters that have the same meaning as introduced in the corresponding subsection. Again, as the Dirac delta function suddenly pushes downward the solutions at the instants $T_i$, if such perturbation is large enough and if a basin of attraction of some other stable equilibrium exists, the trajectories tend toward this alternative equilibrium instead of the former. Thus the human intervention may very well destabilize the ecosystem, as already remarked in case of the wanderer spiders models.

5.3.2 Ecological discussion

We have discovered that the model presented contains three relevant equilibria that are conditionally stable, $E^{(3)}$, $E^{(4)}$, and $E^{(5)}$. In both at least one population vanishes. In the second, the wood insects disappear, causing loss of diversity. Thus this equilibrium should be avoided. The situation for $E^{(3)}$ is even worse, as it is a spider-free equilibrium, leading to the disappearance of predators and survival only of possibly harmful insects. $E^{(5)}$ is desirable as it contains no vineyard-living prey. From the farmer’s point of view, it is the equilibrium one should strive for. Finally, looking at the problem from the perspective of the ecologist, $E^* \equiv E^{(6)}$ shows the whole ecosystem to thrive, which is good for environmental biodiversity.
Stability of all these equilibria is always conditional, for $E^{(3)}$ being given by upper bounds on the woods and vineyard insects’ carrying capacities, for $E^{(4)}$ and $E^{(5)}$ being ensured by keeping both spider populations high enough.

Around the ecological coexistence equilibrium $E^* \equiv E^{(6)}$ a Hopf bifurcation has been shown to arise, with sustained oscillations in time for all the model populations. Its consequences may be relevant for the ecosystem, since if the periodic solutions have too large amplitude, they may end up on one of the coordinate axes and therefore indicate the disappearance of at least one of the species and migration of the solution trajectories of the dynamical system toward one of the other equilibria, with possibly unpredictable consequences for the environment. This is to be noted, as some external unforeseeable circumstances, such as fast climatic variations, may very well result in a change of the spiders’ death rate and lead to such phenomenon.

Similar remarks as for the previous models on the effects of human intervention with spraying hold here as well. In the case of a real application, a thorough study of the basins of attractions of the various stable equilibria is needed to find the impact of human intervention.

Field investigations demonstrate that phytophagous pests may have economically relevant outbreaks after treatments with pesticides that caused a reduction of the spiders feeding on them (see, e.g., Birnie et al. (1998), Putman and Herne (1966), Hukusima and Kondo (1962)), even to become extinct if insecticide is too high or the applications are too frequent (Mansour et al., 1980). The spider population reappears, however, if the interval between sprayings is sufficiently long.

Much richer and much more complex computer models aimed at the Danish farmland are those by Thomas et al. (2003), Thomas et al. (1990), Topping and Sunderland (1994), Topping (1999), and Thorbek and Topping (2005), where the heterogeneous landscape is simulated via a grid of cells for possibly different habitats. Spiders in various states of development are also considered (Topping et al., 2003). All these models, including the ones presented in this chapter, try to provide insights for the management of sustainable agricultural systems, using natural predators for pest control, possibly coupled with meteorological and farming data adapted to the various local conditions.
Chapter 6

Epidemic models

In this chapter we give a brief look at classical epidemics models. For the reader unfamiliar with these topics, this outline is meant as an introduction to some basic terminology, ideas, and equations that will be needed in Chapter 7 and in other parts of the book. Among the many review papers that give a more thorough picture of this field, we mention Hethcote (2000). But of course various chapters are devoted to it in several recent and classical books, for instance, Murray (1989), Anderson and May (1991), Brauer and Castillo-Chavez (2001), and Edelstein-Keshet (1988). The natural outcome of these studies is the determination of rational vaccination policies to prevent epidemics outbreaks and to control infectious diseases propagation (Dieckmann et al., 2002).

We illustrate first the basic classical epidemic models, introducing the terminology for the various subpopulations involved in the dynamical system. Then some more recent models are presented. In Section 6.3, a structured model is presented in which the biological age is accounted for together with the development stage reached by the diseased individuals. The final Section 6.4 presents a case study on an economically relevant disease affecting farmed animals.

6.1 Basic epidemic models

Mathematical epidemiology stems from the papers by McKendrick (1926) and von Foerster (1959), at about the same time of the original researches of Lotka and Volterra in population theory.

Basically, these epidemic models partition the population into several distinct classes, accounting for the individuals that may catch the disease, those that already are infected and can spread the infection, and those that are removed from the population. This last class can be formed by the recognized carriers, who are then quarantined so that they do not further spread the disease among the sound individuals, or by the people that have recovered from the disease, or even by those still affected by it but which are no more able to spread it. An important factor in infectious diseases, the one that
mainly motivates the mathematical investigation of the situation, is the fact that very often the infectious people, those who can infect other individuals, are not recognized as being disease affected, since they do not yet have the symptoms. For instance, people affected by measles and chickenpox can infect other individuals only between the 10th and the 14th days of incubation, and the 11th and the 14th days respectively, and this occurs well before they are recognized as disease carriers. Different other possibilities also arise, such as accounting for an intermediate class of infected but not yet infectious individuals, or individuals that have become infected but for which the disease has not yet fully developed, and they are still in a stage preceding the one of being infectious. Such cases could be modeled for instance by introducing suitable delays in the differential equations governing the dynamics to simulate the incubation period of the disease. Later on in this chapter we will also consider a generalization in which age for the individuals and the “stage” of the disease are explicitly built into the model.

A second reason for the study of mathematical epidemiology is the development of meaningful vaccination policies, with the intent of eradicating the disease. This is possible because the so-called threshold theorems state the conditions under which the disease naturally vanishes.

The diseases we consider at first are the infectious ones, which are transmitted upon contact between the infectious and the susceptible individual. Other situations are possible, such as diseases transmitted by a vector, in general an insect or a worm, or viral diseases.

Now, we are going to list the basic assumptions leading to the classical epidemic model (e.g., see Frauenthal, 1980). They are discussed below and will be further analyzed and removed or changed in the following sections:

a) The disease is transmitted by contact between an infectious individual and a susceptible individual;

b) there is no latent period for the disease, hence a susceptible becomes instantaneously infected upon “successful” contact with a disease carrier;

c) all susceptible individuals are susceptible in the same way;

d) the infected individuals are all equally infectious; and

e) the population under consideration is fixed in size, so no demographics are accounted for in the classical models, this implying that no births or migration occur and all the deaths are taken into account.

A final word concerns the outcome of the disease. There are instances in which the disease is overcome and the individual remains immune for life, such as chickenpox, measles, mumps, and rubella. In other instances the individual recovers from the illness but may catch it again and again, as it happens for example for the common cold. In still more unfortunate cases, the disease, once contracted, cannot be overcome and is carried for life. AIDS represents
such an example for humans, but in a later section we will analyze a specific
eexample related to farming animals.

6.1.1 Simplest models

Consider at first what is called the SI epidemic model; as mentioned pre-
viously, it represents the case of the disease that, once contracted, cannot be
overcome. Let \( I(T) \) represent the sizes of the infectives at time \( T \); see, e.g.,
Hethcote and Levin (1989, p. 204). Let \( \tilde{\sigma}(T) \) denote the per infective capita
rate of contacts leading to new infectives. Taking \( \tilde{\sigma}(T) \equiv \sigma_0 \), a simple balance
consideration similar to the one that led to the Malthus model in population
theory gives
\[
I(T + \Delta T) = I(T) + \sigma_0 I(T) \Delta T,
\]
and letting \( \Delta T \to 0 \), we obtain the simple model with an exponential solution:
\[
\frac{dI}{dT} = \sigma_0 I(T), \quad I(T) = I(0)e^{\sigma_0 T}. \tag{6.1}
\]

Clearly in this model the population is assumed to be infinite in size. In
any case, the prediction is that every individual will eventually contract the
disease, \( I(T) \to \infty \) as \( T \to \infty \). To construct a more meaningful model, let us
now introduce the function \( S(T) \) representing the sizes of the susceptibles. In
this case, we then assume \( S(T) + I(T) = N \), with \( N \) constant, independent of
time. Also, \( \tilde{\sigma} \) now represents the rate of contacts leading to new infectives, per
infective per susceptible. In other words, disease transmission is proportional
to encounters among the individuals of the two subpopulations and the latter
are proportional to the product of their sizes. Another way of viewing the
situation is to say that \( \tilde{\sigma} \), which was formerly constant, is becoming here a
function of time, indirectly through \( S(T) \), namely, \( \tilde{\sigma}(T) \equiv \sigma S(T) \). Thus (6.1)
gets modified as follows:
\[
\frac{dS}{dT} = -\sigma I(T)S(T), \quad \frac{dI}{dT} = \sigma I(T)S(T). \tag{6.2}
\]

On using the constraint on the fixed size total population, we can eliminate
one variable, to obtain the logistic-like equation
\[
\frac{dI}{dT} = \sigma I(T)[N - I(T)], \quad \frac{dI}{T} - \frac{dI}{N - I} = \sigma NdT
\]
whose solution is then
\[
I(T) = I(0)N e^{\sigma NT \left[ N - I(0) \left( 1 - e^{\sigma NT} \right) \right]} = \frac{I(0)N}{N e^{-\sigma NT - I(0)(e^{-\sigma NT} - 1)}} \to N
\]
as \( T \to \infty \). Thus the “final” number of infected individuals equals the total
population size, i.e., again every one catches the disease. Notice also that as
\[ N \to \infty \text{ and } \sigma N \to \sigma_0 \] we find \( I(T) \to I(0)e^{\sigma_0 T} \), i.e., the model (6.2) in the limit as the population size becomes infinite recovers the solution of (6.1). It is also interesting to consider the epidemics curve, defined by how fast the disease propagates, and this is measured by the rate of change of infectives in the population

\[ E(T) \equiv \frac{dI(T)}{dT} = \frac{\sigma(N - I(0))N^2I(0)e^{\sigma NT}}{[N - I(0)(1 - e^{\sigma NT})]^2}. \]

The plots of the solutions and the epidemics curves are contained in Figure 6.1.

\[ \text{FIGURE 6.1: Top: trivial model; left: infectives curve } I(T); \text{ right: epidemics curve } E(T); \text{ bottom: SI model; left: infectives curve } I(T); \text{ right: epidemics curve } E(T). \]

The reason for which the whole population is ultimately affected is the fact that no individual can overcome the illness. Thus we finally allow the disease recovery, and introduce the class of removed individuals \( R(T) \), which come from healing the infected at a per capita rate \( \gamma \). The conservation condition in this case must then be written as \( S(T) + I(T) + R(T) = N \). The complete model is then

\[ \frac{dS}{dT} = -\sigma I(T)S(T), \quad \frac{dI}{dT} = \sigma I(T)S(T) - \gamma I(T), \quad \frac{dR}{dT} = \gamma I(T). \quad (6.3) \]
Notice that the infectives equation can be rewritten as
\[ \frac{dI}{dT} = \sigma I(T) \left[ S(T) - \frac{\gamma}{\sigma} \right], \tag{6.4} \]
and therefore the epidemics outbreak, meaning the rapid increase of the infective numbers, is avoided and the disease disappears if \( S(T) < \frac{\gamma}{\sigma} \), since then the derivative will be negative. \( R_0 \equiv \frac{S(0)\sigma}{\gamma} \) is the basic reproduction number of the disease; if \( R_0 < 1 \) then the epidemics vanishes naturally. On solving formally (6.4) we find
\[ I(T) = I(0) \exp \left[ \sigma \int_0^T (S(t) - \frac{\gamma}{\sigma}) dt \right]. \]

### 6.1.2 Standard incidence

In all the previous models the basic form of the incidence has been borrowed by the considerations made on chemical reactions (e.g., see Gray and Scott, 1990), namely, using the mass action law that takes the rate of encounters between the subpopulations to be constant \( \sigma SI \). However, another form is in common use, named standard incidence. The basic idea is the fact that in a large population one individual cannot encounter all the others all the time; rather, it meets regularly with a small subset and with a larger number of people less frequently. The rate of encounters therefore should reflect this remark, and therefore be an inverse function of the population size. Once again, hidden here is the law of diminishing returns. This way of thinking involves then a nonlinearity, and the constant incidence rate now becomes \( \sigma \equiv \sigma(N) = \sigma_0 \frac{1}{N} \). It thus apparently seems to make things mathematically worse. However, the contrary happens, as it allows us to reformulate the model not using the subpopulation sizes but using their fractions. Indeed, let us define
\[ s = \frac{S}{N}, \quad i = \frac{I}{N}, \quad r = \frac{R}{N}, \quad s + i + r = 1. \tag{6.5} \]

To determine their rates of change, observe for instance that the differentiation and carrying over the resulting fraction and nonlinear incidence into (6.2) gives
\[ \frac{ds}{dT} = \frac{1}{N} \frac{dS}{dT} = \frac{S}{N^2} \frac{dN}{dT} = \frac{1}{N} \frac{dS}{dT} = \frac{1}{N} \sigma(N) IS = \sigma_0 I S N = \sigma_0 i s. \]

As we will see, however, the nice property for which the rates of changes of the subpopulations \( S \) and \( I \) and the fractions \( s \) and \( i \) coincide does not hold in ecoepidemic situations, since in such cases the total population cannot be assumed to be fixed in size. From now on, we identify the constant \( \sigma_0 \) simply with \( \sigma \). As an application, let us consider the classic SIR epidemic model in
its formulation with sizes (Hethcote, 2000):

\[
\frac{dS}{dT} = \mu N - \mu S - \sigma \frac{IS}{N}, \quad \frac{dI}{dT} = \sigma \frac{IS}{N} - \gamma I - \mu I, \quad \frac{dR}{dT} = \gamma I - \mu R. \tag{6.6}
\]

Here the first term on the right in the first equation represents the newborns assumed to be born sound, i.e., belonging to the class of susceptibles. On introducing the fractions as indicated above, the model becomes

\[
\frac{ds}{dT} = \mu - \mu s - \sigma is, \quad \frac{di}{dT} = \sigma is - (\gamma + \mu)i, \quad \frac{dr}{dT} = \gamma i - \mu r. \tag{6.7}
\]

With respect to the population models, notice that in (6.7) the dynamics is confined to the unit simplex, \(0 \leq i + s \leq 1\), i.e., the triangle in the \(si\) phase space with vertices at the origin and at the unit points on the coordinate axes.

For more details on the next considerations, we refer the reader to the review by Hethcote (2000). A more general model is the MSEIR model, which represents a transition toward epidemics models including a demographic effect. The class of passively immune individuals \(M(T)\) is introduced, these being the newborns of mothers that in some way have had the disease, and since they survived it, therefore acquired immunity and transmitted it to their offsprings. The newborns of the susceptible mothers are instead still susceptible, and therefore belong to class \(S\). A possible transition from class \(M\) to class \(S\) is allowed at rate \(\delta\). \(E\) represents the class of exposed individuals, that are infected but not yet infectious, i.e., cannot spread the disease. If \(d\) denotes the death rate for the population and \(b\) its birth rate, the model reads

\[
\frac{dM}{dT} = b(N - S) - (\delta + d)M, \tag{6.8}
\]

\[
\frac{dS}{dT} = bS + \delta M - \beta \frac{SI}{N} - dS,
\]

\[
\frac{dE}{dT} = \beta \frac{SI}{N} - (\epsilon + d)E,
\]

\[
\frac{dI}{dT} = \epsilon E - (\gamma + d)I,
\]

\[
\frac{dR}{dT} = \gamma I - dR,
\]

\[
\frac{dN}{dT} = (b - d)N,
\]

since now the total population size is not constant, and therefore its dynamics must be accounted for. Using fractions with \(s = 1 - m - e - i - r\) and \(q = b - d\)
and introducing the force of infection \( \lambda = \beta i \), it becomes

\[
\begin{align*}
\frac{dm}{dT} &= (d + q)(e + i + r) - \delta m, \\
\frac{de}{dT} &= \lambda(1 - m - e - i - r) - (\epsilon + d + q)e, \\
\frac{di}{dT} &= \epsilon e - (\gamma + d + q)i, \\
\frac{dr}{dT} &= \gamma i - (d + q)r,
\end{align*}
\]

(6.9)

to be studied in the set \( \{ (m, e, i, r) : m \geq 0, e \geq 0, i \geq 0, r \geq 0, m + e + i + r \leq 1 \} \). This model has only one endemic equilibrium, \( E_* \equiv (m_*, e_*, i_*, r_*)^T \), with

\[
\begin{align*}
m_* &= \frac{d + q}{\delta + d + q} \left( 1 - \frac{1}{R_0} \right), \\
e_* &= \frac{\delta(d + q)}{(\delta + d + q)(\epsilon + d + q)} \left( 1 - \frac{1}{R_0} \right), \\
i_* &= \frac{\epsilon \delta}{(\gamma + d + q)(\delta + d + q)(\epsilon + d + q)} \left( 1 - \frac{1}{R_0} \right), \\
r_* &= \frac{\epsilon \delta \gamma}{(\gamma + d + q)(\delta + d + q)(\epsilon + d + q)} \left( 1 - \frac{1}{R_0} \right),
\end{align*}
\]

(6.10)

where the basic reproduction number is related to the equilibrium number of susceptibles,

\[
R_0 = \frac{\epsilon \beta}{(\gamma + d + q)(\epsilon + d + q)}, \quad s_* = \frac{1}{R_0}.
\]

In this case the force of infection becomes positive if \( R_0 > 1 \),

\[
\lambda = \delta(d + q) \frac{R_0 - 1}{\delta + d + q}.
\]

