Chapter 8 Some Basic Epidemic Models



Danijela Rajter-Ćirić

8.1 Introduction

Spreading of infectious diseases has always been a threat to human health and people have been trying to fight against it (which is especially important nowadays when contagious diseases are spreading faster and further than ever). So far great achievements have been made. In order to prevent the spread of a particular disease, one should first try to understand and explain the mechanism how it spreads in the population. However, no experiments are possible due to ethical (and many other) reasons. Therefore, mathematical models present a very useful tool. Although they are only theoretical and usually simplify the real situation, they still describe the behaviour of population members well enough so that they can be successfully used for describing the dynamics of a disease, predicting epidemics, measuring the effects of some prevention measures, etc.

At the beginning of the twentieth century Dr. Ross, later awarded the Nobel Prize for Medicine for his significant contribution to research, used a differential equation model to describe malaria transmission between humans and mosquitoes. Later, William Kermack and Anderson McKendrick formulated a model to study the Black Death outbreak in London and the plague outbreak in Mumbai. They published their results in 1927 in the paper "A Contribution to the Mathematical Theory in Epidemic". They have used one of the simplest forms of, so-called, SIR model which has been studied, improved and generalized afterwards by many authors.

Today there are many different mathematical epidemic models and mathematical approach to the epidemic modelling is widespread. In this paper we introduce

D. Rajter-Ćirić (\boxtimes)

Department of Mathematics and Informatics, Faculty of Sciences, University of Novi Sad, Novi Sad, Serbia

e-mail: rajter@dmi.uns.ac.rs

[©] Springer Nature Switzerland AG 2020

E. Lindner et al. (eds.), Mathematical Modelling in Real Life Problems,

Mathematics in Industry 33, https://doi.org/10.1007/978-3-030-50388-8_8

the readers to some of these models. All mathematical models can roughly be divided into two groups: deterministic and stochastic models. In the paper, we first describe a few deterministic models for spreading of a contagious diseases in a large population, which are based on the, so-called, mass action law (population members make contacts to other members independently of each other and each individual has an equal chance of contacting any other individual). Further on, we present a few ideas of how a stochastic approach can be used in epidemic modelling. A stochastic approach is reasonable and it is justified by the fact that a population does not behave in a precisely determined way as it is assumed in deterministic models.

It is important to emphasize that in this paper models are presented on a very basic level, without complicated mathematical proves and getting deeper into the theory. The paper should simply serve to introduce readers into a beautiful field of applied mathematics called epidemic modelling and to present how nicely mathematics can be applied to such a serious research area. There are no original results in the paper. The models presented here have been considered in many student books and papers.

8.2 Some Deterministic Models

Deterministic models assume that the population behaves exactly as assumed in the model and there are no randomness in the behavior of the population. The population is large and divided in groups by the epidemic state of individuals. The number of groups depends on the disease and hence on the model, as the reader will see. Here we present only some of many models. In all of presented models ordinary differential equation approach has been proposed. Although in most cases, corresponding systems of ordinary differential equations describing the model are not solvable (if not analytically, these ordinary differential equation systems can be solved numerically), they still play a significant role in the mathematical analysis of the disease spread and in the prediction of epidemics. For more about deterministic epidemic models we refer the reader, for instance, to [2, 3].

8.2.1 SIR Model

An SIR model is a very simple epidemic model that one can use to calculate the number of individuals infected with an epidemic (a contagious) disease in a large population over time. One of the simplest SIR models is the Kermack-McKendrick model.

We consider the population of size N, where N is a constant, and assume that the population consists of three types of individuals based on the state of the individual concerning the disease. In this model we assume that there are only three possible states: a subject is sensitive, infected or immune to a virus. Therefore, the

population is divided into three different groups. The first group consists of those individuals who have not developed immunity against the virus. That is the group of susceptibles (population members that are not infected but could become infected). The second group consists of infectives (subjects who are infected with the virus which means that they have the disease and can transmit it to the members of the group of susceptibles). Finally, members of the third group are individuals who have recovered from the disease and gained lasting immunity or who have died from the disease. In both cases, those individuals are said to be removed. (Some authors call the third group Recovered instead of Removed since they consider the individuals who have recovered, since both recovered and dead are, in some sense, immune to the virus).

So, basically, there is a very simple "rule" in SIR model: After becoming infected a susceptible subject immediately enters the infected group. Afterwards (after recovering or dying from the disease) the subject enters the group of removed.

