

EPIDEMSE REPORT

A General Integral Equation Model for Epidemics



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Preface

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Especially when an infectious disease is new, mathematical models help to answer the two most important questions: How will the infection spread and what actions are appropriate and effective to contain it? In order to support local decision makers (health authorities, hospitals and especially municipalities) in planning their actions, researchers of the Fraunhofer ITWM are working on the epidemiological modeling, simulation and decision support of Covid-19 within the framework of the Fraunhofer Society's Anti-Corona Program (project name Epi-DeMSE).

Abstract

We formulate an epidemic model based on an integral equation and identify common SEIR and dSEIR models as special cases. Different properties of the general model, such as the initial development of the epidemic and the infestation, are described as functions of the integral kernel, and a formula for the reproduction number is given. This provides a common basis for comparing the SEIR and dSEIR models. Although the model parameters can be chosen such that the SEIR and dSEIR models agree with respect to certain aspects concerning initial growth and final infestation, they are different in general. We choose two reasonable methods of parameter identification for a comparison by means of analytical and numerical methods. Finally, we demonstrate that the resulting reproduction rates depend on the chosen epidemic model.

Introduction

A first mathematical model for the spread of smallpox was established as early as 1766 by Daniel Bernoulli [Ber60]. It already represented the archetype of the compartment-based models which divide the population into groups defined according to their current state of health. The models in their present form date back to the so-called SIR model by Kermack and McKendrick [KW27] from 1927, where the population is split into susceptible (S), infectious (I) and recovered/removed (R) individuals. The exchange between the compartments is modeled by means of balance differential equations with transfer rates. An improvement are the so-called SEIR-models where the compartment of exposed (E) is added, to take account of the latency between infection and outbreak of the disease. Such models and variations thereof are well established. A good overview is presented in [Het00]. Closely related to these models are the dSEIR-models using time delays instead of transfer rates, leading to delay differential equations, see [Coo67, Het95, Het00, EB01, KK03, YL06, BB11, RA12]. Such models better reflect the real individual course of the disease.

In this work, we present and discuss an integral equation model which generalizes both types of models mentioned above, the SEIR-models as well as the dSEIR-models. Depending on the choice of the integral kernel, the integral equation model is shown to be equivalent to any of these models. As it turned out, this integral equation model for the more special case of a constant infection rate was already formulated in the paper by Kermack and McKendrick [KW27] in 1927 where the classical SIR model was derived as a special case. In [BDdG⁺12] it is pointed out that although this paper has become a classic in theoretical biology and has been cited countless times as the forefather of the SIR model, the much more general integral equation model formulated there is hardly known.

The main feature of such integral models is that the infectious activity of individuals can depend on the time since infection in any way described by the integral kernel. In order to enable the modelling of a real epidemic where contact rates change, e. g. due to political measures, we have additionally introduced a possibly non-constant infection rate.

Integral equation associated with epidemics

In principal, any epidemic dynamics driven by infections only, allows for a description in terms of an integral equation

$$s'(t) = \int_{-\infty}^{\infty} k(t, t') s'(t') dt', \quad s(-\infty) = 1 \quad (s' := d/dt s), \quad (1)$$

with $s(t) = S(t)/S_{all}$ being the proportion of the number $S(t)$ of never infected individuals, usually called the susceptible, at time t . S_{all} is the absolute number of all individuals under consideration. The kernel $k(t, t')$ is the rate by which individuals at time t are infected by individuals which themselves have been infected at time t' . Of course, $k(t, t') \geq 0$ for all (t, t') , $k(t, t') = 0$ if $t < t'$ and $s(t) \in [0, 1] \forall t \in (-\infty, \infty)$.

An important quantity of the epidemics dynamics, which is intuitive to grasp, is the reproduction number

$$R(t') = \int_{-\infty}^{\infty} k(t, t') dt, \quad (2)$$

the mean total number of individuals being infected by a single individual which itself has been infected at time t' (note that integration is now with respect to t , not t').