The age structure previously introduced for structured populations in Chapter 4 can also be used in the context of epidemiology, as we will see in Section 6.3. Here we briefly mention an extension of the MSEIR model with age structure using the fractions, but introduced using the population density \( U(a, T) \),
via relationships of the form, e.g., \( m(a, T)U(a, T) = M(a, T) \), to get
\[
\frac{\partial m}{\partial a} + \frac{\partial m}{\partial T} = -\delta m,
\]
\[
\frac{\partial s}{\partial a} + \frac{\partial s}{\partial T} = \delta m - \lambda(a, T)s,
\]
\[
\frac{\partial e}{\partial a} + \frac{\partial e}{\partial T} = \lambda(a, T)s - \epsilon e,
\]
\[
\frac{\partial i}{\partial a} + \frac{\partial i}{\partial T} = \epsilon e - \gamma i,
\]
\[
\frac{\partial r}{\partial a} + \frac{\partial r}{\partial T} = \gamma i,
\]
\[
\lambda(a, T) = b(a) \int_0^\infty \beta(w)i(w, T)e^{-D(w)-qw}dw.
\]

The boundary conditions at age 0 are homogeneous, except
\[
m(0, T) = \int_0^\infty f(w)[1 - s(w, T)]e^{-D(w)-qw}dw,
\]
\[
s(0, T) = \int_0^\infty f(w)s(w, T)e^{-D(w)-qw}dw,
\]
\[
D(a) = \int_0^a d(w)dw.
\]

Here again a steady state can be calculated, but we just mention the basic reproductive number in this case:
\[
R_0 = \int_0^\infty \beta(w)\rho e^{-D(w)-qw}\int_0^w \epsilon e^{(\gamma - \epsilon)u} \int_0^u b(\chi)e^{\epsilon \chi}d\chi du dw.
\]

A Lyapunov function can be determined, taking it in the following form, where the coefficients need to satisfy the following relationships:
\[
L(T) = \int_0^\infty \alpha(a)e(a, T) + \kappa(a)i(a, T)da,
\]
\[
\frac{d\alpha}{da} = \epsilon[\alpha(a) - \kappa(a)], \quad \frac{d\kappa}{da} = \gamma\kappa(a) - \rho\beta(a)e^{-D(a)-qa}.
\]

Finally, observe that an average age \( A \) of infection can be computed, at the endemic steady state, which for the SEIR and SIR models simplifies to
\[
A = E[a] = \int_0^a \alpha(a)e^{-\Lambda(a)-D(a)}da \int_0^\infty \lambda(a)e^{-\Lambda(a)-D(a)}da, \quad \Lambda(a) = \int_0^a \lambda(w)dw.
\]

Many investigations have appeared on vaccination strategies, also in connection with models accounting for demographic changes. By way of example,
we mention here only Greenhalgh (1992) and Pugliese (1990), and refer the reader to the bibliography of Hethcote (2000).

Let us finally mention pulse vaccination (d’Onofrio, 2002), in which at fixed time intervals $T_*$ a percentage of the population $s(nT_*) = (1 - p)s(nT_*)$, $n \in \mathbb{N}_+$ is inoculated and acquires life-long immunity, and therefore migrates to the removed class. The model written for the subpopulation fractions reads

$$\frac{ds(T)}{dT} = m[1 - s(T)] - \beta(t)s_i,$$

$$\frac{di(T)}{dT} = ae(T) - (g + m)i(T),$$

$$\frac{de(T)}{dT} = \beta(t)s(T)i(T) - (a + m)e(T),$$

$$r(T) = [1 - s(T) - e(T) - i(T)].$$

Here $\rho$ represents the fraction of vaccinated susceptibles at times $T_n = nT_*$, $n \in \mathbb{N}_+$, $m$ is the mortality rate $a$ the inverse latent period of the disease, $g$ is the inverse infection period, and the contact rate $\beta$ is either constant or assumed to satisfy $\beta(T+1) = \beta(T)$, $1$ being the time unit. In d’Onofrio (2002), stability of the limit cycle is investigated, and local and global asymptotic stability conditions of the eradication equilibrium are determined.

### 6.2 Other classical epidemic models

The model by Capasso and Serio (1978) was prompted by the cholera epidemics of 1973 in Bari, southern Italy. Here the incidence term in the SIR classical model is modified to take into account the response the susceptibles give when the epidemics starts to spread. Namely, they take measures not to be infected. This occurs when the number of infectives is high, and therefore it is replaced by a function of $I$. Two possible choices for the latter are possible. At first we can consider a saturation function, which reaches a saturation level with increasing $I$. But also, to model the psychological response of people, the incidence for low values of $I$ can be taken increasing to a maximum and then decreasing if $I$ increases further. A possible function that behaves this way could be provided for instance by the gamma function, $g(I) = I^{\alpha}e^{-\alpha I}$.

The model reads

$$\frac{dS}{dT} = -g(I)S, \quad \frac{dI}{dT} = g(I)S - \gamma I, \quad \frac{dR}{dT} = \gamma I.$$

In Capasso and Serio (1978) results on positivity, global existence, uniqueness, stability, and a threshold-like theorem are shown. The latter states that equilibria with $S \leq \rho^*$ are stable, with $\rho^* = \frac{1}{g'(0)}$. For global stability, a Lyapunov
function is explicitly constructed,
\[ L(S, I) = I + (S - \rho^*)H(S - \rho^*), \]
where \( H(x) \) denotes the Heaviside function. A further modification is then studied,
in which an emigration term for susceptibles is introduced, thereby obtaining
the model
\[ \frac{dS}{dT} = -g(I)S - \lambda S, \quad \frac{dI}{dT} = g(I)S - \gamma I, \quad \frac{dR}{dT} = \gamma I + \lambda S. \quad (6.14) \]

A model that includes a nonlinear incidence is considered in Liu et al. (1986), Liu et al. (1987), in which in simplified form we can write
\[ \lambda(S, I) = \lambda I^p S^q. \quad (6.15) \]

The model considered in Liu et al. (1986) is a SEIRS-type model, which reads for the fractions of the various subpopulations
\[ \frac{dS}{dT} = -\lambda I^p S^q + \mu - \mu S + \delta R, \quad (6.16) \]
\[ \frac{dE}{dT} = \lambda I^p S^q - (\epsilon + \mu)E, \]
\[ \frac{dI}{dT} = \epsilon E - (\gamma + \mu)I, \]
\[ \frac{dR}{dT} = \gamma I - (\delta + \mu)R, \]
\[ S + E + I + R = 1. \]

Another model is considered by Beretta and Kuang (1998), in which the infection is assumed to be caused by a viral agent, and the latter is explicitly built into the system equations. Let \( P \) be the viral population and \( N \) be the bacterial population that may be infected by the virus, and thus as usual is split into susceptibles \( S \) and infected \( I \). Only the susceptibles are assumed to reproduce according to a logistic equation, with carrying capacity \( K \) and to which also the infected contribute in the population pressure term. There is a latent period in which the viruses replicate inside the infected bacteria, after which lysis occurs, i.e., the bacteria die and their virus contents, \( b \) on average, are released into the environment, where the conditions are not ideal for their survival, so that the viral population experiences there a high death rate \( \mu \). The equations then read
\[ \frac{dS}{dT} = rS \left(1 - \frac{N}{K}\right) - \lambda SP, \quad (6.17) \]
\[ \frac{dI}{dT} = \lambda SP - \gamma I, \]
\[ \frac{dP}{dT} = \gamma bI - \lambda SP - \mu P. \]

It is interesting to note here that upon summation of the right-hand sides, not all terms containing the incidence vanish. Upon rescaling, the system
becomes
\[
\frac{ds}{dt} = as(1 - i - s) - sp, \quad \frac{di}{dt} = sp - \ell i, \quad \frac{dp}{dt} = -sp - mp + b\ell i. \quad (6.18)
\]

The model is analyzed to find that it has only two equilibria, \(E_0 \equiv (1, 0, 0)\), which is unstable, and the nontrivial equilibrium \(E_f\) with all nonvanishing subpopulations. For the system the set \(\Omega = \{s + i \leq 1, \quad p \leq \frac{1}{m} b\ell\}\) is shown to be positively invariant. The equilibrium \(E_f\) instead is shown to undergo a Hopf bifurcation.

### 6.3 Age- and stage-dependent epidemic system

In this section a step further for modeling the evolution of infectious diseases is presented, whose basic feature lies in an age description of the populations under scrutiny, following what was previously done for an age description of populations. Here we assume that the infection spreads among such structured population. However, the model is more general than that. In fact, since we have the possibility of describing the population by age cohorts, we also use this feature to account for the stage reached by the disease in the infected individuals. We therefore use the biological age for individuals, and introduce the “stage” of the disease, counting the time elapsed since contagion, hopefully up to the recovery. As it is the case for the age-structured populations, the individuals who just became infected are taken into account via boundary conditions. The same holds true for those who have just been recognized as disease carriers.

Before writing the model, we outline the changes needed in the basic assumptions of the classical model, previously stated in Section 6.1. Assumption a will still be maintained. Becoming infected depends on the age of the susceptible; in fact, children and elderly people contract diseases more easily than individuals of intermediate age because mature people have stronger natural defenses. Thus the age structure in the population removes assumption c. This will be suitably taken into account by the governing equations. To make the model closer to reality, we also remove assumption b. In the usual delay models the incubation period is generally taken as a fixed constant. We introduce instead the parameter \(w\) to more adequately describe a variable time incubation period. Thus the elimination of assumption b allows us to model more realistically diseases such as measles and chickenpox. The carriers are not yet recognizable as infected (or infectious), but the ability of spreading the disease now is not the same for every individual, so that hypothesis d also no longer holds. Assumption e will be eliminated at a later stage, recalling that for the classical epidemics the inclusion of demographic dynamics has led
to more general features (Gao and Hethcote, 1992; Mena-Lorca and Hethcote, 1992).

The population we model is described by a nonnegative density $n(a,t)$, a function with compact support for every $t$. As we know already from Section 4.7, the total population size is

$$N(t) = \int_0^\infty n(a,t) \, da.$$  

(6.19)

Let $m(a,N)$ denote the biological mortality of the population; as in Section 4.7, it is assumed to be age dependent as well as a function of the total population size. As in the classical epidemic models, the population is partitioned into the three distinct classes of susceptibles, infected, and removed individuals. Again, these are nonnegative functions $s, i, r$ of their arguments to be introduced below, with compact support at every fixed time. This partitioning is reflected in the following equation, in which the capitalized densities will be defined below:

$$R(a,t) + s(a,t) + I(a,t) = n(a,t).$$  

(6.20)

The density $i(a,w,t)$ gives the number of individuals aged $a$ at time $t$ in the stage $w$ of the disease, i.e., who were infected $w$ units of time earlier. It evolves according to

$$\frac{\partial i}{\partial a} + \frac{\partial i}{\partial w} + \frac{\partial i}{\partial t} + (g(a,w) + m(a,N)) i(a,w,t) = 0.$$  

(6.21)

Here $g(a,w)$ represents the rate at which an infected aged $a$ and in stage $w$ of the disease is recognized as a disease carrier and thus migrates into the removed class. Thus the boundary condition for the latter takes into account the contributions over all the incubation periods:

$$r(a,0,t) = \int_0^\infty g(a,w) i(a,w,t) \, dw.$$  

(6.22)

Notice that apparently the disease is not lethal. However, the case of an infected individual, not yet recognized as a carrier, who dies of the epidemics, will be accounted for as a death in the class of removed individuals in the stage 0 of the recovery.

The total populations in classes $r$ and $i$ are specified as usual by the integral over all possible recovery stages of their respective densities, which exist by the assumption of $n(a,t)$ being of compact support, since obviously $i(a,w,t), r(a,v,t) \leq n(a,t)$ for every $(a,t)$ and for every $w, v$, the latter denoting the recovery period,

$$I(a,t) = \int_0^\infty i(a,w,t) \, dw, \quad R(a,t) = \int_0^\infty r(a,v,t) \, dv.$$  

(6.23)
Notice also that for $w \geq a$, or $v \geq a$, the above-mentioned densities are identically zero, since the stage of the disease, respectively, of the recovery period, cannot exceed the age of the individual.

The only possible outcomes for an individual in class $r$ are either the transition to class $s$ at rate $q$, or death due to the epidemics at rate $z$, or the disease-unrelated death. Thus, similarly to (6.21), we find

$$\frac{\partial r}{\partial a} + \frac{\partial r}{\partial v} + \frac{\partial r}{\partial t} + (q(a,v) + z(a,v) + m(a,N))r(a,v,t) = 0.$$ (6.24)

An integration over all the recovery stages of the compactly supported density scaled via the function $z$ yields the total number of epidemics-related deaths for individuals aged $a$ at time $t$,

$$D(a,t) = \int_0^\infty z(a,v)r(a,v,t)dv.$$ (6.25)

For the evolution of the susceptibles, notice that, upon recovery, becoming again a susceptible, an individual aged $a$ still remains aged $a$ at the time of the transition. Thus the latter must be accounted for by a nonhomogeneous term, rather than by a boundary condition. A departure from the susceptible class occurs instead only if there is a biological death, or in case of contagion. The very particular case of death immediately following disease contraction is avoided by stating a loss in stage $r(a,0,t)$. Assumption a of the classical model is used here to write again a homogeneous mixing model. In our case, all the infectives of all ages are assumed to be equally able to spread the disease, if they are in the same stage. Hence we introduce the quantity

$$\tilde{I}(w,t) = \int_0^\infty i(a,w,t)da$$ (6.26)

and find

$$\frac{\partial s}{\partial a} + \frac{\partial s}{\partial t} + s(a,t)\left(m(a,N) + \int_0^\infty p(a,w)\tilde{I}(w,t)dw\right) - \int_0^\infty q(a,v)r(a,v,t)dv = 0.$$ (6.27)

The function $p(a,w)$ is related to the stage $w$ of the infectives that cause the encounter, and the age $a$ of the susceptible in question. The “new” infectives come from the interaction term, i.e.,

$$i(a,0,t) = s(a,t)\int_0^\infty p(a,w)\tilde{I}(w,t)dw.$$ (6.28)

To describe the reproduction of the population, thus removing assumption e of the classical model, we consider as in Section 4.7 the age-dependent maternity function $b(a,N)$. However, here we need to state what the effects of
infected individuals are on offsprings. Infected parents can also affect the offsprings, but this might not at all be related to the epidemics in the sense that children of sick parents may not themselves carry the same disease, but may very well have other abnormalities. In the human case, this would possibly occur to children of a mother infected by rubella, for instance. Taking such situations into account is a hard task. We assume simply that only sound individuals reproduce and at birth newborns are sound. Thus

\[
 n(0,t) = s(0,t) = \int_0^\infty b(a,N) s(a,t) \, da. \tag{6.29}
\]

Alternatively, every individual can reproduce, but again newborns are sound:

\[
 s(0,t) = \int_0^\infty b(a,N) n(a,t) \, da.
\]

Suitable initial conditions must complement the above system, that is,

\[
 i(a,w,0) = I^0(a,w) \quad \text{for } a > w, \tag{6.30}
\]
\[
 r(a,v,0) = R^0(a,v) \quad \text{for } a > v,
\]
\[
 n(a,0) = n^0(a),
\]
\[
 s(a,0) = S^0(a) = n^0(a) - \int_0^\infty R^0(a,v) \, dv - \int_0^\infty I^0(a,w) \, dw.
\]

As is the case for the classical models, summing the governing equations (6.21), (6.24), (6.27) and using also (6.20), we obtain the description of the dynamics of the population. The equation governing the evolution of the density \( n(a,t) \) is then

\[
 \frac{\partial n}{\partial a} + \frac{\partial n}{\partial t} + m(a,N) n(a,t) + D(a,t) = 0, \tag{6.31}
\]

and this allows to deduce that the model is well posed. Notice that this equation differs from the classical Kermack–McKendrick or Von Foerster equation as it contains the migration term, i.e., the total loss due to disease-related deaths. To derive the global existence of the solutions, to apply the techniques of Gurtin and McCamy (1974) described in Section 4.7 requires us to handle this migration term. But an upper bound for \( D(a,t) \) is easily obtained from (6.20):

\[
 I(a,t), \quad R(a,t), \quad s(a,t) \leq n(a,t) \tag{6.32}
\]

for every \((a,t), a \geq 0 \text{ and } t \geq 0\). The natural hypotheses on the functions \( m(a,N) \) and \( z(a,v) \) require them to have a nonnegative infimum for \( a \geq 0, v \geq 0, \text{ and } N \geq 0 \). Also, the maternity function \( b(a,N) \) is assumed to be uniformly bounded above for all \( a \geq 0 \text{ and } N \geq 0 \). Then the global existence result holds for the density \( n \). The same result for the model is deduced from (6.32) and \( I, R, s \) being nonnegative.
6.4 Case study: Aujeszky disease

Aujeszky disease (A.D.), caused by the Herpevirus 1 suis (ADV or SHV-1), although not lethal, affects several animal species, among which the hogs constitute the most relevant one for farmers. This disease is diffused in almost every European country with relevant economic consequences. In highly densely populated areas, the Italian law-required control measures are difficult to implement. In northwest Italy, the area of the towns of Villafalletto and Vottignasco in the Cuneo province within a surface of 38 km$^2$ hosts 90000 animals in 91 farms with density in some instances over 3500 individuals per square kilometer. In view of these facts, for modeling purposes we consider the area as a single giant epidemiologic unit. The seroprevalence dropped from the year 1997, in which the new vaccination law was issued, until year 2000 to reach the minimum value of 31.5%. Then it went up again to 54.7% in 2004 and afterwards remained stable. The mathematical model considered here studies the spread of the disease, with the aim of its eradication, if at all possible, or at least a reduction of the viral circulation.