The numbers of group members for these three groups are denoted by the letters S, I and R, respectively, which is the reason why this is called SIR model. All these numbers are actually functions of time *t*:

- S(t) denotes the number of susceptibles at time t
- I(t) denotes the number of infectives at time t
- R(t) denotes the number of removed at time t.

In the simplest SIR model that we first consider, a short time scale has been assumed so that births and deaths (other than deaths from this disease) can be neglected. One can consider the case when births and deaths are taken into account which yields to a slightly more complicated model as we will see later.

The usual assumptions for SIR model are:

- 1. Individuals infect each other directly rather than through disease vectors.
- 2. Contacts between individuals are random.
- 3. Immediately after a contact with the infected person, susceptible person shows symptoms and can infect someone else.
- 4. An arbitrary population member makes βN contacts (within the population) in a unit of time, where β denotes disease transmission rate.
- 5. The number of population members that move from the group of infectives to the group of removed in the unit of time is $\alpha I(t)$, where α denotes, so-called, recovery rate.

Now we want to answer the question: How S(t), I(t) and R(t) vary with time?

As an answer, the SIR model proposes a system of ordinary differential equations representing the transition from one group to another. More precisely, the numbers of susceptibles, infectives and removed change according to the system:

$$S'(t) = -\beta S(t)I(t) \tag{8.1}$$

$$I'(t) = \beta S(t)I(t) - \alpha I(t)$$
(8.2)

$$R'(t) = \alpha I(t). \tag{8.3}$$

Assuming that every population member belongs to one of the three groups one has that, at every time t,

$$S(t) + I(t) + R(t) = N.$$

Therefore Eq. (8.3) can be omitted.

Note that, based on one of the model assumptions, every infected individual makes βN different contacts i.e., contacts with βN members of population, but the chance to make the contact with a susceptible person is $\frac{S}{N}$. Thus

$$\beta N \ \frac{S}{N} \ I = \beta S I$$

is the number of individuals who move from the susceptible group to the group of infected in unit of time. Therefore, S(t) decreases, while I(t) increases for that number. This explains the differential equations in the model above.

The dynamics of the infectious group depends on the ratio $R_0 = \frac{\beta}{\alpha}$. The number

$$R_0 = \frac{\beta N}{\alpha}$$

is the, so-called, basic reproduction number and it represents the expected number of new infections from a single infection in a population where all subjects are susceptible. (For more about this number we refer the reader, for instance, to [4].)

The basic reproduction number is very important since it is a good epidemic indicator. If $R_0 > 1$, many susceptible individuals will be infected, i.e., the epidemic will start. If $R_0 = 1$ the disease becomes endemic. If one wants to prevent the epidemic, it is necessary to keep R_0 less than 1. For instance, the vaccination is a possible way for keeping the basic reproduction number lower than 1. Assume that p is the proportion of population members who have been successfully vaccinated before the appearance of the first infected individual. For preventing the epidemic the following condition has to be satisfied:

$$R_0 = \frac{\beta}{\alpha} (1-p)N < 1,$$

i.e.,

$$p > 1 - \frac{1}{R_0} = 1 - \frac{\alpha}{\beta N}.$$

In the model above the function $F = \beta I$ models the transition rate from the group of susceptible individuals to the group of infectious individuals. Therefore, it is called the force of infection. For many contagious diseases it is more realistic to consider a force of infection that does not depend on the absolute number of infectious subjects, but on their fraction $F = \beta \frac{I}{N}$. Some authors have even proposed nonlinear forces of infection to model more realistically the processes of contagious diseases.

Let us just briefly mention the more general case when birth and death rates influence the model. Suppose that λ denotes the birth rate and that μ denotes the death rate in the population. We still assume that the size of the population is constant. In that case, the SIR model is described by the following system:

$$S'(t) = \lambda - \mu S(t) - \beta I(t)S(t)$$
(8.4)

$$I'(t) = \beta I(t)S(t) - \alpha I(t) - \mu I(t)$$
(8.5)

$$R'(t) = \alpha I(t) - \mu R(t).$$
 (8.6)

Also, one can go a step further and study the (more realistic) SIR model that includes the vital dynamics (birth and death rates) in the population of size which is not a constant anymore, but varies with time. Here we will not consider that case.