Integral equation model

Although k is a real existing quantity of any epidemic, it cannot usually be measured directly. The same is true for R . At the same time k cannot be uniquely reconstructed from the knowledge of $s(t)$. Therefore, and in order to get a usable model, we split $k(t, t')$ into a factor $\gamma(t)$ and a convolution factor $\Theta(t - t')$. For example, the first could be considered as the factor that reflects current social contact behavior, while the second would reflect the characteristic and time-invariant course of disease. Thus, we use

$$k(t, t') := s(t) \gamma(t) \Theta(t - t') \quad (3)$$

with $\gamma(t), \Theta(t) \geq 0$ for all t and $\Theta(t) = 0$ if $t < 0$. We also assume that $\int_{-\infty}^{\infty} \Theta(\tau) d\tau$ exists and finally obtain the integral equation model (**iS**)

$$s'(t) = s(t) \gamma(t) \int_{-\infty}^{\infty} \Theta(t - t') s'(t') dt' =: -\gamma(t) s(t) i[s](t). \quad (4)$$

In consequence the expression for the reproduction number becomes

$$R(t') = \int_{-\infty}^{\infty} s(t) \gamma(t) \Theta(t - t') dt. \quad (5)$$

Assuming Θ to be non trivial, i. e. non vanishing and $\gamma(t) > 0$ everywhere, the solutions of equation (4) have the following important property:

If there is some t_0 such that $s'([-\infty, t_0]) < 0$, then $s'([-\infty, \infty]) < 0$.

This can easily be proven, using $s(-\infty) = 1$ and showing that the interval I with $s'(I) > 0$ is open and closed at the same time, which follows from the continuity of s' and the positiveness of $\gamma(t)\Theta(t-t')$. In the sequel we will study (some of) the further properties of this model and identify the well known SEIR and dSEIR models as special cases. This also facilitates our subsequent comparison of the latter.

SEIR and dSEIR models

To formulate these models, we use $s(t)$, $e(t)$, $i(t)$, $r(t)$ to denote the time dependent proportion of susceptible, exposed, infectious and recovered expressed as ratios of the total population. Of course,

$$s(t), e(t), i(t), r(t) \in [0, 1] \quad \forall t \in (-\infty, \infty) \quad (6)$$

$$\text{and} \quad s(t) + e(t) + i(t) + r(t) = 1 \quad \forall t \in (-\infty, \infty), \quad (7)$$

which means that the evolution of (s, e, i, r) takes place within the standard simplex of 4-space. Then, the **SEIR** model is given by the ODE

$$s'(t) = -\gamma(t) s(t) i(t) \quad (8)$$

$$e'(t) = -s'(t) - \sigma e(t) \quad (9)$$

$$i'(t) = \sigma e(t) - \alpha i(t) \quad (10)$$

$$r'(t) = \alpha i(t) \quad (11)$$

with $\gamma(t) > 0, \sigma > 0, \alpha > 0$. Accordingly, the **dSEIR** model can be written in terms of the DDE

$$s'(t) = -\gamma(t) s(t) i(t) \quad (12)$$

$$e'(t) = -s'(t) + s'(t - \tau_e) \quad (13)$$

$$i'(t) = -s'(t - \tau_e) + s'(t - \tau_i) \quad (14)$$

$$r'(t) = -s'(t - \tau_i) . \quad (15)$$

Integrating eq. (14) (with $i(t \rightarrow -\infty) = 0$) and inserting this into eq. (12) yields the equivalent single equation formulation of the **dSEIR** model:

$$s'(t) = -\gamma(t) s(t) (s(t - \tau_i) - s(t - \tau_e)). \quad (16)$$

Obviously, we allowed for a non constant $\gamma(t)$, while treating σ and α as constants, which directly fits to our choice of the integral kernel, eq. (3).

Now, both the dSEIR-model and the SEIR-model can be identified as special cases of the **iS** model (4) if we use

$$\Theta_{dSEIR}(\tau) = \begin{cases} 1 & \text{if } \tau_e \leq \tau \leq \tau_i \\ 0 & \text{else} \end{cases} \quad (17)$$

$$\Theta_{SEIR}(\tau) = \frac{\sigma}{\sigma - \alpha} \left(\exp(-\alpha\tau) - \exp(-\sigma\tau) \right) \quad (18)$$