The field measures give the following values for the measurable parameters. The size of the epidemiological unit is $N = 90000$. Data on the positivity of the farmed animals have been collected; the average natural mortality of sound animals is $\mu_s = 0.084$, the average disease-related mortality is $\mu_i = 0.087$, and the average birth rate is $\rho = 0.107$.

For the remaining parameters appearing in the mathematical model, namely the disease incidence $\beta$ and the (absence of) biosafety $\tau$, simulations have been performed. Among the latter, biosafety measures play an important role. These are represented by devices like continuous fencing of the farm, quarantine stalls, disinfections, checked visitor access, filtering area between the stalls and other farming areas, and so on.

The disease, once contracted, affects the animal for its life. Hence the epidemics model we consider must be of SI type. The population is partitioned among the susceptible and infective classes as usual, but reproduces so that $S + I = N$, but here the population is not constant, rather $N \equiv N(T)$. The model is then described by the equations

$$\frac{dS}{dT} = \rho N - \mu_s S - \beta \frac{SI}{N} - \tau S,$$
$$\frac{dI}{dT} = -\mu_i I + \beta \frac{SI}{N} + \tau S. \quad (6.33)$$

The first equation describing the susceptible evolution contains a reproduction term, coming from the whole population, as also diseased animals reproduce, but all newborns are considered to be sound. It also accounts for the intrinsic natural mortality $\mu_s$ of the susceptibles, the disease incidence term, and finally a term describing the absence of biosafety measures, for which the disease can be caught not by direct contact with a sick animal, but by vectors brought into the farm by external carriers. Notice that $\tau = 0$ corresponds to the
highest safety measures. The second equation contains the disease-related mortality term, and the terms characterizing the new infected individuals: see, for instance, French et al. (1999).

Let us introduce normalized variables. As new variables we take the fractions of sound and infected individuals with respect to the total population,

\[ s = \frac{S}{N}, \quad i = \frac{I}{N}, \]

so that the population constraint becomes the line

\[ \ell : \quad s + i = 1. \]  (6.34)

Since the latter is a function of time, the former will also be and their time derivatives have to take this into account. Thus, for instance,

\[
\frac{ds}{dt} = \frac{1}{N} \frac{ds}{dT} - \frac{dN}{dT} \frac{s}{N^2} = \frac{1}{N} \frac{ds}{dT} - \frac{s}{N^2} \left( \frac{ds}{dT} + \frac{di}{dT} \right) 
\]

and similarly for \( i \). Substituting from (6.33), we find

\[
\frac{ds}{dt} = \mu_s s^2 + (\mu_i - \beta)si - (\mu_s + \tau + \rho)s + \rho, \tag{6.35} 
\]

\[
\frac{di}{dt} = \mu_i i^2 + (\beta + \mu_s)si - (\rho + \mu_i)i + \tau s. 
\]

The equilibria of this system are then given by the equations

\[
\mu_s s^2 + (\mu_i - \beta)si - (\mu_s + \tau + \rho)s + \rho = 0, \tag{6.36} 
\]

\[
\mu_i i^2 + (\beta + \mu_s)si - (\rho + \mu_i)i + \tau s = 0. 
\]

These represent conic sections. In principle, they could be studied to determine the flow in the \( si \) phase plane, but the advantage of the reduced model is that in this plane trajectories are confined to the straight line (6.34). We can just study then the flow on this “normalized population line” with normal \( n = (1, 1)^T \). If it goes up, the angle \( \theta \) that it makes with \( n \) is \( 0 < \theta < \pi \), while if it is downward, the angle will be \( \pi < \theta < 2\pi \). To distinguish the two cases, notice that in the former we have \( \sin \theta > 0 \), while the converse situation holds in the latter. Hence, to calculate the angle \( \theta \) that \( n \) makes with the flow, we can use the cross product

\[
\frac{dx}{dt} \equiv \begin{pmatrix} ds \\ \frac{ds}{dT} \end{pmatrix}^T, \quad \frac{dx}{dt} \times n \equiv \| \frac{dx}{dt} \| \| n \| \sin \theta 
\]

so that

\[
\text{sgn}(\sin \theta) \equiv \text{sgn} \left( \frac{dx}{dt} \times n \right), 
\]
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and we are led to study the function
\[ \Psi \equiv \text{sgn}(\sin \theta) = \text{sgn} \begin{vmatrix} i & j & k \\ 1 & 1 & 0 \\ \frac{di}{dT} & \frac{dj}{dT} & 0 \end{vmatrix} \equiv ||k||\text{sgn} \left( \frac{di}{dT} - \frac{ds}{dT} \right). \] (6.37)

Upon substitution from (6.36) and (6.34) we obtain the equation \( \Psi(s) = 0 \), where explicitly
\[ \Psi(s) \equiv \mu_i(1 - s)^2 + s(1 - s)(2\beta + \mu_s - \mu_i) + s(\mu_s + 2\tau + \rho) - (1 - s)(\rho + \mu_i) - \mu_s s^2 - \rho, \] which is again a parabola. Recall that when such a function is positive, the projection onto the normalized population line of the flow shows that trajectories must move upwards toward increasing values of the infectives, i.e., the epidemic propagates. To assess this behavior, it is clearly easier to determine its zeros first, so that we need to solve the quadratic equation
\[ s^2(\mu_i - \beta - \mu_s) + s(\beta - \mu_i + \mu_s + \tau + \rho) - \rho = 0, \] whose solutions are
\[ s_{1,2} = \frac{-(\mu_s + \tau + \rho + \beta - \mu_i) \pm \sqrt{\Delta_s}}{2(\mu_i - \beta - \mu_s)}. \] (6.38)

These zeros will be real if the discriminant is nonnegative,
\[ \Delta_s \equiv (\beta - \mu_i + \mu_s + \tau + \rho)^2 + 4\rho(\mu_i - \beta - \mu_s) \geq 0. \] (6.39)

We need then to determine the region in the \( \beta \tau \) parameter space in which this inequality is satisfied. Substituting the data for the known parameters and studying the equation corresponding to (6.39), we find
\[ \phi(\beta, \tau) \equiv \beta^2 + \tau^2 - 0.22\beta + 0.208\tau + 2\beta\tau + 0.0121 = 0. \] (6.40)

To understand its nature, let us consider the matrix of the quadratic form (6.40),
\[ A \equiv (a_{j,\ell}) = \begin{pmatrix} 0.0121 & 0.104 & -0.11 \\ 0.104 & 1 & 1 \\ -0.11 & 1 & 1 \end{pmatrix}, \quad j, \ell = 0, 1, 2. \]

We then investigate the invariants of (6.40):
\[ \Delta \equiv \det A = -0.045796, \quad \delta \equiv \begin{vmatrix} 1 & 1 \\ 1 & 1 \end{vmatrix} = 0. \]

Thus \( \phi \) is a parabola. The slope of its axis is
\[ \tan \alpha = -\frac{a_{1,1}}{a_{1,2}} = -1, \]
i.e., the angle it makes with the $s$-axis is $\frac{3}{2}\pi$. The vertex can be obtained by solving the system

$$a_{1,1}x_0 + a_{1,2}y_0 + (a_{1,1}a_{0,1} + a_{1,2}a_{0,2})\frac{1}{S} = 0,$$

$$\left[a_{0,1} + (a_{0,1}a_{2,2} - a_{1,2}a_{0,2})\frac{1}{S}\right]x_0$$

$$+\left[a_{0,2} + \frac{1}{S}(a_{1,1}a_{0,2} - a_{0,1}a_{1,2})\right]y_0 + a_{0,0} = 0,$$

to give the point $(x_0, y_0) = (-0.031, 0.0253)$. The intersections with the $\beta$ coordinate axis are complex, while with the $\tau$-axis they reduce to a double root, namely, $\beta_1 \equiv \beta_2 = 0.11$. We also determine the intersections with the line $\beta = 1$ to find the equation

$$\tau^2 + 2.208\tau + 0.7921 = 0$$

with roots $\tau_1 = -1.757$, $\tau_2 = -0.451$. There are no real intersections with $\tau = 1$. From these considerations it follows that the discriminant is always positive in the unit square $\{0 \leq \beta \leq 1\} \times \{0 \leq \tau \leq 1\}$. Thus the discriminant $\Delta_s(\beta, \tau) \geq 0$ for all $0 \leq \beta \leq 1, 0 \leq \tau \leq 1$ and the roots (6.38) are

$$s_{\pm} \equiv s_{1,2} = \frac{\beta + \tau + 0.104 \pm \sqrt{(\beta + \tau + 0.104)^2 + 0.428(0.003 - \beta)}}{2(\beta - 0.003)},$$

always real and distinct. Now, for $\beta > 0.003$ we find $\Delta_s < \beta + \tau + 0.104$, so that $0 < s_-, s_+$, while for $\beta < 0.003$ we find $\Delta_s > \beta + \tau + 0.104$ and thus $s_+ < 0 < s_-$. The roots position with respect to the upper bound 1 can be directly checked, and the final inequalities we have are $0 < s_- < 1 < s_+$ for $\beta > 0.003$, and $s_- < 0 < s_+ < 1$ for $\beta < 0.003$.

Remembering that $\theta$ is the angle of the flow with the normal to the population line, then $\Psi(s) = \text{sgn}(|\sin \theta|) \geq 0$ means a downward flow on this line. Also, $\Psi(s) \geq 0$ holds always outside the roots interval. Thus, for the case $0 < s_- < 1 < s_+$, we have $\Psi(s) \geq 0$ for $s < s_-$ and conversely, so that trajectories approach $s_-$, while for $s_+ < 0 < s_- < 1$, we find $\Psi(s) \geq 0$ for $s_- < s$, again with trajectories approaching $s_-$. This entails that the only feasible equilibrium $s_-$ is always asymptotically stable. In the population space, this equilibrium is then

$$E^* \equiv (s_-, i_-) \equiv (s_-, 1 - s_-)$$

$$\equiv \left(-\frac{(\mu_s + \tau + \rho + \beta - \mu_s) - \sqrt{\Delta_s}}{2|\mu_s - \beta - \mu_s|}, \frac{-((\mu_s + \tau + \rho + \beta - \mu_s) - \sqrt{\Delta_s})}{2|\mu_s - \beta - \mu_s|}\right)$$

$$= \left(\frac{\beta + \tau + 0.104 - \sqrt{\Delta_s}}{2|\beta - 0.003|}, \frac{2|\beta - 0.003| - \beta - \tau - 0.104 + \sqrt{\Delta_s}}{2|\beta - 0.003|}\right),$$
with \( \Delta_s = (\beta - \mu_i + \mu_s + \tau + \rho)^2 - 4\rho|\mu_i - \beta - \mu_s| = (\beta + \tau + 0.104)^2 - 0.428|\beta - 0.003| \).

Now the goal would be to keep down the level of the infected population, or increase the one of the sound animals. If \( \beta \to 0 \), we find

\[
i^*_{\beta=0} = \frac{0.006 - \tau - 0.104 + \sqrt{(\tau + 0.104)^2 - 0.428 \times 0.003}}{0.006}.
\] (6.42)

This condition is, however, essentially impossible to attain, as it imposes extremely strict measures within the farmed animals since we want to annihilate the intramural disease incidence. On the other hand, we could impose biosafety measures, to eliminate the imported disease carriers, in which case \( \tau \to 0 \), and we have

\[
i^*_{\tau=0} = \frac{2|\beta - 0.003| - \beta - 0.104 + \sqrt{(\beta + 0.104)^2 - 0.428|\beta - 0.003|}}{2|\beta - 0.003|}.
\] (6.43)

In a more general case, we can try to minimize the surface

\[
i^* \equiv i^*(\beta, \tau)
\] (6.44)

\[
= \frac{2|\beta - 0.003| - \beta - \tau - 0.104 + \sqrt{(\beta + \tau + 0.104)^2 - 0.428|\beta - 0.003|}}{2|\beta - 0.003|}.
\]

Note that the restriction of this surface to the coordinate planes is given by (6.42) and (6.43) above. By cutting this surface with planes parallel to the coordinate ones, we find similar behaviors, namely,

\[
i^*_{\beta=h} = \frac{2|h - 0.003| - h - \tau - 0.104 + \sqrt{(h + \tau + 0.104)^2 - 0.428|h - 0.003|}}{2|h - 0.003|},
\] (6.45)

\[
i^*_{\tau=k} = \frac{2|\beta - 0.003| - \beta - k - 0.104 + \sqrt{(\beta + k + 0.104)^2 - 0.428|\beta - 0.003|}}{2|\beta - 0.003|},
\] (6.46)

at “higher” levels. Note that the line \( \beta = 0.003 \) is a removable singularity, as after simplifications the restriction of the surface to that line is described by the equation

\[
i^*|_{\beta=0.003} = \frac{\tau}{\tau + \rho}.
\]

We can better study the sections of \( i^* \) with \( \tau = k \) by taking the derivative with respect to \( \beta \) to find

\[
\frac{d i^*}{d\beta}|_{\tau=k} (\beta, k) = \frac{1}{2|\beta - 0.003|(|\beta - 0.003|)(\beta + \tau + 0.104 - |\beta - 0.003|)}.| (6.47)
Thus, for $\beta > 0.003$, the numerator of the partial derivative is $\tau + \rho > 0$ and for $\beta < 0.003$ it is $2\beta + \tau + 0.101 > 0$ in the unit square. Thus, the minimum of the surface is to be sought for $\beta = 0$. Also, the sections of $i^*$ with $\beta = h$ can be investigated by taking the derivatives with respect to $\tau$. We have

$$\frac{di^*}{d\tau} \bigg|_{\beta=h} (h, \tau) = \frac{1}{2|h - 0.003|} \left[ -1 + \frac{h + \tau + \cdot04}{\sqrt{(h + \tau + 0.104)^2 - .428|h - 0.003|}} \right].$$

(6.48)

On verifying whether

$$\frac{di^*}{d\tau} \bigg|_{\beta=h} (h, \tau) \geq 0,$$

we find $0.428|h - 0.003| > 0$, which is clearly always true. Thus $i^*_\beta=g(h, \tau)$ is always an increasing function of $\tau$; cf. Figure 6.2. Thus the minimum will be achieved for $\tau = 0$. Combining this information with the former, we just need to check the value $i^*(0, 0) = -0.0613$. The fact that at the origin the surface becomes negative induces us to also investigate the zero level curve $i^*(\beta, \tau) = 0$. Upon simplification we have

$$\beta + \tau = 0.003 + |\beta - 0.003|.$$

For $\beta > 0.003$, we find then $\tau = 0$, while for $\beta < 0.003$, the condition gives the straight line $\tau = 2(0.003 - \beta)$. Below this line, in the small triangle $\nabla$ containing the origin shown in Figure 6.3, the infected become negative, i.e., the infection level drops to zero. This agrees with the findings of Figure 6.2, in which the whole surface over the unit square is shown. In conclusion, the disease can be eradicated if the two parameters $\beta$ and $\tau$ are pushed down to values contained in $\nabla$. Eradication can be achieved by a combination of biosafety measures coupled with measures to contain the horizontal incidence $\beta$ of the disease. These conditions are, however, very difficult to attain, as they impose extremely strict measures within the farmed animals since we want to annihilate the intramural disease incidence. On the other hand, we also impose strict biosafety measures, to eliminate the imported disease carriers.

6.5 Analysis of a disease with two states

In this section we consider an epidemic spreading in a population of fixed size, a disease that has two states, the recoverable mild form and the strong, possibly lethal one. The population is thus subdivided into three classes: the susceptibles $S$, the weakly infected $W$, and the $V$ strongly affected ones. The
FIGURE 6.2: Plot of the surface $I^*(\beta, \tau)$ in the unit square. It becomes negative near the origin.

FIGURE 6.3: Contour line $i^* = 0$; the surface is negative in the triangle $\nabla$ near the origin; compare Figure 6.2 and notice the very small scale $O(10^{-3})$ on both axes.
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 governing equations are then

\[
\frac{dS}{dT} = \rho S - \beta SW - (\gamma_w + \gamma_v)SV + \pi W + \nu V, \quad (6.49)
\]

\[
\frac{dW}{dT} = -\lambda W + \beta SW + \gamma_v SV - \pi W,
\]

\[
\frac{dV}{dT} = \lambda W + \gamma_v SV - \nu V - \mu V, \quad \text{and} \quad N = S + W + V.
\]

The first equation allows reproduction of the susceptibles, whose offsprings are born sound, at the net reproduction rate \(\rho\). It then considers the disease incidence \(\beta\) due to contact with weakly infected individuals, producing again only new weakly infected individuals, and to contact with the strongly affected ones, which, when “successful,” may produce new infected either in the weak or virulent forms, at respective rates \(\gamma_w\) and \(\gamma_v\). Recovery from either infected state is in principle possible, although we suppose that the recovery rate \(\pi\) from the strong infection is much lower than the one from the mild form of the disease, \(\nu\). The dynamics of the infected subpopulations is completed by allowing a transition from the milder to the stronger one at rate \(\lambda\). Finally, the virulent form may be lethal, and that is accounted for by the disease-related mortality rate \(\mu\).