8.2.2 SEIR Model

Now we present a modification of the SIR model that is very realistic for many infectious diseases. Instead of the assumption that after every contact with an infected subject a susceptible subject gets immediately infected and can infect others, here we assume that there is an incubation period during which the individuals have been infected but are not yet infectious themselves. That period is significant for many contagious diseases. Therefore, in this model another group of population members is formed - the group of exposed members (individuals who are in the incubation period), which means that now the population is divided in four groups: susceptible, exposed, infected and removed. Newly infected members do not immediately move from the susceptible group to the infected group, but first they go into the exposed group. Same as in SIR model, after being in the group of infected, subjects move to the group of removed.

Denote by E(t) the number of exposed individuals at time t.

Assuming that the average of the incubation period is κ^{-1} and that births and deaths (other than deaths due to the disease) have no influence to the model, the SEIR model is represented by the following system of ordinary differential equations:

$$S'(t) = -\beta S(t)I(t) \tag{8.7}$$

$$E'(t) = \beta S(t)I(t) - \kappa E(t)$$
(8.8)

$$I'(t) = \kappa E(t) - \alpha I(t). \tag{8.9}$$

The same as in the SIR model that we considered above, it is enough to consider only three out of four differential equations, since S(t) + E(t) + I(t) + R(t) = Nis a constant.

If infectivity of an exposed person can be reduced by some factor δ then one obtains more general SEIR model represented by the system:

$$S'(t) = -\beta S(t)I(t) - \delta\beta S(t)E(t)$$
(8.10)

$$E'(t) = \beta S(t)I(t) + \delta\beta S(t)E(t) - \kappa E(t)$$
(8.11)

$$I'(t) = \kappa E(t) - \alpha I(t). \tag{8.12}$$

Note that Eq. (8.10) describes the following: The number of susceptible subjects decreases by contacts with an infected subject or with an exposed subject but not every contact with an exposed person leads to infection transmission (the number of contacts that lead to infection is reduced by factor δ).

The basic reproduction number is now given by:

$$R_0 = \frac{\beta N}{\alpha} + \frac{\delta \beta N}{\kappa}.$$

It shows how many subjects can be infected by one exposed subject entering the group *S* and it can be explained in the following way: An exposed person makes $\frac{\beta N}{\kappa}$ different contacts in the group during the incubation period of the length $\frac{1}{\kappa}$, but not every contact leads to infection, as we mentioned above. Therefore there are $\frac{\delta\beta N}{\kappa}$ newly infected subjects. After the incubation period, the exposed person from above becomes infected and can make $\frac{\beta N}{\alpha}$ contacts but now every contact leads to the infection transmission.

In the end, let us briefly remark that, similarly as in SIR model, one can consider SEIR model assuming the presence of birth and death rates. It is very common to consider the case with birth and death rates that are equal. If μ denotes the birth/death rate, one has the model:

$$S'(t) = \mu N - \mu S - \beta \frac{I}{N}S$$
$$E'(t) = \beta \frac{I}{N}S - (\mu + \kappa)E$$
$$I'(t) = \kappa E - (\alpha + \mu)I$$
$$R'(t) = \alpha I - \mu R.$$

108

Here we wrote all four equations although we could have omitted the last one since S(t) + E(t) + I(t) + R(t) = N is a constant due to the assumption that birth and death rates are equal. However, in general N is a variable, not a constant.

8.2.3 SLIAR Model

This model includes a, so-called, latent period during which the person is infected, but there are still no symptoms of the disease and the person cannot transmit the virus to other members of the population. This is the case with influenza, and some authors call this model an influenza model.

For this model first it is necessary to form a population group of individuals that are in the latent period. When an individual gets out from the latent period, symptoms of the disease may or may not develop. If symptoms develop, then the individual moves into the infected group, and if that does not happen, then the person is in the, so-called, asymptomatic period when he or she does not have the symptoms of the disease but can transmit the infection to the others with a reduced factor ε . So, the model requires one more group to be formed—the group that consists of population members who are in the asymptomatic period.

Denote by L(t) the number of population members who are in the latent period and by A(t) the number of population members who are in the asymptotic period.

We also assume that the proportion of p out of the total number of those who are in the latent period goes into the infected group, which implies that the proportion of 1-p goes into the group of those who are in the asymptomatic period.

The model is called SLIAR model by the first letters of the names of five groups: Susceptible, Latent, Infected, Asymptotic and Removed.

The system of ordinary differential equation that describes the SLIAR model is:

$$S'(t) = -\beta S(t) [I(t) + \varepsilon A(t)]$$

$$L'(t) = \beta S(t) [I(t) + \varepsilon A(t)] - \kappa L(t)$$

$$I'(t) = p\kappa L(t) - \alpha I(t)$$

$$A'(t) = (1 - p)\kappa L(t) - \eta A(t).$$

From the first equation one sees that the number of susceptibles decreases after the contact with an infected subject or with a subject which is in asymptotic period (but not every contact with a subject in an asymptotic period yields to the infection, that is why one has ε multiplying A in the equation). As in previous cases, the equation that shows how R(t) varies has been omitted due to the fact that number S(t) + L(t) + I(t) + A(t) + R(t) = N is a constant.