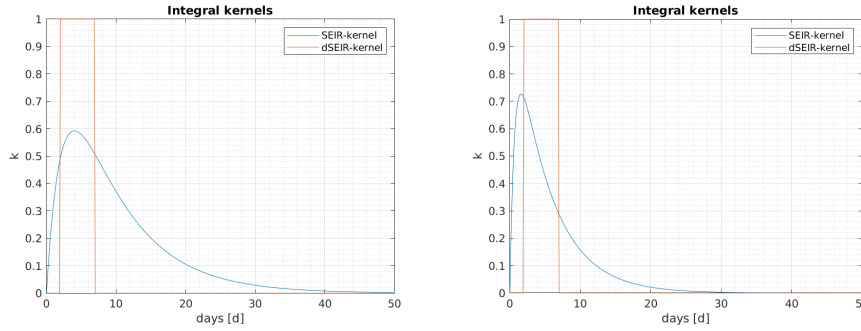


Fig. 1: Examples of convolution kernels for SEIR and dSEIR models.

as convolution kernels. Thereby, Θ_{dSEIR} is obvious from eq. (16), whereas Θ_{SEIR} is derived from equations (9) and (10) by means of the variation of constants formula, treating $s'(t)$ as inhomogeneity.

It is also interesting to note that in case of $\gamma(t) = const.$ the SEIR model allows for an invariant: Summation of equations (8)-(10) results in

$$s'(t) + e'(t) + i'(t) + \alpha i(t) = 0. \quad (19)$$

Now, replacing $i(t)$ by means of equation (8) and integrating yields

$$s(t) + e(t) + i(t) - \frac{\alpha}{\gamma} \ln s(t) = const. = 1 \quad \forall t \quad (20)$$

where the constant is given by means of the initial conditions.

Initial stage of epidemics

In reality epidemics start at a finite time with an integer number of infected individuals. In the homogenized integral equation model this must be substituted by an infinitesimal small initial growth starting at " $t = -\infty$ ". Thus, in order to investigate the initial growth of the epidemics, we linearize the integral equation (4) at $s(t) = s(-\infty) = 1$ and assume a constant γ . We get

$$s'(t) = \gamma \int_{-\infty}^{\infty} \Theta(t-t') s'(t') dt'. \quad (21)$$

Inserting the ansatz $s(t) = 1 - s_0 \exp(\lambda t)$ yields the characteristic equation

$$P_{iS}(\lambda) := \lambda \left(\gamma \int_0^{\infty} \Theta(\tau) \exp(-\lambda \tau) d\tau - 1 \right) = 0, \quad (22)$$

being defined where the integral exists. Its non trivial solutions ($\lambda \neq 0$) are given by the roots of the second factor. Using $\lambda = \eta + i\omega$ its real part becomes

$$\gamma \int_0^{\infty} \Theta(\tau) \cos(\omega \tau) \exp(-\eta \tau) d\tau - 1 = 0. \quad (23)$$

For all $\tau > 0$, the weight $\exp(-\eta \tau)$ is strictly decreasing with respect to η and maps $\eta \in [0, \infty[$ onto $]0, 1]$. Therefore, in case $\omega = 0$ we conclude that there exists a positive real eigenvalue $\lambda = \eta_0 > 0$ if and only if

$$\gamma \int_0^{\infty} \Theta(\tau) d\tau > 1. \quad (24)$$

which is at the same time necessary for the growth of the epidemic. We also deduce that $\eta_0 > 0$ is the only positive real eigenvalue. In case $\omega \neq 0$ we conclude that $\eta < \eta_0$ since $\cos(\omega\tau) < 1$ except for isolated points. Thus, as $t \rightarrow -\infty$, the associated solutions are decaying more slowly than the one belonging to η_0 . Therefore, the presence of such oscillating solution components would imply $s(t) < 0$ for some t sufficiently close to $-\infty$ which is ruled out by equation (6). Finally, this shows that the initial growth is given by the unique (up to s_0) solution

$$s_{initial}(t) = 1 - s_0 \exp(\eta_0 t). \quad (25)$$

Applying equations (18) and (22) we obtain the characteristic equation of the SEIR model

$$P_{SEIR}(\lambda) = \lambda \left(\frac{\gamma\sigma}{(\alpha + \lambda)(\sigma + \lambda)} - 1 \right) = 0 \quad (26)$$

with the solution

$$\eta_0 = \left(\sqrt{(\sigma + \alpha)^2 + 4\sigma(\gamma - \alpha)} - (\sigma + \alpha) \right) / 2, \quad (27)$$

which is real, since $(\sigma + \alpha)^2 + 4\sigma(\gamma - \alpha) = (\sigma - \alpha)^2 + 4\sigma\gamma \geq 0$. Moreover, $\eta_0 > 0$ if and only if $\gamma > \alpha$, which is the condition that allows for the growth of the epidemic.