**Equilibria**

The origin is clearly an equilibrium. To find the nontrivial ones, observe that the equilibrium values of susceptibles solve the quadratic

\[
y(x) = \beta \gamma_v x^2 - (\gamma_v \lambda + \beta \mu + \gamma_v \pi + \beta \nu + \gamma_w \lambda) x + (\lambda + \pi) (\mu + \nu),
\]

giving

\[
S_{1,2} = \frac{(\lambda + \pi) \gamma_v + \gamma_w \lambda + \beta (\mu + \nu) \pm \sqrt{\Delta}}{2 \beta \gamma_v}, \quad (6.50)
\]

\[
\Delta = ((\lambda + \pi) \gamma_v + \gamma_w \lambda)^2 + \beta^2 (\mu + \nu)^2 - 2 \beta (\mu + \nu) ((\lambda + \pi) \gamma_v - \gamma_w \lambda) > 0,
\]

where the last condition on the discriminant is imposed to have real solutions. In such a case it easily follows that \(y(0) > 0\) and \(y'(0) = -(\gamma_v \lambda + \beta \mu + \gamma_v \pi + \beta \nu + \gamma_w \lambda) < 0\) imply that both \(S_1\) and \(S_2\) are feasible. For the infected, we find then

\[
V_1 = \frac{\rho}{\mu} S_1, \quad W_1 = \frac{\rho (\lambda + \pi) (\mu + \nu)}{\beta \lambda \mu} - \frac{\rho (\lambda + \pi) \gamma_v + \gamma_w \lambda}{\beta \lambda \mu} S_1, \quad (6.51)
\]

\[
V_2 = \frac{\rho}{\mu} S_2, \quad W_2 = \frac{\rho (\lambda + \pi) (\mu + \nu)}{\beta \lambda \mu} - \frac{\rho (\lambda + \pi) \gamma_v + \gamma_w \lambda}{\beta \lambda \mu} S_2.
\]

Feasibility then requires the following threshold condition for the susceptibles:

\[
S \geq S_t = \frac{(\lambda + \pi) (\mu + \nu)}{(\lambda + \pi) \gamma_v + \gamma_w \lambda}.
\]
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FIGURE 6.4: Possible forms of the function $\sigma^3 - C\sigma^2$, $C > 0$ on the left, $C < 0$ on the right.

which in turn shows that $S_1$ is infeasible, while $S_* \equiv S_2$ instead satisfies it. The feasible equilibria are then

$$E_0 = (0,0,0), \quad E_* = \left( S_*, \frac{\rho(\lambda + \pi)(\mu + \nu)}{\beta \lambda \mu} - \frac{\rho(\lambda + \pi)\gamma_v + \gamma_w}{\beta \lambda \mu} S_* \frac{\beta \mu S_*}{\mu} \right).$$

**Stability**

The Jacobian of the system is

$$J = J(S,W,V) = \begin{pmatrix} \rho - \beta W - (\gamma_w + \gamma_v) V & -\beta S + \pi & - (\gamma_w + \gamma_v) S + \nu \\ \beta W + \gamma_w V & -\lambda + \beta S - \pi & \gamma_w S \\ \gamma_v V & \lambda & \gamma_v S - \mu - \nu \end{pmatrix}.$$ 

Now, the eigenvalues at the origin are easily evaluated as $\sigma_1 = \rho$, $\sigma_2 = -\lambda - \pi$, $\sigma_3 = -\mu - \nu$ to show that it is unconditionally unstable.

The characteristic equation for $E_*$ can be written as

$$-\sigma^3 + C\sigma^2 + B\sigma + A = 0,$$
FIGURE 6.5: $\beta = 0.1$, $\gamma_w = 0.5$, $\gamma_v = 0.5$, $\lambda = 0.3$, $\mu = 0.1$, $\nu = 0.3$, $\pi = 0.3$, and $\rho = 0.4$, giving $A, B, C < 0$ but $A + BC \neq 0$.

FIGURE 6.6: $\beta = 1.0$, $\gamma_w = 1.0$, $\gamma_v = 9.0$, $\lambda = 1.0$, $\mu = 3.5$, $\nu = 0.1$, $\pi = 0.1$, and $\rho = 0.5$. 
with coefficients explicitly given by

\[ A = -\rho S_2[\mu \beta + \gamma_w \lambda + \nu \beta - 2\beta S_2 \gamma_v + \gamma_v \pi + \gamma_v \lambda], \]

\[ B = \frac{1}{\lambda \mu} \left[ -\rho \mu^2 - \lambda^2 \mu \nu - \rho \nu \lambda^2 - \lambda^2 \mu \nu - \rho \lambda^2 \nu + \lambda \mu^2 \beta S_2 \right. \]

\[ \left. - \lambda \mu \nu + \lambda^2 \mu \gamma_w S_2 + 2\mu \gamma_v S_2 - \lambda \mu \beta S_2^2 \gamma_v + \lambda \mu \beta S_2 \gamma_v + \lambda \mu \gamma_v \lambda S_2 \right. \]

\[ \left. - \rho \nu \lambda \mu - 2\rho \nu \lambda \mu \nu - \rho \lambda \gamma_v S_2^2 \right. \]

\[ \left. - \rho \lambda \gamma_v S_2 - 2\rho \lambda \gamma_v S_2^2 - 2\rho \lambda \gamma_v S_2^2 - 2\rho \lambda \gamma_v S_2^2 - 2\rho \lambda \gamma_v S_2^2 \right. \]

\[ + 2\rho \lambda \gamma_v S_2 + 2\rho \lambda \gamma_v S_2^2 - \rho \lambda \gamma_v S_2^2 - \rho \lambda \gamma_v S_2^2 \gamma_w \right], \]

\[ C = -\frac{1}{\lambda \mu} \left[ \rho \nu \lambda \mu - \lambda \mu \gamma_v S_2 - \lambda \mu \beta S_2 + \lambda \mu \nu + \rho \pi \nu \right. \]

\[ \left. - \rho \pi \gamma_v S_2 + \rho \pi \mu + \lambda \mu \pi + \lambda \mu^2 \right]. \]

We can seek the eigenvalues as intersections of the two functions \( B\sigma + A \) and \( \sigma^3 - C\sigma^2 \), observing that the latter has one of the two forms in Figure 6.4. To impose stability, the case \( C < 0 \) is the most favorable one, as we need only to impose also \( A, B < 0 \) to have only roots with negative real parts.

![FIGURE 6.7: Limit cycle verified over a long time interval: \( \beta = 0.003, \gamma_w = 0.2, \gamma_v = 0.9, \lambda = 0.1, \mu = 0.06, \nu = 0, \pi = 0.1, \) and \( \rho = 0.02 \).](image-url)
FIGURE 6.8: The system dynamics for the same parameters as in Figure 6.7 but in long-time simulations: one can readily see persistent oscillations.

FIGURE 6.9: $\beta = 0.003$, $\gamma_w = 0.2$, $\gamma_v = 0.9$, $\lambda = 0.1$, $\mu = 0.06$, $\nu = 0.005$, $\pi = 0.1$, and $\rho = 0.02$. 
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gree polynomials, so that the following condition must hold in addition to $A, B, C < 0$:

$$
\sigma^3 - C\sigma^2 - B\sigma - A = (\sigma^2 + \alpha_1^2)(\sigma - \alpha_2) = \sigma^3 - \alpha_2\sigma^2 + \alpha_1^2\sigma - \alpha_1^2\alpha_2,
$$

$$A + BC = 0.

The analysis is then continued numerically. Specifically, for $\beta = 0.1, \gamma_w = 0.5, \gamma_v = 0.5, \lambda = 0.3, \mu = 0.1, \nu = 0.3, \pi = 0.3, \text{ and } \rho = 0.4$, we find a stable equilibrium; see Figure 6.5.

The equilibrium can be slightly destabilized in the transient behavior; Figure 6.6 gives an example of this behavior.

To find persistent oscillations, we now set $\nu = 0$ and $\beta = 0.003, \gamma_w = 0.2, \gamma_v = 0.9, \lambda = 0.1, \mu = 0.06, \nu = 0, \pi = 0.1, \text{ and } \rho = 0.02$. The results are in Figure 6.7, with a double check on a smaller time range in Figure 6.8.

If we then change the value of $\nu = 0.001$, without changing the other parameter values, the oscillations are found to diminish in amplitude; see Figure 6.9.
Chapter 7

Ecoepidemic systems

In this chapter, we begin to consider ecoepidemic models. These are demographic models accounting for interactions among different populations of the types described in the preceding Chapter 4 in which a disease also spreads. To our knowledge, the first papers that appeared in this field are by Hadeler and Freedman (1989), Venturino (1992, 1994), and Beltrami and Carroll (1994). The name of the field is due to Joydev Chattopadhyay, following an earlier suggestion of the late Ovide Arino. Since those first investigations a number of papers have appeared in the literature. Among the more recent contributions to the field, we mention also Chattopadhyay and Arino (1999), Chattopadhyay et al. (2002), and Arino et al. (2004). In particular these ecoepidemiological models turned useful for the analysis of harmful algal blooms, see Chattopadhyay and Pal (2002) and Chattopadhyay et al. (2002; 2003; 2004). Ecoepidemic models involving more complex phenomena among the populations involved have been investigated with more sophisticated mathematical techniques in Venturino (2001; 2002; 2004; 2006; 2007), Haque and Venturino (2006a; 2006b; 2007), and Keller and Venturino (2007).

Epidemic models with vital dynamics for the population share the three dimensionality of the dynamical system that we have here (cf. Gao and Hethcote, 1992), but ultimately the trajectories will lie on a plane in the phase space, thus allowing the use of classical tools for their study. Unfortunately, this is not the case here.

The chapter is organized as follows. The ecoepidemic models considered here contain mass-action incidence, in the various demographic aspects of this situation. We start from predator prey models in which the disease affects first the predators (Section 7.1), then the prey (Section 7.2), and then the other two cases of competing (Section 7.3) and symbiotic communities (Section 7.4).

7.1 Prey–diseased-predator interactions

Assume first that the epidemics spreads only among predators and cannot be transmitted to the prey by interaction. This assumption is biologically sound for a wide range of diseases. We count only sound prey $U$, sound
predators, $V$, and infected predators, $V_I$, and in this context the total number of predators is expressed by the variable $P = V + V_I$. By combining the classical predator prey model with the mass action SIS epidemic, with the disease only in the predators we obtain the following model, recalling that all the parameters are nonnegative:

\[
\frac{dU}{dt} = U \left[ a(1 - \frac{U}{K}) - cV - \eta V_I \right], \tag{7.1}
\]

\[
\frac{dV}{dt} = V \left[ d + eU - fP - \delta V_I \right] + (\nu + h) V_I,
\]

\[
\frac{dV_I}{dt} = V_I \left[ k + gU - fP + \delta V - \nu - \mu \right].
\]

Notice that here prey do not represent the only food source for the predators, since in their absence, and in the absence of the disease, the sound predators in the underlying classical demographic model do not die out, but tend to the equilibrium value $\frac{d}{f}$.

The first equation gives the dynamics of the prey, the last term accounting for the predation process due to infected predators. We split the effects produced by sound and infected predators, as their respective rates of predation may be different. Indeed, the parameter $\eta$ represents a prey loss due to hunting, but, contrary to our basic assumption, it could also mean the “instantaneous” death of prey by disease contraction. It differs from $c$ since (a) $\eta < c$ models the situation in which sick predators are less able to catch prey or (b) $\eta > c$ would denote that the hunting abilities of sick predators may be unaffected, but prey surviving an attack may catch the disease and die of it. However, this violates our basic assumption, and then we would need to modify the model introducing also the infected prey. Situation (a) of reduced hunting efficiency is then assumed having in mind the large predators hunting using their speed. If disease affected, their running performance would be inferior to sound individuals. We could say that they are able to catch young or old prey at rate $\eta$, but the mature prey not as effectively as they could if they were sound. This then justifies condition (a).

The second and third differential equations express the predators’ dynamics. In summing them we find

\[
\frac{dP}{dt} = dV + eUV - fP^2 + hV_I + kV_I + gUV_I - \mu V_I, \tag{7.2}
\]

i.e., the demographics of this whole population, which depends on the subpopulations $V$ and $V_I$. In the second differential equation we find the basic quadratic model for the predators, and the interaction term due to the disease, involving the infected predators $V_I$. The last terms account for the disease recovery process, which togheter with the former disappear in the sum of (7.2), and the newborns coming from infected predators. The third differential equation contains a similar quadratic model, in this case formulated for infected
predators (notice the $V_I$ term that premultiplies the bracket) and then the disease process with terms of reversed signs. Here also disease-related deaths are accounted for, expressed by the parameter $\mu$.

We further assume $g \neq e$, to allow for $g > e$, that infected predators get more “reward” from a caught prey, as food may be more valuable for them than for sound individuals.

Feeding on other sources takes place in both sound as well as diseased predators. This occurs at different net rates $d$, $h$, and $k$ and the offsprings of sound individuals are sound, this being expressed by the parameter $d$, while those of infected individuals may be sound, at rate $h$, or disease affected; this vertical disease transmission is expressed by the parameter $k$. Notice that in some instances this is a very reasonable assumption.

**Positively invariant sets**

We begin to examine the long-term behavior of the model on a simplified version of (7.1), in that we take $\mu = 0$, i.e., biologically we state that the disease is not that virulent, and furthermore that infected individuals do not reproduce, $h = k \equiv 0$. An important preliminary question to answer before doing any further analysis is possibly to determine conditions for which trajectories will remain confined to a compact set, so that they will not “escape to infinity” i.e., that no species grows without limit.

We outline two theoretical tools for determining positively invariant sets, one geometric and the other analytic.

**Geometric approach**

This method exploits the following property. Since the system is homogeneous, the axes and the coordinate planes are solutions. The fundamental existence and uniqueness theorem for ordinary differential equations ensures then that the trajectories cannot exit from the first orthant. The first tentative is to cut out these invariant sets in the three-dimensional phase space by using a suitable plane $\pi$ in the positive cone, i.e., the first orthant in the space, where $U, U, V \geq 0$. It will then be enough to show that the trajectories enter the tetrahedron defined by the coordinate planes and by this suitably defined plane. This means showing that the angle made by the “outward” normal to this plane with the tangent vectors to the trajectories on the mentioned plane $\pi$ is larger than $\pi/2$; see (7.4) below. The outward normal is the orthogonal line pointing away from the origin. This may be a usable method also applicable to other cases, for two reasons. Since it gives only sufficient conditions for the existence of such invariant sets, by working differently, at times we may indeed obtain different conditions under which bounded trajectories exist. Moreover, if needed, more complex surfaces might also be used, to possibly give alternative sets of sufficient conditions.

The construction starts from a generic plane in the $(U, V, V_I)$ phase space
\[ \tilde{a}U + \tilde{b}V + \tilde{c}V = \tilde{d} \] not crossing the origin, \( \tilde{d} \neq 0 \), and meeting the positive orthant \( \tilde{a}, \tilde{b}, \tilde{c} > 0 \). Dividing by \( \tilde{d} \) we can cast it into the form

\[ \pi : \quad AU + BV + CV_I = 1, \quad A, B, C > 0. \quad (7.3) \]

This plane has thus the outward unit normal \( n = (A, B, C) \). Let the flow of the differential system be denoted by \( x' = (U', V', V'_I) \). This plane cuts out of the positive cone a compact positively invariant set if

\[ n \cdot x'|_\pi \leq 0. \quad (7.4) \]

See the illustrative two-dimensional Figure 7.1 for a graphical representation, in which the plane \( \pi \) is replaced by a straight line.

**FIGURE 7.1:** For the classical prey–predator dynamical system, the auxiliary line \( \pi : U + V = 10 \) is shown along with its gradient \( \nabla \pi = (1, 1) \) (starred line) and the flow \( \left( \frac{dU}{dT}, \frac{dV}{dT} \right) \) (continuous line). The angle of the latter vectors is obtuse, and if it is on the whole portion of the line \( \pi \) in the first quadrant, the flow enters in the triangle, which is then the positively invariant set.
Use (7.3) to eliminate $V_I$ and obtain

$$AU \left[ a - \frac{a}{K} U - eV - \frac{\eta}{C} (1 - AU - BV) \right] + \frac{B}{C} \nu (1 - AU - BV) \tag{7.5}$$

$$+ BV \left[ d + eU - fV - \frac{1}{C} (\delta + f) (1 - AU - BV) \right] + (1 - AU - BV) \left( \delta V + gU - fV - \nu - \frac{f}{C} (1 - AU - BV) \right) \leq 0.$$ 

Rewrite (7.5) as follows:

$$AU \left[ \left( a - \eta + 2f - \eta \frac{f}{C} + \frac{g}{A} + \nu \left( 1 - \frac{B}{C} \right) \right) \right]$$

$$+ U \left( \frac{A}{C} (\eta - f) - \frac{a}{K} + g \right) + V \left( \frac{B}{C} (\eta - f) - (c + \delta - f) \right) \tag{7.6}$$

$$+ BV \left[ \left( d + \frac{f - \delta}{C} - \frac{f}{B} \delta + \nu \left( 1 - \frac{B}{C} \right) \right) \right]$$

$$+ U \left( e - g + \delta \frac{A}{C} \right) + V \delta \left( \frac{B}{C} - 1 \right) + \nu \left( \frac{B}{C} - 1 \right) - \frac{f}{C} \leq 0.$$ 

If we impose all the above coefficients to be nonpositive, the inequality (7.6) follows. This amounts to satisfying the following system of inequalities, the relative equations of which represent surfaces in the $A$, $B$, $C$ space:

$$S_1 : \quad (a + \nu) AC + gC + (2f - \eta) A - \nu B A \leq 0, \tag{7.7}$$

$$S_2 : \quad A (\eta - f) - C \left( \frac{a}{K} + g \right) \leq 0, \tag{7.8}$$

$$S_3 : \quad (f - c - \delta) C + B (\eta - f) \leq 0, \tag{7.9}$$

$$S_4 : \quad (d + \nu) CB + (\delta - f) (C - B) - \nu B^2 \leq 0, \tag{7.10}$$

$$S_5 : \quad (e - g) C + \delta A \leq 0, \tag{7.11}$$

$$S_6 : \quad \delta (B - C) \leq 0, \tag{7.12}$$

$$S_7 : \quad \nu (B - C) - f \leq 0. \tag{7.13}$$

The most complicated inequality to study is the one related to the quadric surface, i.e., (7.7). All the other ones are "cylinders" with axes parallel to one of the coordinate axes, for which it suffices to study the intersection with the plane of the remaining coordinates. Let $M^{(1)}$ be the solution set of the cylinders with axes parallel to the $A$-axis, $M^{(2)}$ be the respective solution set for the $B$-axis, and let us denote by $M^{(3)}$ the solution set of (7.7).