The basic reproduction number in the SLIAR model is:

$$R_0 = p \frac{\beta N}{\alpha} + (1-p) \frac{\varepsilon \beta N}{\eta}$$

and it shows how many susceptible population members get infected by one subject who is in the latent period.

8.2.4 SIS Model

This model describes the disease that is endemic. It is a model of a disease in which the infected do not acquire immunity after recovery. This means that there are only two groups here: the group of the vulnerable and the group of the infected ones. After recovery the infected subjects return back to the sensitive group. Such a model can be applied in modeling the spread of diseases caused by a bacterium, because then immunity is not acquired against a new infection caused by the same bacterium. A model that does not include birth and death rates will be considered first. We again assume that the population size is constant. Then the system of differential equations corresponding to this model is as follows

$$S'(t) = -\beta S(t)I(t) + \alpha I(t)$$
$$I'(t) = \beta S(t)I(t) - \alpha I(t).$$

Since N = S(t) + I(t), for every t, the previous system reduces to the equation

$$I'(t) = \beta \left[N - I(t) \right] I(t) - \alpha I(t)$$

The equation above can be written in the form:

$$I' = \beta I \left(M - I \right),$$

where $M = N - \frac{\alpha}{\beta}$. The last equation can easily be solved:

$$I(t) = \frac{M}{\exp\{-M(\beta t + c)\} + 1}, \text{ for } M > I$$
$$I(t) = \frac{-M}{\exp\{-M(\beta t + c)\} - 1}, \text{ for } M < I$$

However, in any case one can see that the following holds:

- If M > 0 then $\lim_{t \to \infty} I(t) = M$.
- If $M \le 0$ then $\lim_{t \to \infty} I(t) = 0$.

The basic reproduction number in this model is the same as in SIR model: $R_0 = \frac{\beta N}{\alpha}$. Therefore $M = N\left(1 - \frac{1}{R_0}\right)$ and one concludes the following:

- If $R_0 > 1$ then $\lim_{t\to\infty} I(t) = M$ i.e., the disease remains in the population
- If $R_0 \le 1$ then $\lim_{t\to\infty} I(t) = 0$ i.e., the disease vanishes.

Finally, let us just mention that one can consider the SIS model with birth and death rates both being equal to μ and obtain a generalization of the previous model:

$$I'(t) = \beta [N - I(t)] I(t) - (\mu + \alpha) I(t).$$
(8.13)

8.3 Some Stochastic Models

The assumption that a population behaves exactly as assumed in the model is not very realistic. There are always some randomness that affect the population behavior. Therefore, a stochastic approach to the epidemic modelling problems is reasonable. There are different stochastic approaches depending on many factors and hence there are many different stochastic models. One of the simplest approaches is the one that uses discrete-time Markov chain models. Thus here we present a model of that type first. Some other stochastic models involve stochastic differential equations. Here we just briefly mention one of such models. Finally, there are many stochastic processes that can be used in epidemic modelling and here we present how a Poisson process can be used.

8.3.1 SIS Model in the Form of Discrete-Time Markov Chain

In this section we describe the SIS model with birth and death effects in a form of discrete-time Markov chain (see [1] for details). We assume that birth and death rates are equal and denoted by μ . The population size is constant and denoted by N. Therefore, as we concluded in the SIS deterministic case, it is enough to consider only one variable and this will (again) be the number of infected subjects, I(t).

So, we consider a stochastic process $\{I(t), t \in T\}$, where $T = \{0, \Delta t, 2\Delta t, \ldots\}$, as a discrete-time Markov chain. From the epidemic point of view it is reasonable to assume that the number of infectives at a time moment depends only on the number of infectives in the previous moment, so it is reasonable to assume that I(t) satisfies the Markov property.

As we saw in the deterministic case (see (8.13)) the following holds:

$$I'(t) = \beta [N - I(t)] I(t) - (\mu + \alpha) I(t).$$

Since I(t) is number of infected subjects at time t it is obvious that the set of possible states in this case is $S = \{0, 1, ..., N\}$. The probability that process I is in the state $i \in S$ at time t, is denoted by p(t), i.e. $p(t) = P\{I(t) = i\}$. The, so-called, probability vector is given by

$$p(t) = [p_0(t), \ldots, p_N(t)]^T$$

and $p_0(t) + p_1(t) + \dots + p_N(t) = 1$.

