

Contents

I. Setting the frame	1
1. Introduction to epidemics & networks	3
1.1. Epidemics	3
1.2. Complex networks	3
2. Theory	5
2.1. Models of infectious diseases	5
2.1.1. Development of mathematical epidemiology	5
2.1.2. Infection dynamics	6
2.1.3. SI model	6
2.1.4. SIR model	7
2.1.5. Force of infection	10
2.2. Network theory	10
2.2.1. Network matrix representations	11
2.2.2. Accessibility	12
2.2.3. Network measures	12
2.2.4. Implementation	12
2.2.5. From random mixing to developed structure	12
3. Outlook	13
II. Static Networks	15
4. Basics	17
5. Advanced	19

III. Temporal networks	21
6. Introduction	23
6.1. Temporal networks	23
6.2. Conceptual problems with components in temporal networks	24

Part I.

Setting the frame

1. Introduction to epidemics & networks

Livestock epidemics are a major economic issue.

1.1. Epidemics

1.2. Complex networks

A review on networks is found in Newman [16]. Analyses of livestock trade networks are in Christley [8] Bigras-Poulin et al. [5] Green et al. [9].

The interplay between aggregation window and spreading potential was analyzed in Bajardi et al. [3].

2. Theory

This chapter is devoted to the mathematical formalism that is used to model infectious diseases and networks. The author defines a mathematical framework and summarizes relevant results of earlier research in this area. In addition, section xx describes an efficient computer implementation of networks.

2.1. Models of infectious diseases

Observations. Large scale patterns of epidemics have been measured [?] The spread of infectious diseases is something that everyone is familiar with.

Research field: Epidemiology. One goal of epidemiology is to understand the principles behind the spreading process, i.e. the way how a disease is transmitted through a population. In this context, *conceptional* models are used. They make use of simple assumptions for the local (person-to-person) dynamics and focus on the big picture of the process. Conceptual models are very similar to models in theoretical physics, because they focus on the very essence of the problem (here: the macroscopic view, spreading patterns). However, they have to neglect many details of the real problem (here: physiology, symptoms, individual behavior, infection pathways and many more!) in order to have mathematical feasible models.

Another important issue of epidemiology is the *forecast* of epidemic spreading processes. Forecast models incorporate as much information as possible and the main focus is not an understanding of the basic principles.

This section summarizes the mathematical framework that roughly reproduces the behavior of infectious diseases and briefly discusses some major insights.

2.1.1. Development of mathematical epidemiology

The modeling of infection diseases mostly uses the concept of compartment models as explained in section xx. Major contributions to the modern theoretical framework were provided by [15], [2] and [1]. In his review about the mathematics of infectious diseases Hethcote reports a model for smallpox was already formulated in 1760 by D. Bernoulli ([12] and references therein). In the early 20th century, people developed mathematical models for epidemics: a discrete time model in 1906 [11] and a differential equation model in 1911 [18]. The epidemic threshold (section 2.1.4) was found in the 1920s [14]

[15]. Starting from Bailey's book [2] in the 1950s, the modeling of infectious diseases became a major scientific research field. Modern models of infectious diseases include vaccination, demographic structure, disease vectors, quarantine and even game theory ([4] and references in [12]). The availability of contact data in recent years led to a strong impact on network analysis on epidemiology. Well known concepts of mathematics (graph theory [6]) and social sciences (social network analysis [19]) have been adopted to disease modeling, since the connections between individuals are related to their epidemic spreading potential [13].

2.1.2. Infection dynamics

The spread of infectious diseases can be modeled in terms of compartment models as described in section xx. We differentiate between *conceptional models* and *realistic disease models*. While the former class is used to provide conceptual results as for the computation of thresholds or to test theories [12], realistic disease models use as many aspects as possible to provide a forecast of the spreading process. Realistic disease models can be very complex and are beyond the scope of this work, thus the author focuses on the use of conceptional models. The following section is inspired by the Lecture notes of J. R. Chasnov [7].