The solution set of $S_7$ is a half-space containing also the solution set of $S_0$. $S_3$ also gives a half-space, but feasibility requires three distinct subcases corresponding to the signs of the coefficients in the inequality $C \leq \frac{f - \eta}{f - c - \delta} B$. The last alternative arising, $c + \delta < f < \eta$, indeed gives an empty intersection with the first quadrant. We thus require either
a) \( f > \max(\eta, c + \delta) \) and the slope in the \( BC \) coordinate plane must be larger than one, i.e., \( c + \delta > \eta \) must hold, giving in conclusion \( f \geq c + \delta \). The intersection with the solutions of (7.12) and (7.13) is nonempty.

b) Alternatively, we impose \( f < \min(\eta, c + \delta) \); the inequality then reverses the sign, and the intersection with the solution sets of (7.12) and (7.13) is always nonempty, given by the smaller of the two sets, i.e., \( C \geq \max \left( 1, \frac{f - \eta}{f - c - \delta} \right) B \).

c) Finally, if the coefficient is negative, \( \eta < f < c + \delta \), the whole first quadrant of the \( BC \) plane is a solution and no additional restrictions are required.

If nondegenerate, the conic (7.10) is a hyperbola, since the invariant given by the sum of the products of the coefficients of squared terms with the coefficient of the mixed product, i.e., \(-\frac{1}{2}(d + \nu) - \nu \times 0\), is negative (e.g., see Bronshtein and Semendyayev, 1964, p. 254). It crosses the origin, with slope 1. Thus a possible nonempty intersection with the solution set of the former inequalities is possible only if its vertical asymptote lies to the right of the \( C \)-axis, i.e., the \( B \) coordinate of its center is positive, \( f > \delta \). Otherwise, a detailed analysis on the slopes of the asymptote and of \( S_6 \) shows that no crossings between the two lines are possible.

The condition \( f > \delta \) found in this case, combined with the alternatives for (7.9), give respectively the possible modified alternatives

\[
f \geq c + \delta > \eta; \quad \delta \leq f \leq \min(\eta, c + \delta); \quad \max(\eta, \delta) \leq f \leq c + \delta. \quad (7.14)
\]

The solution set of inequalities (7.14) is thus the region \( M^{(1)} \).

The cylinders with axes parallel to the \( B \)-axis are given by \( S_5 \) and \( S_2 \). \( S_5 \) gives half the space as a feasible solution if \( g > e \), a cylinder with axes parallel to the \( B \)-axis, while \( S_2 \) has either the whole first quadrant as a solution for \( \eta < f \), or an upper section for \( \eta > f \). In the latter case, combining with \( S_5 \), we always find the nonempty intersection given by \( C \geq \max \left( \frac{\delta}{g - \tau}, \frac{y - f}{g + \tau} \right) A \), the solution set for \( S_5 \) and \( S_2 \), i.e., \( M^{(2)} \).

Applying together the above conditions, the two sets of cylinders \( M^{(1)} \) and \( M^{(2)} \) always give nonempty intersections, since they have mutually orthogonal axes. Thus a feasible solution set for the system of inequalities (7.8)–(7.13) exists.

The quadric (7.7) is in general a hyperbolic paraboloid. Indeed, its invari-
ants are

\[
\Delta \equiv \begin{vmatrix} 0 & -\frac{\nu}{2} & a + \frac{\nu}{2} & f - \frac{q}{2} \\ -\frac{\nu}{2} & 0 & 0 & 0 \\ a + \frac{\nu}{2} & 0 & 0 & \frac{q}{2} \\ f - \frac{q}{2} & 0 & \frac{q}{2} & 0 \end{vmatrix} = \left( -\frac{g\nu}{4} \right)^2 > 0,
\]

\[
\rho_3 \equiv \text{rank} \begin{pmatrix} 0 & -\frac{\nu}{2} & a + \frac{\nu}{2} \\ -\frac{\nu}{2} & 0 & 0 \\ a + \frac{\nu}{2} & 0 & 0 \end{pmatrix} = 2,
\]

\[
\rho_4 \equiv \text{rank} \begin{pmatrix} 0 & -\frac{\nu}{2} & a + \frac{\nu}{2} & f - \frac{q}{2} \\ -\frac{\nu}{2} & 0 & 0 & 0 \\ a + \frac{\nu}{2} & 0 & 0 & \frac{q}{2} \\ f - \frac{q}{2} & 0 & \frac{q}{2} & 0 \end{pmatrix} = 4.
\]

Instead of a complete study of the quadric, we provide conditions to ensure that the solution set of (7.7) has a nontrivial intersection with the solution set \(M^{(1)} \cap M^{(2)}\) of the former ones, (7.8)–(7.13), by investigating the restrictions of \(S_1\) with planes parallel to the coordinate planes.

For \(A = k > 0\), the inequality depends on a straight line, \(C \leq k \frac{\nu B + (\eta - 2f)}{(a + \nu)k + g}\), whose ordinate at the origin is positive if \(\eta \geq 2f\). This ensures a nonempty intersection with the solution set \(M^{(1)}\). Should this ordinate at the origin instead be negative, an empty intersection with \(M^{(1)}\) always exists, as the slope is always smaller than 1, \(\frac{\nu k}{(a + \nu)k + g} < 1\). Combining with (7.14), for which the first and last conditions have to be neglected, we obtain the second equation in (7.15).

Restricting \(S_1\) to \(B = l \geq 0\) gives instead the inequality ruled by the hyperbola \(C \leq L \frac{\nu B + (\eta - 2f)}{(a + \nu)l + g}\). A feasible solution in the first quadrant of the \(AC\) plane exists only if \(\eta + \nu l \geq 2f\). But the latter follows from \(\eta \geq 2f\), which is true because it was derived above. The slope at the origin is \(C'(A)|_{A=0} = \frac{1}{g} (\eta - 2f + \nu l)\). If it is larger than the biggest of the two slopes of \(S_2\) and \(S_5\), a nonempty intersection with \(M^{(2)}\) can be found. Thus we need \(\frac{1}{g} (\eta - 2f + \nu l) \geq \max \left( \frac{\delta}{g - e}, \frac{\frac{\nu k}{(a + \nu)k + g}}{\eta - f} \right)\). The fractions on the right are positive, since \(g > e\) and \(\eta > f\). In these conditions, a feasible solution for inequality (7.6) can then be found, thus giving the second equation in (7.15) by pushing down \(l\) to its lower possible bound, \(l = 0\).

In summary, for the SIS model with \(\nu \neq 0, h = 0, k = 0, \mu = 0\), a compact positively invariant set exists if the next conditions are satisfied:

\[
g > e, \quad \delta \leq f \leq \min \left( \frac{\eta}{2}, c + \delta \right), \quad \frac{1}{g} (\eta - 2f) \geq \max \left( \frac{\delta}{g - e}, \frac{\frac{\nu k}{(a + \nu)k + g}}{\eta - f} \right) \tag{7.15}
\]
Notice that since \( \nu \) does not appear in the inequalities just found, the sufficient conditions for the existence of a compact positively invariant set for the SI model with mass action incidence coincide with the above ones. Moreover, since (7.15) are a number of inequalities smaller than the number of parameters appearing in them, they can be satisfied very likely. But should they be violated, no feasible region found with this method would then exist, i.e., bounded by a plane. It may, however, be provided by the choice of another appropriate surface instead of the plane \( \pi \), still satisfying (7.4). In any case, one should always remember that there are cases when the approach may fail to give a suitable set of parameters.

**Analytic approach**

The approach we are presenting here applies to the model (7.1) without the simplifying assumptions, so that in this paragraph, we can still take \( h \neq 0, k \neq 0, \mu \neq 0 \). Let us define the new function \( \Psi = U + V + V_I \), i.e., the total population living in the environment. Using \( \kappa \geq 0 \), and adding \( \kappa \Psi \) to the sum of the right-hand sides of (7.1) gives

\[
\frac{d\Psi}{dT} + \kappa \Psi = \chi(U, V, V_I),
\]

where we have, recalling that \( P = V + V_I \),

\[
\chi(U, V, V_I) \equiv (a + \kappa)U + (d + \kappa)V + (h + k + \kappa - \mu)V_I - \frac{a}{K}U^2 \\
- U(cV + \eta V_I) + U(eV + gV_I) - f(V + V_I)P \\
= (a + \kappa)U - \frac{a}{K}U^2 + (d + \kappa)V + (h + k + \kappa - \mu)V_I \\
+ U[(c - e)V + (g - \eta)V_I] - fV^2 - fV_I^2 - 2fVV_I.
\]

Assume \( c > e, \eta > g \) to get an upper bound

\[
\chi(U, V, V_I) \leq (a + \kappa)U - \frac{a}{K}U^2 + (d + \kappa)V \\
+ (h + k + \kappa - \mu)V_I - fV^2 - fV_I^2 \\
= (a + \kappa - \frac{a}{K})U + (d + \kappa - fV)V \\
+ (h + k + \kappa - \mu - fV_I)V_I.
\]

The three parabolas in (7.17) are bounded above by the heights of their respective vertices, so that

\[
(a + \kappa - \frac{a}{K})U \leq \frac{(a + K)^2}{4a} = H_1, \quad (d + \kappa - fV)V \leq \frac{(d + \kappa)^2}{4f} = H_2, \\
(h + k + \kappa - \mu - fV_I)V_I \leq \frac{(h + k + \kappa - \mu)^2}{4f} = H_3,
\]
and it then follows

\[ \frac{d\Psi}{dt} \leq -\kappa \Psi + (H_1 + H_2 + H_3) \equiv -\kappa \Psi + H. \]

Solving, from Gronwall’s inequality, we find

\[ \Psi(t) \leq \Psi(0) e^{-\kappa t} + \frac{H}{\kappa} (1 - e^{-\kappa t}) \leq \max(\Psi(0), \frac{H}{\kappa}). \quad (7.18) \]

More generally, we could try to study the nature of the function \( \chi \) on the right-hand side of (7.16), i.e., what kind of quadric surface it is, and determine the largest point, i.e., the point farthest from the origin. Indeed, such a surface can be seen as a level surface of a 4-dimensional hypersurface, the maximum of which will correspond to its largest value.

**Equilibria analysis**

We now study of the linearized system (7.1) with \( h = k = \mu = 0 \), near the equilibria, whose superscript in the notation emphasizes that they belong to the “mass”-action incidence model. We find the following points:

\[ E^{(m)}_0 \equiv (U_0, V_0, V_{10}) \equiv (0, 0, 0), \]

with eigenvalues \( a, d, -\nu \), which show it to be unstable, and

\[ E^{(m)}_1 \equiv (U_1, V_1, V_{11}) \equiv \left( \frac{af - cd}{\frac{aKf + ce}{\frac{aKf + ce + aKd + ce}}}, 0 \right), \]

with the feasibility condition \( af \geq cd \). The matrix of the linearized system factors, to give the eigenvalue

\[ \left( \delta - f \right) \left( aKf + ce \right) - \nu \left( \frac{aKf + ce}{\frac{aKf + ce + aKd + ce}} \right) + g \left( af - cd \right); \]

the other eigenvalues are the roots of the quadratic polynomial \( P(\mu) \equiv \mu^2 + \mu b_1 + b_2 \), with

\[ b_1 = \frac{aK}{f} (fa - cd) + f \left( \frac{ae + \frac{aKd}{f + ce}}{\frac{aKf + ce}{\frac{aKf + ce + aKd + ce}}} \right), \quad b_2 = \frac{af - cd}{\frac{aKf + ce}{\frac{aKf + ce + aKd + ce}}}. \]

Because of the feasibility condition, both coefficients are nonnegative, \( b_1, b_2 \geq 0 \), implying that, if we exclude particular cases, both roots of the quadratic have a negative real part. The stability condition is thus given by the negativity of the first eigenvalue, i.e.,

\[ gU_1 + (\delta - f) V_1 < \nu. \quad (7.19) \]
This equilibrium is a focus if the discriminant of the quadratic \( P(\mu) \) is negative. Notice that for \( \nu = 0 \), i.e., for the SI case, if feasible, the equilibrium is unstable for \( \delta \geq f \):

\[
E_2^{(m)} \equiv (U_2, V_2, V_t) \equiv \left( 0, \frac{d}{T}, 0 \right),
\]

with eigenvalues

\[
\frac{af - cd}{f}, \quad -d, \quad \frac{d\delta - d - \nu}{f}.
\]

It is stable if \( af < cd \) and \( d\delta < f(\nu + d) \).

\[ E_3^{(m)} \equiv (U_3, V_3, V_t) \equiv (K, 0, 0) \]

with eigenvalues

\[
-a, \quad \left( ac + \frac{a}{K}d \right) \frac{K}{a}, \quad \left( ga - \nu \frac{a}{K} \right) \frac{K}{a},
\]

from which its instability follows:

\[ E_4^{(m)} \equiv (U_{4.5}, V_{4.5}, V_{14.5}) \equiv \left( 0, V_{4.5}, [V_{4.5}(\delta - f) - \nu] \frac{1}{f} \right). \]

Here \( V_{4.5} \) solves the quadratic equation \( \phi(V) \equiv \delta^2 V^2 - (df + 2\nu\delta) V + \nu^2 = 0 \). For feasibility, observe that since the discriminant reduces to \( df(d + 4\nu\delta) \geq 0 \) and \( \phi(0) = \nu^2 \geq 0, \phi'(0) = -(df + 2\nu\delta) \leq 0, \phi''(0) = 2\delta^2 \geq 0 \), the trinomial has two nonnegative roots. Thus \( V_{4.5} \geq 0 \) is ensured. For \( \delta \leq f \), \( V_{14.5} \geq 0 \) is impossible. Thus \( \delta > f \) is necessary for feasibility, and \( V_{14.5} \geq 0 \) gives a lower bound on \( V_{4.5} \), i.e., \( V_{4.5} \geq \frac{\nu}{\delta} \). As \( \phi\left( \frac{\nu}{\delta} \right) = \frac{f\nu}{(\delta - f)^2} (f \nu - d(\delta - f)) \), if \( d(\delta - f) < f\nu \), for feasibility of both equilibria we need to impose \( \phi'\left( \frac{\nu}{\delta} \right) = \frac{\nu}{\delta} (d(f - \delta) + 2\nu \delta) < 0 \), but the two conditions combined give the contradiction \( 2f\nu < d(\delta - f) < f\nu \); hence both \( E_4^{(m)} \) are infeasible. For \( d(\delta - f) > f\nu \) we have \( \phi\left( \frac{\nu}{\delta} \right) < 0 \), so that only \( E_5^{(m)} \) is feasible. The eigenvalues of \( E_5^{(m)} \) are then given by

\[
-\frac{V_5}{f\nu^2} \left[ \delta^2 V_5 (af + \eta \nu) - af (df + 2\nu \delta) - \nu^2 (c\nu - \delta\eta) \right]
\]

and by the roots of the trinomial \( \theta(\lambda) = f\lambda^2 + \lambda \theta_1 + \theta_0, \theta_1 = \delta(\delta - f) V_{4.5} - (\nu \delta + 2\nu f + df), \theta_0 = df(\delta - f) V_{4.5} + \nu^2 (\nu - \delta V_{4.5}) \). The negativity of the first one is then necessary, again entailing the following lower bound on \( V_5 \):

\[
V_5 > \frac{af(df + 2\nu \delta) + \nu^2 (\delta\eta - cf) + f\eta \nu (d + \nu)}{\delta^2 (af + \eta \nu)} \equiv r^*. \]

This can be discussed as above in terms of the trinomial \( \phi \). The conditions for the sign of the remaining ones are algebraically very much involved; only in
some cases we are able to state some clearcut results. In particular, \( \nu > \delta V_5 \) is impossible, since \( \phi \left( \frac{\nu}{\delta} \right) = -\frac{d\nu}{\delta} < 0 \); we must then have \( \nu < \delta V_5 \), and this entails that \( \theta(0) > 0 \) follows if we require \( d[V_5(\delta - f) + \nu] > \delta V_5 - \nu \).
Since the parabola \( \theta(\lambda) \) is convex, the case \( \theta(0) < 0 \) gives immediately the instability of \( E_5^{(m)} \). We also obtain that \( \phi(p^*) = -\frac{d\nu}{\delta} < 0 \); we must then have \( \nu < \delta V_5 \), and this entails that \( \theta(0) > 0 \) follows if we require \( d[V_5(\delta - f) + \nu] > \delta V_5 - \nu \).

