Introduction

The temporal development of a natural or artificial system can conveniently be modeled by a semiflow. A semiflow consists of a state space, X, a time-set, J, and a map, Φ.

The state space X comprehends all possible states of the system: the amounts or densities of the system parts and, if there are one or several system structures, their structural distributions.

According to the interests of the authors, this book concentrates on biological, ecological, and epidemiological systems. For the last, for example, the state space typically contains the amounts or densities of susceptible and infective and possibly exposed and removed individuals. For spatial spread, spatial distributions are included in the state space. If age-structure is thought to be important, age-distributions are included as well.

Time can be considered as a continuum or in discrete units; the most common choices for the time set J are the nonnegative reals or the nonnegative integers, $\mathbb{R}_+ = [0, \infty)$ and $\mathbb{Z}_+ = \mathbb{N} \cup \{0\} = \{0, 1, \ldots\}$. Depending on the model, the time unit can be a year, month, or day.

The most important ingredient of a semiflow is the semiflow map $\Phi : J \times X \to X$. Often Φ itself is called the semiflow. If $x \in X$ is the initial state of the system (at time 0), then $\Phi(t, x)$ is the state at time t. This interpretation immediately leads to the identity

$$\Phi(0, x) = x, \qquad x \in X.$$

Further, semiflows are characterized by the semiflow property:

$$\Phi(t + r, x) = \Phi(t, \Phi(r, x)), \qquad r, t \in J, \quad x \in X.$$

This property has the following interpretation: If x is the initial state and the system develops for a time r and if the state $\Phi(r, x)$ is taken as a new initial state and the system develops for another time t, then the resulting state is the same as if the system develops from x for the time $t + r$.

It may be that a system does not last for all future times. Then the semiflow map Φ is only defined on a subset of $J \times X$ and the semiflow property must be appropriately supplemented. If seasonal influences are important, nonautonomous semiflows need to be considered (Chapter 13).

Semiflows are induced by differential equations of all kind (ordinary, partial, functional, and combinations of these): in the case of an ordinary differential equation, $\Phi(t, x)$ is the solution at time t when x is the initial datum (at time 0).

To be more concrete, consider the following endemic model for a fertility reducing infectious disease caused by a viral, bacterial, or fungal parasite. S and I denote the respective numbers of susceptible and infective hosts,

$$S' = (\beta - \mu)S + q\beta I - \kappa SI, \qquad I' = \kappa SI - (\mu + \alpha)I. \tag{0.1}$$

Here $\beta > \mu > 0$ are the per capita birth and death rates, $\kappa > 0$ is the per capita infection rate and $\alpha \geq 0$ the additional death rate due to the disease. The factor $q \in [0, 1]$ expresses the reduction of fertility for an infective individual.

It follows from standard arguments in ordinary differential equations that, for each pair $S_0, I_0 \geq 0$, there exist unique solutions $S, I : \mathbb{R}_+ \to \mathbb{R}_+$ with $S(0) = S_0$, $I(0) = I_0$, where $\mathbb{R}_+ = [0, \infty)$ is the set of nonnegative real numbers. Then

$$\Phi(t, (S_0, I_0)) = (S(t), I(t))$$

defines a semiflow with state space $\mathbb{R}_+^2$ and time-set $\mathbb{R}_+$. The semiflow property follows from the uniqueness of solutions.

It is an important question whether the dynamical system persists (remains safely away from extinction) as a whole or at least in parts (which parts?). This question can be mathematically formulated and addressed by using a *persistence function*

$$\rho : X \to \mathbb{R}_+. \tag{0.2}$$

For $x \in X$, $\rho(x)$ is the amount of the part of the system that is of particular interest. For the model of a fertility-reducing infectious disease, if emphasis is on whether the disease becomes endemic or can be eradicated, $\rho(S, I) = I$ is the number or density of infective (or infected) individuals. If emphasis is on whether the disease threatens to drive the host population into extinction, then $\rho(S, I) = S + I$ is the total number of hosts.

The semiflow Φ is called *uniformly ρ-persistent* if there exists some $\epsilon > 0$ such that

$$\liminf_{t\to\infty} \rho(\Phi(t,x)) \geq \epsilon \quad \text{whenever } x \in X, \rho(x) > 0. \tag{0.3}$$

A stepping-stone to uniform persistence is uniform weak persistence: Φ is *uniformly weakly ρ-persistent* if (0.3) holds with $\limsup$ replacing $\liminf$. If Φ is uniformly ρ-persistent, the amount of the system part of interest is eventually bounded away from 0 with the bound being independent of the initial state (as long as the interesting part is present initially). If Φ is only uniformly weakly ρ-persistent, then the amount can come arbitrarily close to 0 but always bounces back.

In terms of the infectious disease, we talk about uniform (weak) host persistence if $\rho(S,I) = S + I$ and about uniform (weak) parasite persistence (or disease endemicity) if $\rho(S,I) = I$.

This book embarks on the strategy of establishing uniform weak persistence first and then deriving uniform persistence.