Application of equations (17) and (22) yields the characteristic equation for the dSEIR model

$$P_{dSEIR}(\lambda) = \gamma (\exp(-\tau_e \lambda) - \exp(-\tau_i \lambda)) - \lambda = 0. \quad (28)$$

We deduce from the discussion above, that there is a single real positive solution $\eta_0 > 0$ if and only if $\gamma(\tau_i - \tau_e) > 1$. In summary, we have seen that epidemics can arise and grow if and only if

$$\gamma > \alpha \quad (\text{SEIR model}) \quad (29)$$

$$\gamma > 1/(\tau_i - \tau_e) \quad (\text{dSEIR model}) \quad (30)$$

and the initial growth is then given by (25).

Final stage of epidemics

Assuming that the contact rate is constant for all $t > t_0$, we can expect that $s(t)$ tends to some limit value s_∞ , reflecting the final infestation. Reformulating the iS model, i. e. equation (4), we get:

$$\begin{aligned} \frac{s'(t)}{\gamma(t) s(t)} &= \int_{-\infty}^{\infty} \Theta(t - t') s'(t') dt' \\ &= \int_{-\infty}^{\infty} \Theta'(t - t') s(t') dt'. \end{aligned}$$

Integrating between t_0 and t_1 gives

$$\begin{aligned} \int_{t_0}^{t_1} \frac{s'(t)}{\gamma(t) s(t)} dt &= \int_{-\infty}^{\infty} \Theta(t_1 - t') s(t') dt' \\ &\quad - \int_{-\infty}^{\infty} \Theta(t_0 - t') s(t') dt' \\ &= \int_{-\infty}^{\infty} \Theta(\tau) s(t_1 - \tau) d\tau \\ &\quad - \int_{-\infty}^{\infty} \Theta(\tau) s(t_0 - \tau) d\tau. \end{aligned}$$

This can be used to predict the limit $s_\infty := s(t \rightarrow \infty)$ if we assume $s(t)$ to be known for $t < t_0$ and $\gamma = \text{const.}$ for $t > t_0$. Sending $t_1 \rightarrow \infty$ we conclude that

$$\begin{aligned} 0 &= \ln s_\infty - \ln s(t_0) - s_\infty \gamma \int_0^\infty \Theta(\tau) d\tau \\ &\quad + \gamma \int_0^\infty \Theta(\tau) s(t_0 - \tau) d\tau =: f(s_\infty). \end{aligned} \quad (31)$$

Since $f''(s) = -1/s^2 < 0$, $f(s \rightarrow 0^+) = -\infty$ and

$$\begin{aligned} f(s(t_0)) &= \gamma \int_0^\infty \Theta(\tau) (s(t_0 - \tau) - s_\infty) d\tau \\ &> \gamma \int_0^\infty \Theta(\tau) (s(t_0) - s_\infty) d\tau > 0 \end{aligned} \quad (32)$$

there exists one and only one solution s_∞ of (31) which, moreover, can be written in terms of the Lambert W-function. If we even assume γ to be constant for all times, we may deduce

$$\ln s_\infty = (s_\infty - 1) \gamma \int_0^\infty \Theta(\tau) d\tau \quad (33)$$

by sending $t_0 \rightarrow -\infty$ and using $s(t \rightarrow -\infty) = 1$. Since

$$\int_0^\infty \Theta_{SEIR}(\tau) d\tau = \frac{1}{\alpha} \quad (34)$$

$$\int_0^\infty \Theta_{dSEIR}(\tau) d\tau = \tau_i - \tau_e \quad (35)$$

the application of equations (31) or (33) to SEIR and dSEIR models is obvious.