2.1.3. SI model

Let us consider a population of N individuals. In the simplest case, the infection status of each individual is either susceptible or infected and there are no births and deaths on the population. Susceptible individuals become infected, if they are in contact with an infected. This mimics the behavior of an infectious disease without immunization, i.e. infected individuals stay permanently infected.

Provided that α is the rate, under which new susceptible become infected, the SI-model is as follows:

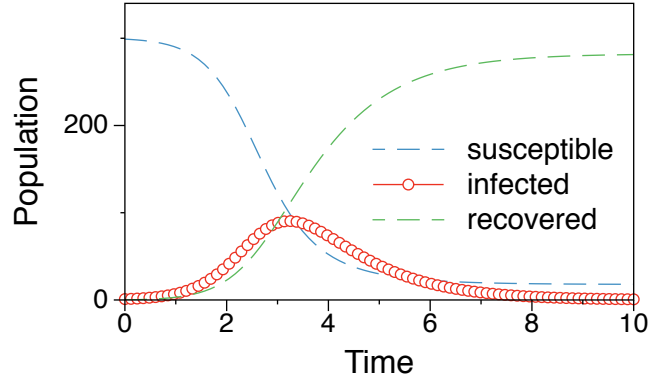
$$\begin{aligned}\frac{dS}{dt} &= -\alpha SI \\ \frac{dI}{dt} &= \alpha SI,\end{aligned}\tag{2.1}$$

where S and I are the numbers of susceptible and infected individuals respectively. The total population is $N = S + I$. Thus, (2.1) can be rewritten as

$$\frac{dI}{dt} = \alpha(N - I)I,$$

i.e. a logistic differential equation. Hence, in the limit $t \rightarrow \infty$ the whole population is infected ($I(\infty) = N$).

Figure 2.1. Solution of the susceptible-infected-recovered (SIR) model (2.2). The number of infected shows that the spreading process is a single event. Note that a fraction of the population is still susceptible at the end of the process. Parameters: $\alpha = 3$, $\gamma = 1$, $N = 300$, $S_0 = 1$.



2.1.4. SIR model

In contrast of the infection dynamics introduced in the previous section, many epidemics allow for an immunization of the individuals. Examples are measles or whooping cough [10] [1]. In this case, individuals recover from the disease after being infected for a certain time period. The infection scheme has to be extended to susceptible-infected-recovered (SIR) as in the following infection model [15]:

$$\begin{aligned}\frac{dS}{dt} &= -\alpha SI \\ \frac{dI}{dt} &= \alpha SI - \gamma I \\ \frac{dR}{dt} &= \gamma I\end{aligned}\tag{2.2}$$

where α is the infection rate and γ is the immunization or recovery rate. There is no analytic solution for the system (2.2), but some fundamental conclusions can be obtained analytically. We show a typical solution of (2.2) in figure 2.1.

The SIR model shows more sophisticated features than the SI model (2.1). To begin with, we analyze the fixed points of the system, i.e. (S_*, I_*, R_*) where

$$\frac{dS_*}{dt} = -\alpha S_* I_* = 0, \quad \frac{dI_*}{dt} = \alpha S_* I_* - \gamma I_* = 0, \quad \frac{dR_*}{dt} = \gamma I_* = 0.\tag{2.3}$$

It follows from the last equation that $I_* = 0$ at the fixed point, where S_* and R_* can be arbitrary. Hence, a fixed point is $(S_*, 0, R_*)$.