The next step is to determine the transition probabilities from one state to another for a short period of time Δt :

$$p_{ij}(t + \Delta t) = P\left\{I(t + \Delta t) = j | I(t) = i\right\}.$$

Based on the deterministic case, we assume that the Markov chain I(t) is homogeneous i.e., that transition probabilities do not depend on time. Thus, we can write $p_{ij}(\Delta t)$ instead of $p_{ij}(t + \Delta t)$.

In order to make the model as simple as possible, we also assume that Δt is small enough so that during that time period the number of infected subjects can change for one at most, i.e., there are three possible state changes:

$$i \to i+1, i \to i-1 \text{ or } i \to i.$$

Now the transition probabilities are given by:

$$p_{ij}(\Delta t) = \begin{cases} \beta \ i \ (N-i) \ \Delta t, & j = i+1 \\ (\mu + \alpha) \ i \ \Delta t, & j = i-1 \\ 1 - [\beta i (N-i) + (\mu + \alpha)i] \ \Delta t, & j = i \\ 0, & \text{otherwise} \end{cases}$$
(8.14)

If we set $b_i := \beta i (N - i)$ and $d_i := (\mu + \alpha)i$ we obtain:

$$p_{ij}(\Delta t) = \begin{cases} b_i \Delta t, & j = i+1 \\ d_i \Delta t, & j = i-1 \\ 1 - [b_i + d_i] \Delta t, & j = i \\ 0, & \text{otherwise} \end{cases}$$
(8.15)

Note that Δt has to be small enough to provide that $p_{ij} \in [0, 1]$. Therefore, the following must hold:

$$\max_{i \in \{1, ..., N\}} \{ (b_i + d_i) \Delta t \} \le 1.$$

Using the transition probabilities from (8.15) one can determine the probability that there are *i* infected subjects at time $t + \Delta t$:

$$p(t+\Delta t) = p_{i-1}(t)b_{i-1}\Delta t + p_{i+1}(t)d_{i+1}\Delta t + p_i(t)\left(1 - [b_i + d_i]\Delta t\right), \quad i = 1, \dots, N.$$

Finally, although we will not prove it here, let us mention that for expected number of infected subjects the following holds:

$$E(I(t + \Delta t)) = E(I(t)) + [\beta N - (\mu + \alpha)] E(I(t))\Delta t - \beta E(I^{2}(t))\Delta t$$

Using the fact that $E(I^2(t)) \ge E^2(I(t))$ and letting $\Delta t \to 0$ one obtains that

$$\frac{dE(I(t))}{dt} \le \beta \left[N - E(I(t)) \right] E(I(t)) - (\mu + \alpha) E(I(t)).$$
(8.16)

8.3.2 A Note on Stochastic Differential Equation for SIS Model

Here we introduce and just briefly describe the stochastic differential equation for SIS model. For details we refer the reader to [5].

As already mentioned, for the SIS model Eq. (8.13) holds. This equation can be written as

$$dI(t) = \beta (N - I(t)) I(t) dt - (\mu + \alpha) I(t) dt.$$
(8.17)

Consider the first summand in the sum above: $\beta (N - I(t)) I(t)dt = \beta S(t)I(t)dt$. It represents the number of the newly infected individuals in the time interval od the length dt. If we make a reasonable assumption that β is actually the random variable and that instead of βdt in (8.17) one can write $\beta dt + \sigma dW(t)$, where W(t) is standard Brownian motion (Wiener process), then we obtain a stochastic differential equation:

$$dI(t) = I(t) \left(\left[\beta(N - I(t)) - (\mu + \alpha) \right] dt + \sigma \left(N - I(t) \right) dW(t) \right).$$
(8.18)

One can prove the following: If $R_0^S = R_0 - \frac{\sigma^2 N^2}{2(\mu + \alpha)} = \frac{\beta N}{\mu + \alpha} - \frac{\sigma^2 N^2}{2(\mu + \alpha)} < 1$ and $\sigma^2 \le \frac{\beta}{N}$ then, for every initial data $I(0) \in (0, N)$, the solution I(t) to stochastic differential equation (8.18) exponentially tends to zero, almost surely. In another words, the disease vanishes with probability 1.