In summary, while maintaining the present infection rate and knowing the previous course of the disease, equations (31) or (33) make it possible to predict the final infestation. Thereby, equation (31) clearly shows that even with the same future infection rate, different disease courses in the past usually lead to different final infestations.

Reproduction number R

As already mentioned above, the defining formula for the reproduction number R is

$$R(t') = \int_{-\infty}^{\infty} s(t) \gamma(t) \Theta(t - t') dt. \quad (36)$$

If $\gamma(t)s(t) := \gamma_s = \text{const.}$ this simplifies to

$$R_{dSEIR} = \gamma_s(\tau_i - \tau_e) = \text{const.} \quad (37)$$

$$R_{SEIR} = \gamma_s/\alpha = \text{const.} \quad (38)$$

However, if $\gamma(t)s(t) \neq \text{const.}$ these formulas provide an approximation only.

In real applications to ongoing epidemics, $\gamma(t)$ is usually unknown and definition (5) is therefore not applicable. However, making use of equation (4), it is possible to compute R in terms of $s(t)$ and $\Theta(\tau)$ alone by means of

$$R(t) = \int_0^\infty \frac{\Theta(\tau) s'(t + \tau)}{\int_0^\infty \Theta(\tau') s'(t + \tau - \tau') d\tau'} d\tau. \quad (39)$$

This formula clearly shows that for a given course of the epidemic, the calculated reproduction figures depend on the chosen integral kernel, i. e. on its projectivation only, since multiplication with a non zero constant doesn't affect the result.

Comparison of SEIR and dSEIR models

The SEIR and dSEIR models are represented by families of integral kernels, which are described by two parameters each (α, σ and τ_i, τ_e , resp.). Also the transfer rate $\gamma(t)$ can be freely selected. However, no matter how the parameters are chosen, the kernels are different. This means that the parameters cannot be adjusted such that the two models will produce the same results in general. Agreement can only be reached on some aspects.

In the sequel we present **two methods** of comparison:

- (1) **Method (1)** is to adjust the parameters of the SEIR and the dSEIR model such that the (infinitesimal) initial growth coincides with respect to the exponential growth rate η_0 and all ratios $s(t) : e(t) : i(t)$. (Due to equation (7), $r(t)$ can be ignored.) However, this will lead to markedly different results at later times t including a different final infestation.
- (2) **Method (2)** (alt. appr.) is to demand equality of η_0 , the ratios $s(t) : i(t)$ and the final infestation s_∞ .

Adjusting the solutions

Here we describe in more detail how the two models are adjusted in case of **method (1)** and **method (2)**, respectively.

In both cases, SEIR and dSEIR, we extend the initial solution (25) to all variables s, e, i, r and write

$$u(t) = u_{-\infty} + u_0 \exp(\eta_0 t), \quad (40)$$

using the definitions

$$u(t) := \begin{pmatrix} s(t) \\ e(t) \\ i(t) \\ r(t) \end{pmatrix} \quad u_{-\infty} := \begin{pmatrix} 1 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad u_0 := \begin{pmatrix} -(1-s_0) \\ e_0 \\ i_0 \\ 1-s_0-e_0-i_0 \end{pmatrix}. \quad (41)$$

Thereby we have to assume $e_0 > 0$, $i_0 > 0$ and $1 - s_0 - e_0 - i_0 = r_0 > 0$ (see conditions (6) and (7)).

Now, regarding the dSEIR model, we use equations (4),(7), (12-15) and (16) to express the initial solution, i. e. u_0 , in terms of the model parameters (γ, τ_e, τ_i) and some amplitude $s_0 \leq 1$. (Choosing s_0 is equivalent to a shifting of time). We get

$$u_0 = (1-s_0) \begin{pmatrix} -1 \\ 1 - \exp(-\eta_0 \tau_e) \\ \exp(-\eta_0 \tau_e) - \exp(-\eta_0 \tau_i) \\ \exp(-\eta_0 \tau_i) \end{pmatrix}. \quad (42)$$

and define a mapping $U_0(\eta_0, \tau_e, \tau_i, s_0) := u_0$. We have already seen, that when solving the characteristic equation η_0 becomes a function of the parameters γ, τ_e, τ_i , i.e. $\eta_0 =: \Lambda_0(\gamma, \tau_e, \tau_i)$.