Thus, if we require \( d > \delta \), it follows \( \phi(p^*) < 0 \), i.e., the equilibrium is unstable. To impose \( \phi(p^*) > 0 \), i.e., \( V_5 < p^* \), gives the much stronger requirement \( f\nu \delta + 2f^2\nu d + f^2d^2 < f\nu^2 \delta \). In such a case, however, both roots of the trinomial \( \theta(\lambda) \) are either real and negative, or complex conjugate, but with a negative real part. In this case, thus, \( E_5^{(m)} \) would be stable.

We find then

\[
E_6^{(m),7} \equiv \frac{\psi_2 V^2 + \psi_1 V + \psi_0}{\psi_2 (\delta - f) V_0 - \nu \frac{\nu}{\delta} + g (a - c V_6,7)}.
\]

Here the values \( V_0,7 \) are the roots of the quadratic \( \psi(V) \equiv \psi_2 V^2 + \psi_1 V + \psi_0 = 0 \), where

\[
\psi_2 = e\eta (\delta - f) + fg (\eta - c) + \delta^2 \frac{a}{K} + c (\epsilon f - g\delta) ,
\]

\[
\psi_1 = \delta ga - eaf - \frac{a}{K} df + afg - 2\delta \frac{a}{K} \nu - dhg - e\eta + \nu gc ,
\]

\[
\psi_0 = \nu^2 \frac{a}{K} - \nu ga .
\]

For the SI model \( (\nu = 0) \), as \( \psi_0 = 0, V_0 = 0 \) and there is only one nontrivial equilibrium \( V_7 = -\frac{\psi_1}{\psi_2} \). Feasibility for the SIS model, i.e., for the case \( \nu \neq 0 \), for \( U_{0,7} \geq 0 \), gives

\[
V_{0,7} \leq \frac{af + \eta\nu}{(c - \eta) f + \delta \eta} \equiv V^* \text{ for } \delta \eta + (c - \eta) f > 0. \quad (7.20)
\]

No corresponding limitation exists for \( \delta \eta + (c - \eta) f \leq 0 \) and feasibility is always ensured. We have thus an upper bound on the size of the predator population. For \( V_{16,7} \geq 0 \), we obtain instead the following bounds on the size
of $V_{6,7}$:

$$V_{6,7} \geq \frac{\frac{a}{K} \nu - ag}{(\delta - f) \nu - cg}, \quad \text{for} \quad \frac{a}{K} \nu \geq ag, \quad \frac{a}{K} \delta \geq f \frac{a}{K} + cg, \quad (7.21)$$

$$V_{6,7} \leq \frac{ag - \frac{a}{K} \nu}{cg - (\delta - f) \frac{a}{K}}, \quad \text{for} \quad \frac{a}{K} \nu \leq ag, \quad \frac{a}{K} \delta \leq f \frac{a}{K} + cg.$$  

For

$$\frac{a}{K} \nu \leq ag, \quad \frac{a}{K} \delta \geq f \frac{a}{K} + cg, \quad (7.22)$$

$V_{6,7} \geq 0$ holds automatically, while for $\frac{a}{K} \nu \geq ag$, $\frac{a}{K} \delta \leq f \frac{a}{K} + cg$, it is impossible. To study the bounds (7.21) we consider the trinomial $\psi(V)$, from which we have a quadratic inequality. Concentrating again only on the discussion of sufficient conditions for feasibility, we assume (7.22) to hold, and consider (7.20). Since in this case $\psi(0) = \psi_0 = \nu \left(\frac{a}{K} \nu - ag\right) < 0$, there is exactly one positive root, $V_7$. To satisfy (7.20) it is sufficient to require that $\psi'(V^*) > 0$, a condition that reduces to

$$cf + (2\nu \eta + 2a \delta + \eta d) \frac{\nu c}{ad} + \frac{\eta^2 \nu \delta}{af} \leq \eta(f - \delta) + \frac{\nu^2 \nu}{d} + \frac{1}{ad} (a \delta + \nu \eta)^2 + \frac{\nu^2 \epsilon^2}{ad}. \quad (7.23)$$

Thus (7.22) and (7.23) ensure the feasibility of $E_{6}^{(m)}$. An alternative approach for feasibility consists in assuming both $U_{6,7}^{(m)}$ and $V_{6,7}^{(m)}$ to be feasible, i.e., (7.22) and the inequality

$$\eta \delta + (c - \eta) f \leq 0. \quad (7.24)$$

Then no restrictions on the size of $V_{6,7}^{(m)}$ are necessary, and we need only to require the latter to be nonnegative. This can be achieved by observing that the quadratic form $\psi(V)$ possesses at least one positive root. Since $\psi(0) < 0$, it is enough to require $\psi'' > 0$, which gives

$$\psi_2 > 0. \quad (7.25)$$

We do not study the stability of this equilibrium point.

### 7.1.1 Some biological considerations

Our analysis has shown some sufficient conditions on the parameters ensuring the boundedness of the system trajectories; interestingly, they are independent of the parameters $a, d$. Thus net reproduction rates do not affect the existence of this invariant set.

Also, since the conditions do not depend on $\nu$, the positive invariant set exists for both the SIS ($\nu > 0$) and SI ($\nu = 0$) models. Geometrically, the net
reproduction rates push the trajectories of the dynamical system away from
the origin, but this effect in a sense disappears far away from the origin, a fact
reflected by their absence from the sufficient conditions giving the invariant
set.

Notice also that the condition \( g > e \) is necessary, in this analysis, for the
existence of the invariant set. For \( g \leq e \), indeed, condition (7.11) becomes
impossible and thus the existence of the invariant set is not ensured. However,
by rearranging the grouping of terms, another existence proof might be set up.
The boundedness of the \( f \) parameter in (7.15) shows that the “competition”
term in the predators must not be too low, since it exceeds the infection rate
of the disease, \( \delta \), but also not too large, being limited by the returns in hunting
\( c \) and \( \eta \) obtained by sound and diseased predators.

The points \( E_0^m, E_1^m, E_2^m, E_3^m \) correspond to the equilibria of the underlying
demographic model, and therefore are biologically important as being disease
free. The origin remains an unstable equilibrium also in the ecoepidemic
model. The same feasibility condition for the demographic equilibrium \( E_1 \),
which is also globally asymptotically stable, also holds for \( E_1^m \), but the latter
is stable only if the additional condition (7.19) holds.

As for the prey–predator model, \( E_2^m \) is stable when \( E_1^m \) is infeasible, but
in the ecoepidemic model another condition must be satisfied \( \delta d < f (\nu + d) \),
which for the SI model (\( \nu = 0 \)) becomes \( \delta < f \). \( E_3^m \) is unstable as its under-
lying demographic counterpart.

In the ecoepidemic model there are also new equilibria not appearing in the
classical prey–predator model. No prey survive at \( E_4^m, E_5^m \). Notice that \( E_5^m \) is feasible for \( \delta > f \), i.e., when \( E_2 \) of the classical model is unstable. To make
\( E_5^m \) unstable, the level of the predator equilibrium population, \( V_5 \), should not
exceed the ratio of recovery to contact rates, namely, \( \frac{\nu}{\delta} \). But this is impossible
for the SI model (\( \nu = 0 \)). If \( V_4 \) and \( V_5 \) do not exceed the quantity \( \frac{\nu}{\delta + \eta} \), both
equilibria \( E_4^m, E_5 \) are infeasible.

Feasibility for equilibria \( E_6^m, E_7 \) is very difficult to be interpreted biologically.
But combining the two inequalities (7.22) we find again \( \delta > f \), so that once
more feasibility is related to the instability of the prey–predator equilibrium
\( E_2 \).

The disease is endemic at \( E_5^m \), but the prey are also wiped out. If the goal
is the eradication of an obnoxious pest, one could introduce a diseased predator
in the ecosystem and aim for \( E_5^m \). But the same result could be achieved in
the classical prey–predator model by striving for equilibrium \( E_2 \). Thus the
introduction of the disease seems to be useless.

Let us now attempt a partial answer to one biologically relevant question,
namely, whether the introduction of a prey can eradicate a disease spreading
in the predators. From \( E_4^m \), in case of the SI model, \( \nu = 0 \), we find that \( E_4^{(m)} \)
migrates to the origin, which as we know is always an unstable equilibrium,
but we also have \( V_5^{(m)} = \frac{d}{\nu \eta} \), from which it follows \( V_5^{(m)} \geq 0 \) for \( \delta \geq f \). Thus
$E_5^m$ is feasible, with eigenvalues

$$\frac{1}{\delta^2} \left[ \delta (a\delta - d\eta) + df (\eta - c) \right], \left[ -f \pm \sqrt{f (5f - 4\delta)} \right] \frac{d}{2\delta}.$$

They have negative real parts if $d\eta > a\delta$, $c > \eta$, $4\delta > 5f$. Thus the introduction of the prey, although they in the end disappear, causes the predators to partly survive the epidemics unaffected, since part of the population survives, at $V_5^{(m)} > 0$. Comparing with the SI epidemics model, for which all individuals ultimately become infected, the presence of the prey acts as a “recovery” from the disease, i.e., what the SIS model prescribes. The prey thus allows the survival of a part of the predator population unaffected by the disease.

We conclude the Section by mentioning a result on the more general model allowing disease-related deaths. For small values of $\mu$ the behavior near the interior endemic equilibrium is similar to the one seen above, but increasing its value moderately, a bifurcation occurs; see Figures 7.2 and 7.3. Increasing it further however, the persistent oscillations disappear and the trajectories tend to a different (boundary) equilibrium.

### 7.2 Predator–diseased-prey interactions

We now consider the disease spreading among the prey. We count sound prey $U$, infected prey $U_I$, and sound predators $V$. Since the epidemics spreads only among the former, the system takes the form

$$\frac{dU}{dT} = U \left[ a - bU - cV - \lambda U_I \right] + \gamma U_I, \quad (7.26)$$

$$\frac{dU_I}{dT} = U_I \left[ \lambda U - kV - \gamma \right],$$

$$\frac{dV}{dT} = V \left[ d + eU - fV - hU_I \right].$$

The equations state that the disease can be overcome at rate $\gamma$. The sign of $h$ is understood to be undetermined, to describe interactions possibly leading to quick deaths of predators upon contact, $h > 0$, or predators hunting of infected animals occurs at a different rate $h < 0$ than the one for sound prey.

Let us examine the boundedness of the system trajectories first. Proceeding from the easier SI model, if $h > 0$, the set $[0, U^\circ] \times [0, \infty) \times [0, V^\circ], U^\circ \geq a/b, d + eU^\circ - fV^\circ \leq 0$, is positively invariant in the $U \ U_I \ V$ space.

Indeed, it suffices to determine the sign of $\frac{dU}{dt}$ and $\frac{dV}{dt}$, deleting the terms $-\lambda U_I$ and $-hU_I$ from the equations (3.1). The given conditions ensure that $U' < 0$ on $U = U^\circ$, while the one for $V^\circ$ states that the point $(U^\circ, V^\circ)$ lies in the region $V' < 0$. 
FIGURE 7.2: Persistent oscillations around the endemic equilibrium, for a moderate value of $\mu = 0.2$, top prey, middle diseased predator, bottom sound predators.

FIGURE 7.3: Phase space view of the above limit cycle around the endemic equilibrium, for a moderate value of $\mu = 0.2$. 
For $h < 0$ and $b > k$, instead, an invariant set is given by the finite part of the positive cone cut out by the plane $\pi: AU + BU_I + CV = 1$, where $A, B, C > 0$ satisfy

$$aAB + \lambda(B - A) < 0, \quad \lambda A < B(c + \lambda), \quad hA < B(\lambda - c); \quad (7.27)$$

$$dCB - (kB + hC) < 0, \quad \lambda C < B(b - k), \quad bC < B(f - k).$$

Now we must also consider the term $-hU_I$, but working with the plane $\pi$ as above, imposing that its normal $n \equiv (A, B, C)$ makes an obtuse angle with the flow $x \equiv (U, U_I, V)$, we must then have $n \cdot x'|_\pi < 0$. This becomes

$$UA[(aAB + \lambda(B - A))(AB)^{-1} + V/B(\lambda C + (k - b)B)$$

$$+ U/B(\lambda A - (c + \lambda)B)] + CV[(dCB - (kB + hC))(CB)^{-1}$$

$$+ V/B(hC + (k - f)B) + U/B(hA + (e - \lambda)B)] < 0.$$
and in the $AB$ parameter plane:

\[
B < A(\lambda + \gamma A)/(\lambda + aA), \quad (\lambda - \gamma)A - (b + \lambda)B < 0, \quad (e - \lambda)B + (h - \gamma)A < 0, \quad A < 1.
\]

Examining in detail all possible situations, we are led to the following set of inequalities, giving ranges for the parameters for which (7.28) and (7.29) have solutions. Namely, (7.28) is solvable for $h > 2\gamma$ if either

\[
f > k > c, \quad \frac{\gamma - \lambda}{k - c} > \frac{h - \gamma}{f - k}, \quad \gamma > \lambda
\]

or

\[
k < \min(c, f)
\]

hold. For $2\gamma > h > 0$ solutions exist by picking one set of conditions from either

\[
f > k > c, \quad \gamma > \lambda, \quad \frac{\gamma - \lambda}{k - c} < \frac{h - \gamma}{f - k}
\]

or

\[
k < \min(c, f),
\]

and a second set from either

\[
k > c, \quad \gamma > \lambda, \quad \frac{2\gamma - h}{k} < \frac{\gamma - \lambda}{k - c}
\]

or

\[
k < c.
\]

Finally, for $h < 0$ we need

\[
k > f, \quad \frac{\gamma - h}{k - f} > \frac{2\gamma - h}{k},
\]

and one set from either

\[
k > c, \quad \gamma > \lambda, \quad \frac{2\gamma - h}{k} > \frac{\gamma - \lambda}{k - c}
\]

or

\[
k < c.
\]

The system (7.29) possesses solutions if we take one set of conditions from either

\[
e < \lambda, \quad \gamma < h, \quad \gamma - h < e - \lambda,
\]

or

\[
e > \lambda.
\]

The parameter ranges giving solutions for the system (7.28) need to be paired then with those solving the system (7.29). In particular, notice that
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A set of easily understandable sufficient conditions are given by \( h > 0, k < \min(c, f), e > \lambda \). Thus the system trajectories are bounded in the case of the diseases prey having a strong impact on the predators, if the predation rate on infected prey is less than both the predators’ logistic pressure term and the hunting rate on the sound prey, and the conversion rate of the predators exceeds the disease incidence.

For \( h < 0 \) some sufficient conditions for the existence of a compact positively invariant set are given, for instance, by \( c > k > f, e > \lambda \), which are easily interpretable as suitable rates in the model, and the more complicated condition involving also the recovery rate of diseased individuals:

\[
\gamma(k - f) < f(\gamma - h).
\]

Equilibria and their stability

The origin is easily found to be the first of the equilibria \( E_i \equiv (U_i, U_{1,i}, V_i) \). \( E_0 \equiv O \) has eigenvalues \( a, -\gamma, d \), and thus it is unstable.

Then we have \( E_1 \) with only a nonzero component given by \( V_1 = \frac{d}{f} \). Its eigenvalues are \( -c\frac{d}{f} + a, -k\frac{d}{f} - \gamma, -d \). It is locally asymptotically stable then if \( fa < cd \).

Another equilibrium with only a nonzero component is \( E_2 \), with \( U_2 = \frac{\psi}{d} \). The eigenvalues are \( -\lambda\frac{d}{f} - \gamma, \frac{1}{d}(db + ea) \), thus it is always unstable. The next equilibrium is the one corresponding to the demographic model without disease. Letting \( D = bf + ce \), it is \( E_3 \) with \( U_3 = \frac{\psi}{d}(af - cd) \), \( U_{1,3} = 0 \), \( V_3 = \frac{1}{D}(db + ea) \). It is feasible for \( af \geq cd \). One eigenvalue can be explicitly evaluated as \( \lambda U_3 < kV_3 + \gamma \). The other ones are the roots of the quadratic \( T(\mu) \equiv \mu^2 + \mu b_1 + b_2 \), with

\[
\begin{align*}
b_1 &= -\frac{t_1}{D}, \quad b_2 = -\frac{t_3}{D}, \\
t_3 &= (db + ea)(dc - fa), \\
t_1 &= -fa(b + e) + bd(e - f).
\end{align*}
\]

Explicitly, they are given by

\[
T_{1,2} = [2(ce + fb)]^{-1}[t_1 \pm t_2^{1/2}], \quad t_2 = t_1^2 + 4t_3(bf + ce).
\]

The feasibility condition \( U_3 > 0 \) implies \( t_3 < 0 \), so that \( t_2 < t_1^2 \). Hence both roots of the quadratic have a negative real part. Stability is thus ensured by negativity of the first eigenvalue, \( \lambda U_3 < kV_3 + \gamma \).