From uniform weak to uniform persistence. Trivially, uniform persistence implies uniform weak persistence; the converse is not always true. Let us return to the example of the fertility-reducing disease. If $q = 0$ (i.e., the disease sterilizes), the host-parasite system becomes a special case of the Lotka-Volterra prey-predator system for which the whole first quadrant is filled with periodic orbits. This means that the system is uniformly weakly persistent for both host and parasite, but uniformly persistent for neither.

Strangely enough, the lack of uniform persistence concurs with a lack of the solutions to be eventually uniformly bounded: there is no $c > 0$ such that $\limsup_{t\to\infty}(S(t) + I(t)) < c$ for all nonnegative solutions.

To continue this line of thought in more generality, let us assume that the state space X is a metric space (though persistence theory is also possible without a topology on X).

The preliminary insight (which will turn out to be not completely correct) that some boundedness is needed for proceeding from uniform weak to uniform persistence can now be formulated in various ways. The strongest such formulation assumes the existence of a compact global attractor [**91**], and it becomes a natural question under which conditions a compact global attractor exists (Chapter 2). The presentation in this book differs from others [**91, 197, 238**] as an approach is chosen that deals with continuous and discrete time in a unified way.

Beyond that, two opposite directions are pursued: on the one hand, harvest the full fruit of assuming a compact global attractor (persistence à la Caesar) and, on the other hand, relax the assumption of a compact

attractor as much as possible (persistence à la Münchhausen, persistence via Arzela-Ascoli, and persistence via Laplace transform).

Persistence à la Caesar (*Attractor est omnis divisus in partes tres*, cf. De bello gallico) divides the compact global attractor, A, in three invariant parts: the extinction attractor on which the persistence functional ρ is zero, the persistence attractor, A_1, on which ρ is strictly positive, and a set of orbits connecting the extinction attractor to the persistence attractor. Every compact set on which ρ is strictly positive has a neighborhood U that is attracted by A_1:

$$d(\Phi(t,x), A_1) \to 0, \quad t \to \infty, \qquad \text{uniformly for } x \in U.$$

Here $d(y, A_1)$ is the distance from the point y to the set A_1. This convergence result implies that A_1 is stable.

In several examples (Chapter 8.7 and Chapter 9), we will find conditions under which the persistence attractor is a singleton set and thus, automatically, a locally asymptotically stable equilibrium. The techniques used for this involve Lyapunov type functionals [**163**] and Fourier transforms [**151**].

Persistence à la Münchhausen (Chapter 4.5) tries to get away with as few compactness assumptions as possible. It is called that way as it has some resemblance to the feat of Lügenbaron (lying baron) Karl Friedrich Hieronymus Freiherr von Münchhausen (1720-1797) who, in one of the tales told about him, escapes from a swamp lifting himself and his horse up pulling at his own hair [**17**] (see the drawing by Theodor Hosemann (1807-1875), Figure 4.1). While compactifying properties of the semiflow cannot be completely eliminated as assumptions (recall the Volterra predator-prey model), one can get quite far in this direction, though, at the expense of a considerable increase in technicality. For illustration, let us return to the model (0.1) for a fertility-reducing infectious disease.

Recall that, for $q = 0$ (sterilizing disease), this is a Lotka-Volterra predator-prey model which is known to be uniformly weakly persistent, but not uniformly persistent.

If $q \in (0, 1]$, one can show that both the susceptible and the infective part of the population persist uniformly, i.e., both the host and the parasite persist uniformly, though their numbers can grow without bound for certain parameter values (if $q\beta > \mu + \alpha$).

The reason for the different persistence scenarios becomes a little clearer when we reformulate the equations in terms of the total host population size $N = S + I$ and the fraction of infective hosts $y = I/N$,

$$\begin{aligned} N' &= N\Big(\beta(1-y) - \mu + (q\beta - \alpha)y\Big), \\ y' &= y\Big((\kappa N - \alpha - \beta)(1-y) - q\beta y\Big). \end{aligned} \tag{0.4}$$

Note the change in state space that becomes $X = \mathbb{R}_+ \times [0,1]$. Whatever q, as long as $0 \leq q \leq 1$, this system has the invariant set $\{(N,0); N > 0\}$ on which host population grows exponentially. This lack of boundedness does not necessarily impede uniform host or parasite persistence. Notice that

$$N' \geq N(q\beta - \mu - \alpha).$$

Assume that $q\beta - \mu - \alpha > 0$. Then $N(t)$ grows exponentially if $N(0) > 0$. This has the consequence that $y(t) \to 1$ as $t \to \infty$ if $0 < y(0) \leq 1$, i.e., the disease pervades the host population, and both host and disease persistence are uniform in the strongest possible way.

For $q = 0$, the case of a sterilizing disease, the state space has another invariant set, $\{(N,1); N \geq 0\}$, on which $N(t) \to 0$ as $t \to \infty$. This set has an attracting part where N is large and a repelling part where N is small.