8.3.3 A Poisson Process Model for Tracking the Number of HIV Infections

We present a very simple Poisson process model for tracking the number of HIV infections, as done in [6].

One of many difficulties with HIV infection is the fact that the incubation period is relatively long. So, there may be many individuals who are infected with the virus but still not showing the symptoms. The following model is a very simple approximation model that helps obtaining a rough estimate of the number of such individuals.

In this model we assume that

- HIV infections appear in accordance with a Poisson process with unknown rate λ ,
- the time from the moment when an individual becomes infected until symptoms of the disease appear is a random variable that has a known distribution *G*,
- the incubation periods of different infected individuals are independent.

Let $N_1(t)$ denotes the number of individuals who have shown symptoms of the disease by time t and $N_2(t)$ denotes the number of individuals who are HIV positive but still don't show any symptoms of the disease by time t.

Since a subject who gets infected at time *s* will have symptoms by time *t* with probability G(t-s) and will not with probability $1-G(t-s) = \overline{G}(t-s)$, it follows that $N_1(t)$ and $N_2(t)$ are independent Poisson random variables with means

$$E(N_1(t)) = \lambda \int_0^t G(t-s) \, ds = \lambda \int_0^t G(y) \, dy,$$
$$E(N_2(t)) = \lambda \int_0^t \bar{G}(t-s) \, ds = \lambda \int_0^t \bar{G}(y) \, dy.$$

Since λ is unknown, we must estimate it. Suppose that we have reliable records and that we know how many individuals are ill by time *t*. Denote that number by n_1 . Then we can estimate that

$$n_1 \approx E(N_1(t)) = \lambda \int_0^t \bar{G}(y) \, dy.$$

8 Some Basic Epidemic Models

So, we can estimate λ by $\tilde{\lambda}$ given by

$$\tilde{\lambda} = \frac{n_1}{\int_0^t G(y) \, dy}.$$

Using this estimation of λ , we can estimate the number of infected individuals with no symptoms at all at time *t* by

$$\tilde{N}_2(t) = \tilde{\lambda} \int_0^t \bar{G}(y) \, dy = n_1 \frac{\int_0^t \bar{G}(y) \, dy}{\int_0^t G(y) \, dy}$$

If, for example, G is exponential with mean μ , then $\overline{G}(y) = e^{-\frac{y}{\mu}}$, and

$$\tilde{N}_2(t) = \frac{n_1 \mu (1 - e^{-\frac{t}{\mu}})}{t - \mu (1 - e^{-\frac{t}{\mu}})}.$$

In [6] Ross gives the following concrete example based on the previous assumptions and calculations: If we suppose that t = 16 years, $\mu = 10$ years, and $n_1 = 220,000$, then the estimation of the number of infected but symptomless individuals at time 16 is

$$\tilde{N}_2(16) = \frac{220 \cdot 10(1 - e^{-1.6})}{16 - 10(1 - e^{-1.6})} = 218.96.$$

So, if the incubation period is exponential with mean 10 years and if the total number of individuals who have AIDS symptoms during the first 16 years of the epidemic is 220,000, then we can expect that approximately 219,000 individuals are HIV positive but with no symptoms at time 16.

So, the model above can be used for getting a rough estimation of number of HIV infections. However, the assumption that the infection rate λ is a constant is not very realistic. It would be much better to use an infection rate that changes over time.

References

- Allen, L.J.S.: An Introduction to Stochastic Epidemic Models. In: Brauer F., van den Driessche P., Wu J. (eds) Mathematical Epidemiology. Lecture Notes in Mathematics, Vol 1945. Springer, Berlin, Heidelberg (2008)
- Brauer, F., Castillo-Chavez, C.: Epidemic Models. In: Mathematical Models in Population Biology and Epidemiology. Text in Applied Mathematics, Vol 40. Springer, New York, NY (2012)
- 3. Brauer, F., Castillo-Chavez, C.: Models for Endemic Diseases. In: Mathematical Models in Population Biology and Epidemiology. Text in Applied Mathematics, Vol 40. Springer, New York, NY (2012)

- 4. Brauer, F., Driessche, P.V.D., Wu, J.: Mathematical Epidemiology. Springer Science and Business Media, Berlin (2008)
- 5. Gray, A., Greenhalgh, D., Hu, L., Mao, X., Pan, J.: A Stochastic Differential Equation SIS Epidemic Model. University of Strathclyde, Glasgow, Donghua University Shanghai (2006)
- 6. Ross, S.: Introduction to Probability Models (Tenth Edition). Academic Press as an Imprint of Elsevier (2010)