Let us analyze the stability of the fixed point in the early phase of an infection. Almost all individuals are susceptible and consequently $I_* = N - S_*$. An outbreak occurs, if

2. Theory

and only if $dI/dt > 0$ in this phase, i.e.

$$\frac{dI}{dt} = \alpha S_*(N - S_*) - \gamma(N - S_*) = (N - S_*)(\alpha S_* - \gamma) > 0. \quad (2.4)$$

It follows from (2.4) that the number of infected grows, if

$$\alpha S_*/\gamma > 1. \quad (2.5)$$

Equation (2.5) is extremely important in epidemiology, because it defines a threshold for the unfolding of an infection spreading process. We call this fraction the *basic reproduction number* R_0 . Recall that $S_* \approx N$ in the fixed point. Thus it follows that the outbreak condition is

$$R_0 = N \frac{\alpha}{\gamma} > 1. \quad (2.6)$$

The basic reproduction number describes the average number of follow-up infections by each infected individual. It is one of the main goals in epidemiology to bring down the basic reproduction number of a disease below the critical value $R_0 = 1$. This is the reason for the implementation of mass vaccination. As one can immediately see from equation (2.6), this can be done by reducing the infection rate α or by increasing the immunization rate γ . In principle, one could also reduce the size of the initial population S_* . As an example, reducing the infection rate can be done by increasing hygiene standards or appropriate behavior, say wearing warm clothes in winter time to avoid common cold. The immunization rate can be increased by vaccination.

Let us now focus on the late phase of an SIR-infection. In contrast to the SI-model of section 2.1.3 an SIR like outbreak does not necessarily infect the whole population, even if $R_0 > 1$. The reason is that there has to be a critical mass of susceptible individuals in order to keep an infection alive (see equation (2.5)). The total number of infected during an infection given by the number of recovered at the end of the infection, since every recovered has to be in the infected state in the first place. A central measure throughout this work is therefore the *outbreak size* R_∞ .

To compute the outbreak size, we consider the second fixed point of (2.2), i.e. the fixed point for $t \rightarrow \infty$. At this point there are no infected and a fraction of the population is recovered. Hence the fixed point is $(N - R_\infty, 0, R_\infty)$. A simple way to obtain the outbreak size R_∞ is to use equations (2.2) and compute

$$\frac{dS}{dR} = -\frac{\alpha}{\gamma} S$$

and separate the variables [7]. This yields

$$\int_{S_*}^{N-R_\infty} \frac{dS}{S} = -\frac{\alpha}{\gamma} \int_{R_*}^{R_\infty} dR.$$

We integrate from the initial condition at $t = 0$ to the final condition at $t \rightarrow \infty$, where $S_\infty = N - R_\infty$. Using that $R_* = 0$ at $t = 0$ gives

$$R_\infty = S_* - S_* e^{-\frac{\alpha}{\gamma} R_\infty}. \quad (2.7)$$

This transcendental equation can be solved numerically using a Newton-Raphson technique. The outbreak size R_∞ only takes finite values for $\alpha/\gamma > 1$. A solution of equation (2.7) is shown in figure 2.2

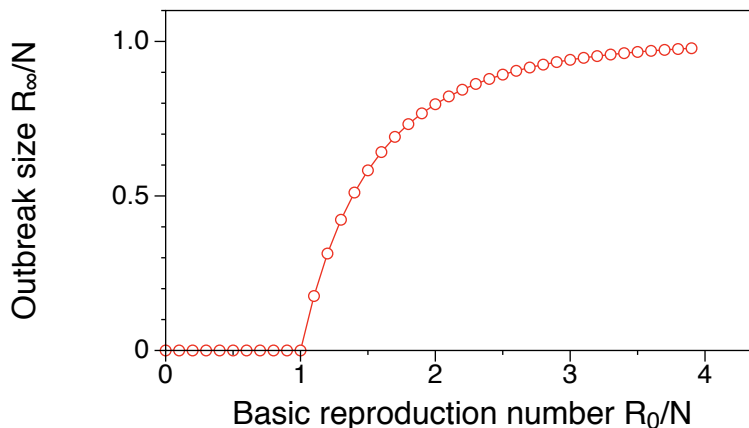


Figure 2.2. Relative outbreak size vs. basic reproduction number. The outbreak size takes finite values only for $R_0/N > 1$. Note that even for supercritical R_0 the outbreak size is in general smaller than the total population.