The equilibrium \( E_4 \) with all nonnegative components is analyzed only for the \( SI \) model, \( \gamma = 0 \). Here

\[
\begin{align*}
V_4 &= \frac{\lambda}{k}U_4, & U_{1,4} &= \frac{Z}{\Delta}, & U_4 &= \Psi \frac{k}{\Delta}, & \Psi \equiv \lambda d - ah, \\
\Delta &\equiv \lambda(af - hc) - k(bh + \lambda e), & Z &\equiv \lambda(dc - fa) + k(db + ea).
\end{align*}
\]
Feasibility gives either
\[ \Psi > 0, \quad \Delta > 0, \quad Z < 0, \quad (7.30) \]
or
\[ \Psi < 0, \quad \Delta < 0, \quad Z > 0. \quad (7.31) \]
The characteristic polynomial is \( \Xi(\mu) = \mu^3 + a_1\mu^2 + a_2\mu + a_3 \) with
\[
a_1 = \frac{\Psi}{\Delta}(f\lambda + bk), \quad a_2 = \frac{\Psi}{\Delta^2}k\lambda[Z(h - \lambda) + \Psi D], \quad a_3 = -k \left( \frac{\Psi}{\Delta} \right)^2 \lambda Z.
\]
The Routh–Hurwitz criterion gives the following results. If (7.30), then both \( a_1 > 0 \) and \( a_3 > 0 \) are consequences of the existence assumptions and the stability requirement follows if and only if \( a_1a_2 > a_3 \), i.e., explicitly,
\[ (f\lambda + bk)[D\Psi + Z(h - \lambda)] + Z\Delta > 0. \quad (7.32) \]
For the case \( h < 0 \), we can restate it, since the last two terms are equivalent to
\[ Z[(f\lambda + bk)(h - \lambda) + \Delta] = Z \lambda [b(f - c) - k(b + c)]. \]
Then it suffices to ask \( f > c \) for (7.32) to hold. If (7.31) are satisfied, then again \( a_1 > 0 \) follows from the feasibility, but \( a_3 > 0 \) contradicts the assumption \( Z > 0 \). Thus (7.31) never give stable equilibria.

Notice that the equilibrium \( E_4 \) for the SIS model splits to give rise to a pair of equilibria.

Finally, note that for the SI model \( \gamma = 0 \), we also have the following extra equilibrium, \( E_* = (U_*, U_I, *, V_*) \equiv (0, U_{I,*}, 0) \), with an arbitrary value for \( U_{I,*} \). The eigenvalues are \( a - \lambda U_{I,*}, 0, d - hU_{I,*} \). The trajectories then lie on the plane \( U_I = U_{I,*} \). The equilibrium is locally asymptotically stable if \( U_{I,*} \) is large enough, i.e., for \( \frac{a}{\lambda} < U_{I,*}, \frac{d}{h} < U_{I,*} \).

**Summary of findings**

In this model, the instability of the equilibria \( E_0, E_2 \) is inherited from the corresponding equilibria of the underlying predator prey system. The other equilibrium \( E_1 \) on the coordinate planes of the \( U \ U_I \ V \) space is stable if and only if the corresponding point of the underlying prey–predator model is infeasible. Also, \( E_3 \) is stable exactly as the underlying demographic equilibrium, and this occurs if and only if \( E_3 \) is infeasible. Notice that a general feature of ecoepidemic models is that the interior equilibrium of the classical model becomes a boundary equilibrium in the ecoepidemic model, since it lies on one of the the coordinate planes of the phase space. In this case it is the point \( E_3 \).

The model also possesses the nontrivial equilibrium \( E_4 \), in which the disease is endemic. The two species survive at a different level than prescribed by the
underlying demographic model. For stability, the analysis gives the following sufficient condition. The crowding effect of predators should be larger than the predation rate on prey dynamics, at least for the case when the predators do not contract the disease. In addition, the level of both populations can be different than the prescribed values at the nontrivial equilibrium of the classical model by imposing that

\[
\lambda \frac{\Psi}{\Delta} \neq \frac{db + ea}{D}, \quad k \frac{\Psi}{\Delta} \neq \frac{af - cd}{D}.
\]

These are two extra conditions, we already have another three given by (7.30) to ensure existence, and one more for stability, (7.32). Overall, then, there are six conditions to be satisfied, with nine parameters, as \( \gamma = 0 \) here. We can thus conjecture that for a suitable selection of the parameters of the model, the equilibrium is feasible, stable, and at a level not prescribed by the classical prey–predator model. Thus the disease in the prey acts as a controller for the size of the predators. In particular, for the case of pest control, the parameters could be chosen so that the prey equilibrium is at a smaller level than the one provided by the classical model.

### 7.3 Diseased competing species models

In the literature [e.g., see Begon and Bowers (1995a), Holt and Pickering (1986) and the bibliography cited therein] models of competition type have been treated and the role of multispecies interactions discussed, where the host density is itself considered as a dynamical variable, and an “ecological” perspective is envisaged. An investigation of two exponentially reproducing populations, each of them affected by the disease, can be found in Holt and Pickering (1986). Coexistence is governed by inter- and intra-specific infections. The numerical simulations do not reveal stable limit cycles. At the same time, the complications of the theoretical analysis of the stability are stressed. This fact will also be reflected in this section, in which the most complicated equilibria are essentially intractable, and therefore investigated mainly by numerical means.

In Holt (1977) a comparison with the Lotka-Volterra model with apparent competition among the hosts is discussed. The main inferences state that the prevalence of infection does not depend on alternative species being present; stable coexistence is impossible for a disease spreading only among the host species. In Begon and Bowers (1995b) two species share an infective agent, and limit cycles are discovered around the equilibrium whose stability is regulated by inter- and intra-specific infections. However, a whole range of possible complex behaviors is found. The system is also examined from an ecological
viewpoint, i.e., allowing one of the hosts to be a pest that the pathogen is supposed to fight.

Closely related to the present investigation are the competing systems with a disease-free species together with another epidemics-affected host, which are considered in Begon and Bowers (1995a). The classical paper by Anderson and May (1986) considers two competitors, of which one is disease affected. The effect of the latter is to forbid reproduction of the infected individuals. The results highlight the possibility that the epidemics-affected superior competitor that if unaffected, would eliminate the other competitor, settles instead toward coexistence with it.

Two pathogens “competing” over the same host have been considered for instance in Dobson (1985) and Hochberg et al. (1990). Hochberg and Holt (1990) demonstrates that coexistence is allowed at an intermediate level between the efficient exploitation of the infected hosts and the less virulent exploitation of healthy ones.

We consider two species whose sizes at time $T$ are denoted by $P(T)$ and $Q(T)$. They may represent two predators feeding on each other, or two species in the same habitat using the same resources, for instance, goats and cattle grazing on the same meadows. We take the $Q$ species to be affected by an epidemic, which cannot be transmitted to the $P$ population, indicating by $Q_I$ the infected $Q$’s. The model with mass-action incidence and the possibility of disease recovery is

$$\frac{dP}{dT} = P [a - bP - cQ - \eta Q_I],$$
$$\frac{dQ}{dT} = Q [d - eP - f (Q + Q_I) - \delta Q_I] + \nu Q_I,$$
$$\frac{dQ_I}{dT} = Q_I [\delta Q - gP - f (Q + Q_I) - \nu].$$

Each equation describes the logistic growth for the $P$ and the $Q$ species; it contains the negative cross-product interaction terms whose negative sign describes competition. The last equation gives the dynamics of the diseased population, which is less able to compete for resources, this being described by the different parameter $\eta$ in the first equation.

**Local stability analysis**

Some biologically important questions that can be answered in part by the stability analysis concern the possibility that the action of a competitor in an environment in which an epidemic spreads among a population might eradicate the disease, if and how a disease can push to extinction one of the competing species, and under what circumstances the disease creates unstable dynamics.

The equilibria $E_i \equiv (P_i, Q_i, Q_{I,i})$ of the system are the following always feasible points: the origin $E_0$ with eigenvalues $a, -\nu, d$, giving instability; $E_1$
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with only a nonzero component $P_1 = \frac{a}{b}$ and eigenvalues $-a, -\frac{1}{b}(ag + bv)$, $\frac{1}{f}(bd - ae)$, showing it is stable if $ae > bd$; $E_2$ again with only one nonzero component, $Q_2 = \frac{d}{f}$, with eigenvalues $-d, \frac{1}{f}(af - cd), \frac{1}{f}(d\delta - df - f\nu)$ and stability conditions

$$cd > af, \quad f(\nu + \delta) > d\delta. \quad (7.34)$$

These points are the corresponding phase space equilibria of the underlying demographic model, also with similar stability conditions. Only for $E_2$ does an extra condition on the epidemiological parameters arise (7.34) showing that the disease then affects the infected species dynamics. Considering the SI model $\nu = 0$, (7.34) reduces to $f > d$, i.e., it gives an extra demographic condition on the model. Thus, disease introduction may alter the stability of $E_2$ simply since $f < d$ destabilizes the otherwise stable equilibrium in the disease-free environment, for $cd > af$. It also helps the weaker species if it is introduced in the “winning” species, as the former would thrive in an ecosystem where normally it is wiped out by the adversary competitor.

The role of (7.34) is important also from the epidemics point of view; if $Q$ were the only species in the environment, by the epidemics action it would tend to $(\hat{Q}_Q, \hat{Q}_I) = (\frac{\nu}{\delta}, N - \frac{\nu}{\delta})$. If a competitor is introduced with interaction parameters satisfying (7.34), it would then remove the disease, although by being eliminated itself altogether. This allows control of the epidemics by a demographic device. If the action is done immediately after an epidemic outbreak, the environment could return to the previous conditions and the final level of the $Q$ species would be given by $Q_2$, independent of the epidemic parameters, another important point.

$E_3$ with nonvanishing components $P_3 = \frac{1}{D}(cd - af), \quad Q_3 = \frac{1}{D}(ae - bd), \quad D = ce - bf$ is again a counterpart of the demographic model, with the same feasibility condition, namely, $bf - ce, bd - ae, af - cd$ must all have the same sign. The three eigenvalues are

$$\lambda_3 = \frac{1}{D}[g(af - cd) + (f - \delta)(bd - ae)] - \nu,$$

and the two roots of the same quadratic polynomial of the underlying two-dimensional demographic model, implying the very same instability situation. This equilibrium remains also a saddle in the three-dimensional phase space. Notice further that imposing $f > \delta$ entails $\lambda_3 < 0$, so that in turn from (7.33) $\frac{dg}{df} < 0$. In these conditions the trajectories in the phase space tend then to the coordinate plane $Q_I = 0$, so that the final behavior of the model reduces to the classical competing system unaffected by the presence of the disease. Since $\nu$ appears negative in $\lambda_3$, the epidemic in general cannot influence the demographics, but again it can be removed by introducing a competitor. Although the situation is similar to the one discussed for $E_2$, in this case, however, the system settles to an equilibrium that also contains the competitor population, while the original population attains the level $Q_3 \neq Q_2$. 

Other endemic equilibria in which the sound species disappear are $E_{4.5}$ with $Q_{1.4.5} = \frac{1}{4}((\delta - f)Q_{4.5} - \nu)$, where $Q_{4.5}$ solve $\delta^2 Q^2 - (df + 2\nu \delta)Q + \nu^2$. Its roots are always positive as geometrically they can be represented by the intersections of the parabola $y_1 = (\delta Q - \nu)^2$ with the straight line $y_2 = dfQ$.

For feasibility we need to investigate only $Q_{1.4.5}$, ensured if $Q_{4.5} > \frac{\nu}{\delta^2}$. For $f > \delta$, the point $E_{4.5}$ is infeasible, and, as seen above, for $E_3$ the system approaches the underlying demographic model behavior. Take then $\delta > f$; to ensure feasibility for $E_{4.5}$, we require the value of $y_2$ at the vertex of the parabola to be larger than $\frac{\nu}{\delta^2}$, or $df(\delta - f) > \delta$.

For $\nu = 0$, we find in particular $E_4 \equiv E_0$ and $E_5 = \left(0, \frac{df}{\delta^2}, \frac{df}{\delta^2}(\delta - f)\right)$, feasible for $\delta > f$. Stability can easily be established, the eigenvalues being

$$\mu_1 = -\frac{1}{\delta^2}[df(\delta - \eta) - \delta(a\delta - d\eta)], \quad \mu_{2,3} = -\frac{d}{2\delta}\left[f \pm \sqrt{f^2 - 4f\delta}\right].$$

From feasibility it follows that $5f^2 - 4f\delta < f^2$ and $Re(\mu_{2,3}) < 0$, so that stability is then governed by $\mu_1$. Sufficient conditions for stability are the alternative pairs of inequalities

$$cf > \delta \eta, a\delta^2 > df \eta; \quad a\delta < d\eta, c > \eta. \quad (7.35)$$

At $E_5$ the $P$ species disappears and $Q$ survives at a lower level $df\delta^2$ than the one exhibited by the classical, disease-free model, which would be given by $df^{-1}$. This contrasts with the classical SI epidemics model in which no sound individual is ultimately present. The use of an external competitor to control a disease results in a mistake, since the disease attains the nonzero value $d(\delta - f)\delta^2$ and the competitors vanish. In case the incidence is very high, the endemic level of the epidemics is however low, $\approx d\delta^{-1}$, and in such a circumstance the introduction of the competitor might be beneficial.

There are two other equilibria $E_{6.7}$ with components

$$P_{6.7} = \frac{1}{bf - g\eta}[Q_{6.7}(f\eta - \eta \delta - cf) + \eta \nu + af],$$
$$Q_{1.6.7} = \frac{1}{bf - g\eta}[Q_{6.7}(b(\delta - f) + cg) - (ag + b\nu)],$$

where $Q_{6.7}$ solve the equation $p(Q) \equiv a_2Q^2 + a_1Q + a_0 = 0$ with coefficients

$$a_2 = \eta(\delta - f) + cf(e - g) + g(f\eta - c\delta) - b\delta^2,$$
$$a_1 = a [f(g - e) + g\delta] + d(bf - g\eta) + \nu(-e\eta + 2b\delta + cg),$$
$$a_0 = -\nu(b\nu + ag).$$

Again consider $\delta > f$. If $a_2 > 0$ in view of $a_0 \leq 0$, there is always a positive root of the above quadratic. Imposing $e > g$, to ensure the positivity of $a_2$ we could require

$$e\eta(\delta - f) + cf(e - g) > g(c\delta - f\eta) + b\delta^2. \quad (7.36)$$
If instead $a_2 < 0$, two positive roots exist in case the discriminant is positive. Consider in particular the SI model for which $\nu = 0$ implies $a_0 = 0$; we find $E_7 \equiv E_0$, $Q_6 = -\frac{a_1}{a_2}$. We must require $a_1$ and $a_2$ to differ in sign, i.e.,

$$\eta_\delta (\delta - f) + c f (e - g) + g (f \eta - c \delta) < b \delta^2,$$

(7.37)

together with sufficient conditions ensuring that $a_1 > 0$, such as $bf > g\eta$ and $g > e$. We finally need to ensure the positivity of the other populations; $P_6 > 0$ can be ensured by bounding the size of $Q_6$ while for $Q_{I,6}$ we need $Q_6$ to be bounded below:

$$-a_1 \frac{a f}{e f + \eta (\delta - f)} \equiv \hat{Q}, \quad -a_2 > \frac{ag}{b(\delta - f) + cg} \equiv \tilde{Q}.$$

From the former we have

$$- (bf - g\eta) (d\eta (f - \delta) + \delta^2 a) + 2caf^2 (g - e) + df c (bf - g\eta)$$

$$- (e\eta (\delta - f) + c f (g - e) + g (f \eta - c \delta) - b \delta^2) (e f + \eta (\delta - f)) < 0,$$

with positive denominator, using (7.37). From the conditions on $a_1$, the above inequality gives

$$(bf - g\eta) \delta^2 a > (bf - g\eta) d\eta (\delta - f) + 2caf^2 (g - e) + df c (bf - g\eta).$$

(7.38)

For (7.36), we find that the denominator is positive, so that the numerator must be negative, giving

$$2caf^2 (g - e) + df c (bf - g\eta) > (bf - g\eta) (d\eta f + \delta^2 a - \delta \eta d).$$

(7.39)

For the lower bound, proceeding similarly, the inequality is rewritten as

$$\frac{(bf - g\eta) (bd - ae) (\delta - f) - g ((bf - g\eta) (af - cd) - 2afe c (g - e))}{(e\eta (\delta - f) + c f (g - e) + g (f \eta - c \delta) - b \delta^2) (e f + \eta (\delta - f)) + cg} > 0.$$  

Assuming (7.36) and $\delta > f$, the denominator is positive and finally we have

$$(bf - g\eta) (bd - ae) (\delta - f) > g ((bf - g\eta) (af - cd) - 2afe c (g - e)).$$

For (7.37), the opposite condition must be required. We do not analyze the stability. In this equilibrium, the three competing subpopulations coexist at the nonzero level. Thus, if the competitor has been introduced to eradicate the disease, the net result is a failure, since the disease persists endemically, and the same holds true by introducing a disease to fight a competitor, as the latter survives.

Invariant sets for the trajectories

The results that follow show that it is enough to consider the region around the origin to understand the $\omega$-limit dynamics of the system. Since we model
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competing species, this boundedness result should be expected. To prove it, consider the box in the \( PQQ_1 \) phase space with opposite vertices at the origin and at \( X^* = (P^*, Q^*, Q_1^*) \), where \( P^* > \frac{a}{b}, Q_1^* > \frac{d}{f}, Q^* > \max \left( \frac{b}{c}, \frac{\nu}{\eta} \right) \).