Next, we compute the parameters of the SEIR model in terms of u_0 and the growth rate η_0 , which, by definition, is an eigenvalue of the system matrix

$$A = \begin{pmatrix} 0 & 0 & -\gamma & 0 \\ 0 & -\sigma & \gamma & 0 \\ 0 & \sigma & -\alpha & 0 \\ 0 & 0 & \alpha & 0 \end{pmatrix} \quad (43)$$

with respect to the eigenvector u_0 . The eigenvalue equation

$$A u_0 = \eta_0 u_0 \quad (44)$$

may be reformulated as a system of linear equations for the unknown parameters (γ, σ, α) . Its unique solution ($e_0, i_0 > 0$) is:

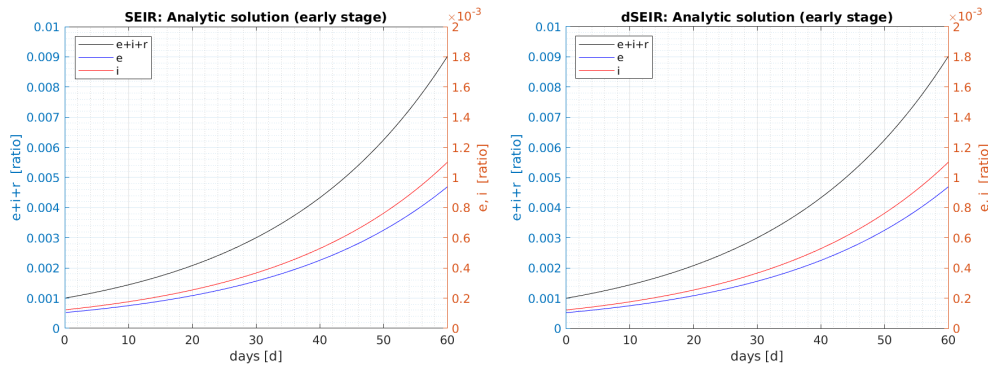
$$\gamma = \frac{1-s_0}{i_0} \eta_0 \quad (45)$$

$$\sigma = \frac{1-s_0-e_0}{e_0} \eta_0 \quad (46)$$

$$\alpha = \frac{1-s_0-e_0-i_0}{i_0} \eta_0 \quad (47)$$

Setting $\Pi(\eta_0, u_0) := (\gamma, \sigma, \alpha, s_0)$ defines a mapping from the growth rate and the state vector of the system at $t = 0$ to the parameters realizing that growth rate and state. Combining the two mappings defines a mapping of the parameters defining the dSEIR model to those defining the SEIR model, such that the initial epidemic developments of both models agree. It is given by means of the composition

$$(\gamma, \sigma, \alpha, s_0) = \Pi(\Lambda_0(\gamma, \tau_e, \tau_i), U_0(\Lambda_0(\gamma, \tau_e, \tau_i), \tau_e, \tau_i, s_0)). \quad (48)$$



Comparison of SEIR and dSEIR models

Fig. 2: Equal Analytic solutions for the SEIR and dSEIR models (method (1), initial stage).

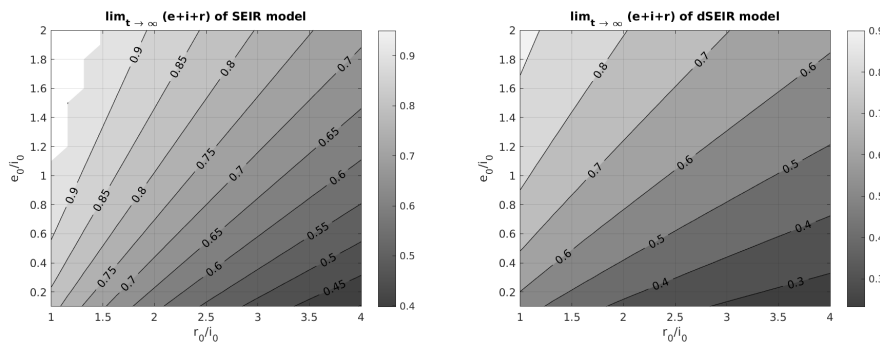
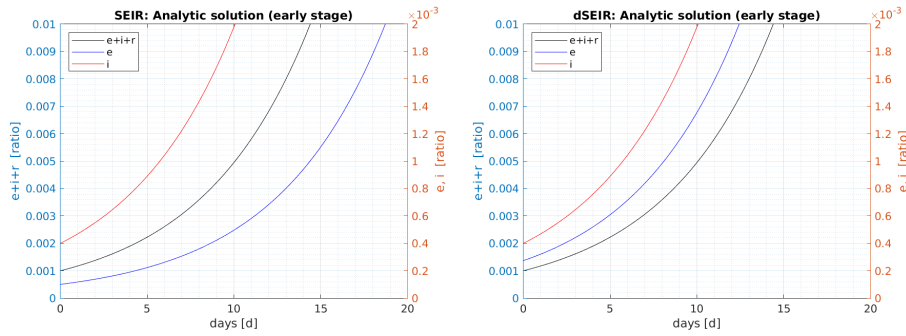