Uniform weak host persistence can be shown whether or not $q = 0$. However, if $q > 0$, it can be shown on the state space

$$X = \{(N,y); N > 0, 0 \leq y \leq 1\},$$

while for $q = 0$ it can only be shown on the state space

$$\tilde{X} = \{(N,y); N > 0, 0 \leq y < 1\}.$$

See Theorem 3.3 for details. For $q = 0$, it is not the lack of boundedness in the host component that is an impediment for showing uniform host persistence, but the loss of completeness of the state space by the necessary exclusion of $y = 1$. So uniform host persistence only holds for $q > 0$ (Theorem 4.14).

Using uniform host persistence, uniform persistence of the disease can now be established, but it requires the full Baron von Münchhausen hair-pulling stunt which we cannot explain here (see Theorem 4.17 and the subsequent application).

Interestingly enough, if $0 < q\beta < \mu + \alpha$, the ideas of ρ-persistence can be used to show that the host population size is eventually uniformly bounded: there is some $c > 0$ such that $\limsup_{t\to\infty} N(t) < c$ for all solutions N and y with $y(0) > 0$. Simply use $\rho(N,y) = \frac{1}{1+N}$ as persistence function. But uniform disease persistence must be established first before it can be established that the disease imposes a bound on the host population size that is eventually uniform (Exercise 4.9).

Since our ODE model is two-dimensional, one can alternatively use phase-plane methods. This way, for $0 < q\beta < \mu + \alpha$, one can directly (without using persistence theory) obtain a compact host and disease persistence attractor that attracts all compact sets in $\{(N,y); N > 0, 0 < y \leq 1\}$.

The use of persistence theory becomes unavoidable, however, if one considers several competing parasite strains which provide complete cross-protection [**218**] or several stages of infection (Section 4.7).

The existence of an attractor with host and disease persistence concurs with the existence of an equilibrium in $(0, \infty) \times (0, 1)$ which is called either an *endemic equilibrium* or a *host-parasite-coexistence equilibrium* depending on whether the view point is mainly epidemiological or ecologic. By the Poincaré-Bendixson limit set trichotomy, any solution in the host and disease persistence attractor is either this equilibrium or a periodic orbit or a homoclinic orbit connecting the equilibrium to itself. The Bendixson-Dulac criterion (use the Dulac function $\frac{1}{Ny(1-y)}$) rules the second and third possibility out, and the host and parasite persistence attractor is the singleton set consisting of the endemic equilibrium. Notice that the stability of the endemic equilibrium follows without a linearized stability analysis. Of course, the latter would be easy for a small system like this, but could be quite harrowing for a large system.

If the state space is infinite dimensional, a compact attractor can be elusive for other reasons than the lack of eventual uniform boundedness or completeness as in the case of a model for cells with age-dependent division growing in a chemostat (Chapters 12 and 13.13.4). In this case, the Arzela-Ascoli theorem can come to the rescue and imply that, for certain sequences (x_j) that may have no convergent subsequences, $\rho(\Phi(t, x_j))$ has a subsequence that converges as $j \to \infty$ uniformly for t in compact subsets of $\mathbb{R}_+$. Notice that this remedy relies on the persistence function ρ and even works without a topology on the state space. It extends to persistence for nonautonomous semiflows (nonlinear evolutionary systems) (Chapter 13).

Another line of keeping assumptions at minimum concerns the continuity of the semiflow if time is a continuous variable. In certain models of physiologically structured populations, it is either necessary or convenient to choose the space of measures as a state space: either the solutions become measure-valued even for smooth-initial data by some mild form of shock-formation [**3, 213**], or there are equilibria to be taken care of that are measures [**2, 227**]. For a state space of measures, the semiflow is typically continuous in the space variable but sometimes not in the time variable (at least not in the same, the strong, topology). To illustrate this phenomenon and various ways of overcoming the associated difficulties, we consider the endemic model with variable infectivity also for infection-age distributions that are measures (Section 9.9.10).

How to get uniform weak persistence. Historically, there have been two main approaches to persistence theory. The first considers the semiflow on the "extinction boundary" of the state space, which can be facilitated by

Morse decompositions or acyclic decompositions of its point attractor (Chapter 8). This approach again relies on compactness assumptions and has a topological character (repeller-attractor pairs, chain-recurrence). The second uses so-called average Lyapunov functions. The concept of ρ-persistence can reconcile both approaches by either letting $\rho(x)$ be the distance from x to the boundary of extinction or by identifying ρ with the average Lyapunov function (Chapter 15). It also applies to the persistence of nonautonomous semiflows [**216**] (Chapters 13 to 15).

By example, we also present various ad hoc methods for proving uniform weak persistence. Typically, they work by contradiction. One is the method of fluctuations [**102**], together with differential inequalities and the Perron-Frobenius theory of quasipositive matrices (Appendix A and Chapter 3); another is the use of the Laplace transform. The resurgence of this classical tool is not so surprising as many semiflows are generated by nonlinear perturbations of linear semiflows (alias operator semigroups, Chapter 10) for which the Laplace transform is a major tool of investigation [**5**]. The Laplace transform can be quite effective (Chapter 5.7, Chapter 9) and also works in cases where the existence of a compact attractor cannot be established (Chapter 12).