It should be noted that an SIR epidemic is a single event, i.e. it possesses a *characteristic time scale*. The analysis of the late phase of an epidemic also gives information about these time scales. Let us consider the second equation of (2.2).

$$\frac{dI}{dt} = \alpha SI - \gamma I \quad (2.8)$$

In the late phase of an SIR-type epidemic, the fraction of infected is small. Given sufficiently large values of R_0 , the fraction of recovered is also small in this phase (see figure 2.2). Thus, we neglect the quadratic term in (2.8). This gives $\frac{dI}{dt} = -\gamma I$, which has the solution

$$I(t) = I(0)e^{-\gamma t}.$$

Hence, the infection decays exponentially for large t and the inverse recovery rate $1/\gamma$ defines the characteristic time of the epidemic.

2.1.5. Force of infection

The model presented in section 2.1.4 describes only the very basic behavior of epidemic dynamics, and is therefore a conceptual model. However, it is one of the main objectives in epidemiology to have an understanding of the exact infection rates in the process. Infection rates their selves can cause complex infection dynamics.

The term αI used in section 2.1.4 is a special, very simple case of an infection rate. More generally, we have to replace αI by an abstract infection rate λ containing more information about the interaction between susceptible and infected individuals [13]. Thus, the equation for the infected becomes

$$dI/dt = -\lambda S - \gamma I.$$

The rate λ is called the *force of infection*. In principle, this parameter can be arbitrarily complex, because it contains detailed information about the mixing properties of the population. This information could be given as contact networks, demographic contact structures, etc.

In most cases, detailed information about mixing is not available. Instead, we assume *random mixing* of the population, i.e. every individual can be in contact with every other individual. This yields a transmission rate [13]

$$\lambda = \tau n \frac{I}{N} \equiv \beta \frac{I}{N}, \quad (2.9)$$

where τ is the transmission rate, n is the effective contact rate and I/N is the fraction of infectious contacts. It is therefore reasonable to replace the infection term α in (2.2) by β/N to explicitly include the force of infection. Nevertheless, the results presented in section 2.1.4 remain qualitatively the same.

Although the force of infection gives a more reasonable description of the infection process, the assumption of random mixing remains inappropriate for many real world systems. Due to the availability of contact data, the random mixing assumption can be improved in terms of contact networks. Even if the exact data of an epidemic system is not available, research on complex networks allows us to give more realistic models about mixing. In the next section, we briefly report important results in complex network research and focus on the interplay between networks and epidemics in section xx.

2.2. Network theory

As we have seen in the previous section, standard epidemic models make use of the random mixing assumption. This assumption seems reasonable, if no further information about the contact structure within a population is available, because it gives a worst case scenario of the infection dynamics. Even an overestimation of the outbreak size can

be corrected by introducing smaller, effective disease parameters. However, the random mixing assumption does not allow for non homogenous mixing, i.e. each individual is considered equal. Nevertheless, the equality of individuals is not a reasonable assumption for many epidemic substrates. Examples are contact structures of humans, livestock trade, vehicles as disease vectors of links between computers.

2.2.1. Network matrix representations

A network is a system consisting of nodes that are connected by edges. Edges can be undirected, directed and weighted. In principle, a network can consist of edges of different types. This can be represented by multiple networks sharing the same set of nodes, but different edges.

Networks are called graphs in mathematical literature. A graph $G = (V, E)$ is a set of nodes (or vertices) V and edges (or arcs) E , where each edge is given by the tuple of nodes it connects, i.e. $e_1 = (u, v) \in E$ connects nodes u and v . An edge (u, v) being present in an undirected network implies an edge (v, u) . Apparently, this does not hold in directed networks. Edges of weighted networks carry additional meta information about their weight. This meta information can be their importance, capacity, number of transported items or the geographical distance between nodes u and v .