Fig. 3: Final infestation $e+i+r$ (SEIR and dSEIR, method (1)).

We remark, that the mapping leaves the parameter γ unchanged, since equations (8) and (12) agree. This already defines **method (1)** of comparison of the two models.

For **method (2)** we omit the equality of $e(t)$ (i. e. e_0) during the initial growth. Instead we demand the equality of the final infestations $s_\infty := \lim_{t \rightarrow \infty} s(t)$. This means that we set $\alpha^{-1} = \tau_i - \tau_e$ (see equations (33), (34) and (35)). However, since the procedure is very similar to that for method (1) we will not present the further details. At this place we want to note, that if we want γ to be a real quantity (i. e. the infectious rate, most likely), then it should be the same for both models. This immediately implies (see equations (8) and (12)) that $s(t)$ coincides if and only if $i(t)$ coincides. At the same time $r(t)$ cannot be freely chosen, due to condition (7). Thus, we can only give up the equality of $e(t)$, because, if we renounce the equality of one more variable, then we renounce the equality of all four variables.

An example where the two models are adjusted according to **method (1)** is demonstrated in figures (2) which totally coincide. However, as already mentioned, the disadvantage of this method is that it usually produces large differences in the final infestation. This is shown in figure (3) which is a level plot of the final infestation in dependence of the initial solution given by means of the ratios r_0/i_0 and e_0/i_0 (which are enough for it to be determined). An example where the two models are adjusted according to **method (2)** is demonstrated in figures (4). Now, only $i(t)$ and $s(t) = 1 - e(t) + i(t) + r(t)$ are the same, but $e(t)$ is different. However, this time s_∞ coincides by definition.



Comparison of SEIR and dSEIR models

Fig. 4: Analytic solutions for the SEIR and dSEIR models (method (2), initial stage).

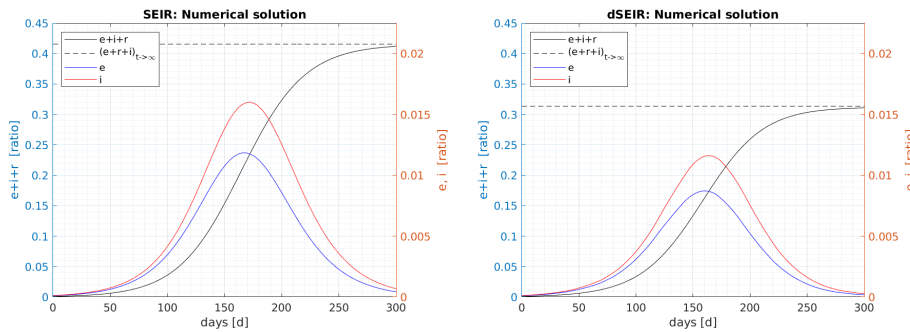


Fig. 5: Numeric solutions for SEIR and dSEIR models (method (1)).

Comparing numerical solutions

The more the epidemics develops, the more the nonlinearity in equations (8), (12) or (16) plays a role. Therefore, in order to determine the development during its entire duration, we use numerical integration routines of Matlab. Figures (5) and (6) show an example of comparing the models according to **method (1)** and **method (2)**, respectively. As we see the numerical solutions resemble the analytic solution very well during the initial stage. However, at later times the SEIR model shows a higher but slightly delayed peak of infestation. Moreover, **method (1)** yields different final infestations s_∞ in the SEIR and the dSEIR case.