We calculate the angle of the system’s flow with each face of \( B \) through \( X^* \). Consider the plane \( \pi_Q : Q = Q^* \), with outward unit normal vector \( n_Q \); similar notations hold for the other two variables. We have

\[
n_Q \cdot (P', Q', Q_1') \mid_{\pi_Q} = Q^* (d - fQ^*) - cPQ^* - Q_1^* [(f + \delta) Q^* - \nu] \\
\leq -cPQ^* < 0,
\]

thus the angle between the flow and the outward normal to the face of \( B \) lying on \( \pi_Q \) is obtuse, i.e., the trajectories enter into \( B \) on this face. Similarly, taking the plane \( \pi_P : P = P^* \), with outward unit normal \( n_P \), we have again

\[
n_P \cdot (P', Q', Q_1') \mid_{\pi_P} = P [a - bP - cQ - \eta Q_1] \mid_{\pi_P} \\
= P^* [a - bP^* - cQ - \eta Q_1] \leq P^* [-cQ - \eta Q_1] < 0.
\]

An analogous inequality can also be computed for \( n_Q^I \), i.e., \( n_Q^I \cdot (P', Q', Q_1') \mid_{\pi_Q^I} < 0 \). The trajectories thus enter into \( B \) but cannot exit from it, as the existence and uniqueness theorem for dynamical systems prevents the crossing of the faces lying on the coordinate axes; notice indeed that (7.33) is homogeneous. In conclusion, \( B \) is a positively invariant box.

7.3.1 Simulation discussion

Our simulations show the occurrence of persistent oscillations in various parameter ranges. Figure 7.4 shows them for the parameter values

\[
a = 1.1, \quad b = 0.9, \quad c = 1.2, \quad d = 2, \quad e = 1.1, \\
f = 1, \quad g = 19, \quad \delta = 20, \quad \eta = 1, \quad \nu = 0.
\]

A similar feature is obtained in Figure 7.5 where the oscillations arise by allowing disease recovery, i.e. \( \nu \neq 0 \), for the parameter values

\[
a = 2.0, \quad b = 1.0, \quad c = 1.0, \quad d = 1.0, \quad e = 0.25, \\
f = 0.99, \quad g = 5, \quad \delta = 15, \quad \eta = 15, \quad \nu = 0.09.
\]

These sustained oscillations are remarkable also from the ecological viewpoint, especially because they are not induced by the presence of a periodically driven forcing term. Observe, however, that the populations drop to very low levels, a fact that perhaps hints at an internal structural instability of the model, since small perturbations of the environmental conditions might drive the populations to extinction. When the disease-free equilibrium is a saddle, \( E_1 \equiv (2, 0) \), namely for

\[
a = 2, \quad b = 1, \quad c = 1, \quad d = 1, \quad e = 1, \quad f = \frac{1}{4},
\]

(7.40)
FIGURE 7.4: Diseased competing species: persistent oscillations for the three subpopulations.

FIGURE 7.5: Diseased competing species: persistent oscillations for the three subpopulations after a transient phase, indicating the occurrence of a bifurcation depending on the disease recovery parameter $\nu$.

introducing the disease alters the system’s behavior as follows. For $\eta = 30$, $\delta = 1$, $g = 0$, $\nu = 0$, the model settles to $(0, \frac{1}{4}, \frac{3}{4})$. Taking $\delta = \frac{1}{2}$ the
equilibrium becomes \((0, 1, 1)\), but for \(\delta = \frac{1}{10}\) the equilibrium is \((1.9, 0.003)\). Thus, from the equilibrium of the disease-free environment, in the diseased environment the system may settle to a point where either the sound species \(Q\) or the infected one disappear starting from the same initial conditions, for different values of the incidence of the disease. Also the SIS model has similar consequences. For \(\nu \geq \frac{1}{5}\), the equilibrium is \(E_1 \equiv (2, 0, 0)\), but for \(\nu = \frac{1}{10}\), the equilibrium becomes \(E_5 \equiv (0, 1.37, .97)\) and for \(\nu = \frac{1}{20}\), it is \(E_5 \equiv (0.19, .99)\). Thus, the introduction of a recoverable disease or a disease with moderate incidence in the succumbing competitor may lead to the extinction of the predominant species, provided it also deeply influences the competitor (i.e., if \(\eta\) is large).

Conversely, a disease affecting the dominant species but with low effect on the dominated species, (i.e., for \(\eta\) small), may have the effect of wiping the former out, in favor of the succumbing one, or to force the dominant one to a lower value, where the disease remains endemic, when it has a bigger impact on the dominated population (i.e., for larger \(\eta\)). Indeed, for the same demographic parameters (7.40), the initial condition \((P_0, Q_0) \equiv (1, 3)\) leads to the equilibrium \(E_2 \equiv (0, 4)\). Now for the ecoepidemic system with \(\nu = 0\), \(g = 0\) and \(\delta = 1\), and initial condition \((1, 3, 1)\) we find the equilibrium \(E_1 \equiv (2, 0, 0)\) for \(\eta \leq .46\) and the equilibrium \(E_5 \equiv (0, 1.64, .92)\), for larger values of \(\eta\).

We notice, finally, that the epidemics can be eradicated for suitable choices of the parameter values, by introducing a competitor \(P\) of the affected species. The final value attained by the sound subpopulation of the latter may however be at a higher or lower level than the one attained by the system without the species \(P\).

### 7.4 Ecoepidemics models of symbiotic communities

Specific biological examples of symbiotic species affected by diseases that we can encompass with the models under investigation here are, for instance, the chestnut trees (\(Castanea sativa\)) in symbiosis with mushrooms (\(Cantharellus cibarius, Boletus spp., Amanita spp.\)) and affected by chestnut cancer (\(Endothia parasitica\)), the L-form bacteria that form nonpathogenic symbiosis with a wide range of plants, and the latter then become capable of resisting further bacterial pathogens (Walker et al., 2002). Also, very recently it has been discovered that the soil nematode \(Caenorhabditis elegans\) transfers the rhizobium species \(Sinorhizobium melotiti\) to the roots of the legume \(Medicago truncatula\) (Horiuchi et al., 2005).

Instead of providing a more or less complete analysis, as was done in the former sections, our study here concentrates mainly on two fundamental biological questions. Of interest is to provide an answer on whether and how
the epidemics in one of the species affects also the dynamics of the “sound” population in a mutualistic community. Moreover, we consider if the introduction of a symbiotic population in an environment where an already infected species lives may fight the disease to the point of eradicating it. We will analyze the equilibria but also mainly provide simulation results obtaining some remarks that are not intuitive. Our findings indicate that for suitable parameter ranges, the model exhibits an interesting feature: The equilibrium shifts to a higher value, indicating that the disease seems to have a positive effect on the environment. This also agrees with recent field experiments revealing the same phenomenon.

We need here two variables for the symbiotic species $P$ and $Q$ and one more $Q_I$ for the infected individuals of population $Q$. By assumption, the disease will not be able to propagate to the other species. Notice that in the equation for the sound individuals there will now be two interaction terms, one of each sign, as the commensalism is beneficial for that population, but the negative sign accounts for the disease incidence. Also, encounters with a diseased individual may not result in the very same advantage gained by associations with a sound one. To model the benefit obtained by the sick individuals by interacting with the unaffected population, we may assume either that the gain may be larger for the former than for the latter, or, conversely, in particular if the disease impairs the commensalism or for instance the wandering in the environment. Notice also that the demographic aspect has to account for both sound and infected individuals in the population pressure terms hindering the growth of the species. More specifically, in both equations for the affected species the logistic terms must be given by products of the relevant populations with the totality of the population $Q + Q_I$.

We may even allow reproduction of sound $Q$’s begetting sound offsprings at rate $d$ and of diseased individuals giving birth to both sound and disease-affected newborns at rates $h$ and $\ell$, respectively, i.e.,

$$
\frac{dP}{dT} = P(a - bP + cQ + kQ_I),
$$
$$
\frac{dQ}{dT} = Q(d + eP - f(Q + Q_I) - \delta Q_I) + (\nu + h)Q_I,
$$
$$
\frac{dQ_I}{dT} = Q_I[\ell + \delta Q + gP - f(Q + Q_I) - \nu].
$$

Equilibria

Let the equilibria be denoted by $E_k \equiv (P_k, Q_k, Q_{I,k})$. The origin $E_0$ is unstable, as its eigenvalues are $a$, $d$, $\ell - \nu$, thus showing the same nature as its demographic counterpart. Among the boundary equilibria, we find first the ones with only one nonzero component, namely, $E_1$ and $E_2$ with $P_1 = \frac{a}{b}$ and eigenvalues $-a$, $\frac{d}{f} + d$, $\ell - \nu + \frac{\nu}{f}$, showing again instability, and with $Q_2 = \frac{d}{f}$ and eigenvalues $-d$, $a + \frac{ed}{f}$, $\frac{\delta}{f} + \ell - d - \nu$ once again unstable. These
instabilities once again arise from the corresponding results for the equilibria of the disease-free symbiotic model. For $E_3$, letting $D \equiv bf - cc$, the two nonzero components are $P_3 = \frac{1}{f} (af + cd)$, $Q_3 = \frac{1}{f} (ae + bd)$, i.e., it is a disease-free point. The sign of the first eigenvalue $\lambda_1^{[3]} \equiv \ell - \nu + (\delta - f) Q_3 + g P_3$ governs stability as the other two are the pair of roots of the characteristic equation of the underlying classical demographics equilibrium.

For stability, the condition $\ell + (\delta - f) Q_3 + g P_3 < \nu$ needs to be satisfied.

Let us remark that for a very virulent epidemic, i.e., for a large $\delta$, or for a very small recovery rate $\nu$, the stable equilibrium of the disease-free symbiotic system becomes unstable, and this is even more stringent for an SI infection. In such a case, indeed, a very large $f$ is needed to keep stability, which entails a large crowding effect on the epidemics-affected species. Conversely, if the latter is bounded above by disease incidence, $E_3$ is surely unstable.

There is then a pair of equilibria $E_{4,5}$ with only the affected species surviving with disease at the levels $Q_{1,4,5} = \frac{1}{f} [(\delta - f) Q_{4,5} + \ell - \nu]$, where $Q_{4,5}$ are the roots of

$$\Pi(Z) \equiv \delta^2 Z^2 - [f(\ell - d + h) + \delta(\ell - 2\nu - h)] Z + (\nu + h)(\nu - \ell) = 0. \quad (7.42)$$

For feasibility we must impose that $\delta Q_{4,5} + \ell - \nu > f Q_{4,5}$. One eigenvalue is immediate,

$$\lambda_1^{[4,5]} \equiv a + Q_{4,5}(c - k) + \frac{k}{f} (Q_{4,5}\delta + \ell - \nu)$$

$$> a + Q_{4,5}(c - k) + k Q_{4,5} = a + c Q_{4,5} > 0,$$

implying that $E_{4,5}$ are always unstable, if feasible.

The last two equilibria, $E_{6,7}$, in which the species coexist, have components

$$P_{6,7} \equiv \frac{1}{bf - gk} [Q_{6,7}(cf + k(\delta - f)) + af + k(\ell - \nu)]$$

$$Q_{1,6,7} \equiv \frac{1}{bf - gk} [Q_{6,7}(cg + b(\delta - f)) + ag + b(\ell - \nu)].$$

They will only be investigated numerically, as they arise from the solution of the quadratic equation $e_0 Z^2 + e_1 Z + e_2 = 0$, with

$$e_2 = (h + \nu)[b(\ell - \nu) + ag],$$

$$e_1 = \delta [b(2\nu - \ell + h) - ag] + f[b(d - h - \ell) + a(e - g)]$$

$$+ k[e(\ell - \nu) - dg] + cg(h + \nu).$$

$$e_0 = f(g - e)(c - k) + \delta(ek - \delta b - gc).$$

**Boundedness**

To show that the trajectories ultimately lie in a compact set, let us define $E = \max(e, g)$, $D = \max(d, \ell + h)$, $K = \max(k, c)$. Summing the last two
equations, and denoting the total disease-affected population by $R = Q + Q_I$, we find from (7.41)

$$\frac{dR}{dT} = dQ + (\ell + h)Q_I + (cQ + gQ_I)P - fR^2 \leq R[D + EP - fR] \equiv Rr(P, R),$$

$$\frac{dP}{dT} \leq P(a - bP + KR) \equiv Pp(P, R).$$

The setting of $p$ and $r$ to zero will give the loci in the $RP$ phase plane solutions of the inequalities obtained from the isoclines $\frac{dR}{dT} \leq 0$ and $\frac{dP}{dT} \leq 0$, respectively. The lines $p(P, R) = 0$ and $r(P, R) = 0$ intersect at $T \equiv (TP, TR) = \left(\frac{aE + bD}{b(aE + bD)} - \frac{bf}{E}, \frac{aE + bD}{b(aE + bD)} - \frac{bf}{E}\right)$, a feasible point whenever $bf > EK$. Assuming this condition, the quarter-circle centered at the origin and of radius given by $\rho \equiv \sqrt{T_F^2 + T_R^2}$, lying in the first quadrant, is easily seen to be a positively invariant set for (7.41), as the flow of the dynamical system there points inward. Hence all trajectories are bounded as ultimately they must enter into it.

### 7.4.1 Disease effects on the symbiotic system

Our simulations are aimed at observing the consequences entailed by the disease in the species $Q$ on the demographic equilibrium of the symbiotic system $P-Q$. In general, the equilibrium $E_3^{(d)}$ suffers from this change in the system, but there are parameter values for which interestingly the equilibrium shifts to a higher value. Thus the disease positively affects the environment.

We fix the parameters of the symbiotic system $a = 5, b = 1, c = 1, d = 5, e = 1, f = 2$, so that the equilibrium lies at $P_3 = 15, Q_3 = 10$. The effect of the modification of the remaining population parameters for the infected individuals, $g, k$, i.e., the return obtained from mutualism for both infected and disease unaffected species, $Q_I$ and $P$, and the infected reproduction rates resulting in sound and diseased offsprings, respectively, $h, l$, and the epidemic parameters $\delta, \nu$ can be investigated.

In Figure 7.6 we report one such typical simulation. When relevant the six graphs contain the horizontal lines indicating the symbiotic equilibria in absence of the disease. Columnwise and left to right, we have respectively the plots of the solutions of the system (7.41) for the disease-immune population $P$ and total population subject to disease, $R \equiv Q + Q_I$, for the disease-affected population $Q$, and the fraction infected versus sound individuals, $Q_I/Q$, and finally for the infected population $Q_I$, and the disease prevalence $Q_I/R$. A dramatic improvement in the equilibrium is found for the following choice of parameters $g = 2.4, h = 4.7, k = 0.8, l = 4.3, \delta = 13, \nu = 0.6$.

A large improvement of the equilibrium is found for the unaffected species $P$, which is more than one order of magnitude larger than $P_3$. While the $Q$’s are almost wiped out, an outsurge of $Q_I$ is observed, which drives the
FIGURE 7.6: Disease effects on symbiotic system, for the ecoepidemic parameter values \( g = 2.4, h = 4.7, k = 0.8, \ell = 4.3, \delta = 13, \nu = 0.6 \) and demographic parameters \( a = 5, b = 1, c = 1, d = 5, e = 1, f = 2 \).

If one wants to harvest either one population, and the disease does not constitute a problem, this situation would be certainly an option to be seriously considered, to sustain the populations at much higher levels.

7.4.2 Disease control by use of a symbiotic species

Notice that disease eradication by introduction of a symbiotic species is not possible, as the best possible case for reducing the infected class \( Q_I \), is to zero out the parameters expressing the return sound and infected obtain from the contact with the \( P \) species, i.e. setting \( g = 0, e = 0 \). But in such case we have the following results.

Consider always the same epidemic model, for the parameters \( d = 12, f = 2.25, \delta = 2.7, \nu = 2.5, h = 3, \ell = 10, \) so that the equilibrium is \( Q = 6.0576 \), \( Q_I = 4.5458 \). Introduce then the symbiotic species with intrinsic reproduction rate and crowding coefficient \( a = 10, b = 1 \), i.e. at carrying capacity \( P_3 = 10 \). The remaining parameters express the gain \( P \) have from interactions with sound and infectives of the other species and can only positively influence the
FIGURE 7.7: Disease control through parameters $e$ and $g$; each curve corresponds to values of $g$ in the range $[0, 0.6]$ with stepsize 0.03

combined system. We take them as $c = 1$, $k = 1$. The new equilibrium is $P = 15.6268$, $Q = 1.9112$, $Q_I = 3.7156$, so that is not possible to obtain $Q_I = 0$, since the above choice provides the minimum return for the infected individuals $Q_I$. Thus it is not possible in general to eradicate the disease from the $Q$ species by introducing the symbiotic species $P$. But, on the contrary, it is possible to keep the ratio $Q_I/Q$ and the prevalence $Q_I/R$ small, by acting on the parameters $e$ and $g$, i.e., changing the return sound and infected get from the $P$ species. Figure 7.7 shows that the maximum possible reduction can be obtained by taking $g = 0$, $e \approx 2.6$. The horizontal line gives the ratio $Q_I/Q$ (left) and the prevalence $Q_I/R$ (right) for the original SIS equilibrium for the $Q$ species. The lines illustrate the behavior of the very same ratio at the equilibrium after introduction of the symbiotic species $P$. The lines from left to right correspond to increasing values for the parameter $g$ from $g = 0$ to $g = .6$ with stepsize $g = 0.03$. The lowest value of the curves gives the minimum prevalence, in the left plot.

What we just remarked seems in contradiction with the fact that the disease-free equilibrium $E_3$ may be stable. But the focus of this subsection are the consequences of introducing a new symbiotic species in the environment, once the biologic parameters related to the disease-affected species $Q$ are fixed. The eigenvalue $\lambda^{[3]}$ is only positively affected by $P_3$, so that it can only be destabilized, if before it was stable.