Graphs can be represented by different graph matrices, where different matrix representation emphasize typical properties of the network. The most common graph matrix is the *adjacency matrix* \mathbf{A} with entries

$$(\mathbf{A})_{ij} = \begin{cases} 1 & \text{if } i \text{ is connected to } j \\ 0 & \text{else.} \end{cases} \quad (2.10)$$

An adjacency matrix contains the edges of the graph and can be seen of the most fundamental graph representation. Weighted networks can be represented by weight matrices, where the values of the entries in (2.10) are not restricted to zero and one.

Figure 2.3. A simple network.

The adjacency matrix of an undirected network is symmetric, because every non-zero entry $(\mathbf{A})_{ij} = 1$ implies an edge into the opposite direction, $(\mathbf{A})_{ji} = 1$. Entries on the main diagonal $(\mathbf{A})_{ii}$ correspond to nodes with self loops, i.e. nodes with edges pointing back to themselves. The i -th row the adjacency matrix contains non-zero entries $(\mathbf{A})_{ij} = 1$, whenever node i is connected to node j . Hence, every row can be interpreted as the neighborhood of one node. This holds for undirected and for directed networks. The columns of \mathbf{A} give the same information as the rows. In the directed

case, however, rows contain the out-neighborhood of each node and columns contain the in-neighborhood, respectively.

A Markov chain is a random process without memory and with discrete state space and discrete time. It is called time-homogenous, if the transition rates are constant. Time-homogenous Markov chains can be represented as weighted networks and the corresponding weighted adjacency matrix is the *transition matrix*. Transition matrices are stochastic matrices, i.e. the elements of every row sum up to unity. Each node represents a different state of the system and the edges are weighted with the probabilities to transition into other states adjacent to these edges. It is obvious that a transition matrix representation is useful to describe random walks on networks.

Figure 2.4. A Markov chain.

As a special case of transition matrices, the author would like to name the *Google matrix*. It describes a random walk on a network, but allows for shortcuts to any node in the network with a certain probability. The eigenvectors of Google matrices are used for the computation of node rankings according to the PageRank-Algorithm [17].

2.2.2. Accessibility

2.2.3. Network measures

Before we address ourselves to models of real world networks, we have to introduce methods to measure structural properties of networks. On the micro scale, this can be done in terms of *node centrality* measures. These measures are very important to assess the importance of single nodes in the network. On the macroscopic side, we are interested in the large-scale properties of networks, i.e. percolation, distributions of centralities, connected components or other large scale structures.

2.2.4. Implementation

2.2.5. From random mixing to developed structure

3. Outlook

We give a description of temporal networks later.

Part II.

Static Networks

4. Basics

5. Advanced

Part III.

Temporal networks

6. Introduction

6.1. Temporal networks

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6.2. Conceptual problems with components in temporal networks

Die Accessibility erlaubt eine Ansicht auf ein temporal network als Ganzes.

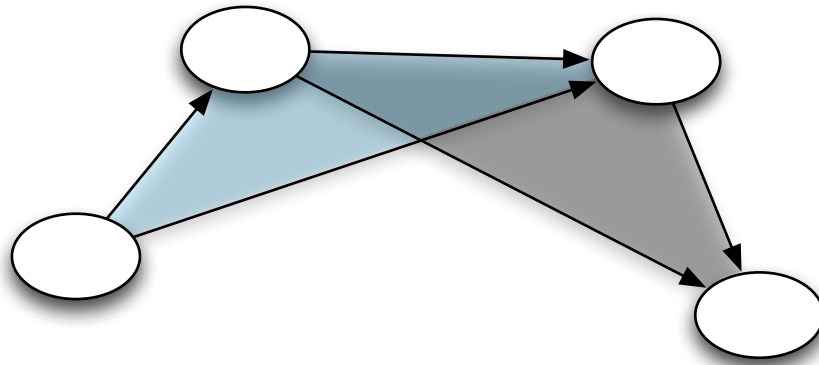


Figure 6.1.. Overlapping transitive hyperedges.

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