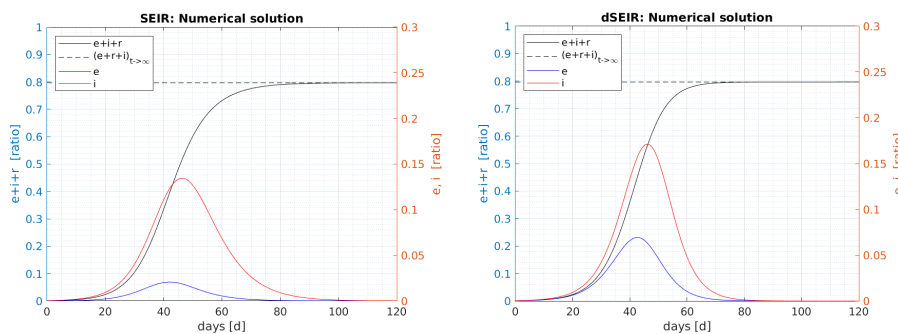
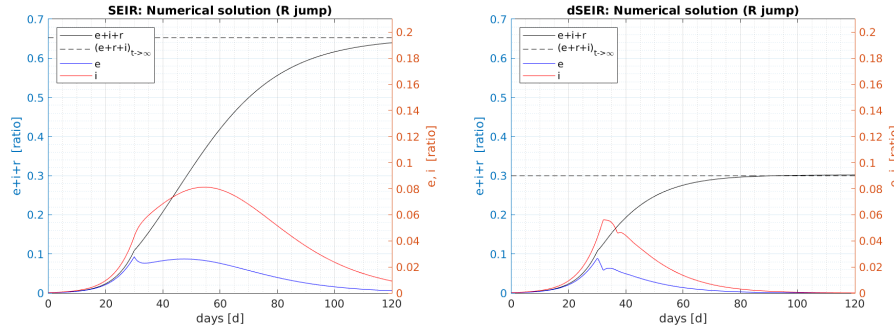


Fig. 6: Numeric solutions for SEIR and dSEIR models (method (2)).



Conclusion

Fig. 7: Numeric solutions with γ -jump: $\gamma = 0.4 \rightarrow 0.2$ (method (1)).

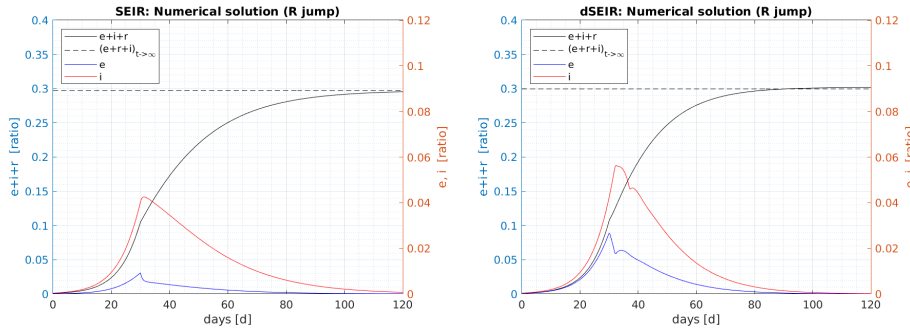


Fig. 8: Numeric solutions with γ -jump: $\gamma = 0.4 \rightarrow 0.2$ (method (2)).

An analogous comparison, however with a jump from $\gamma = 0.4$ down to $\gamma = 0.2$ after $t = 30$ days is shown in figures (7) and (8), respectively. The associated plot of the reproduction numbers $R(t)$ together with the respective integration kernels is shown in figures (9) and (10), respectively. Note the differences between the exact values according to formula (5) and the approximations according to formulas (38) which are shown in dotted lines.

Conclusion

We presented a general integral equation model which is used as a framework to compare epidemic models based on cohorts such as the SEIR and the dSEIR models. Moreover, the integral equation model allows for a uniform definition of the reproduction number.

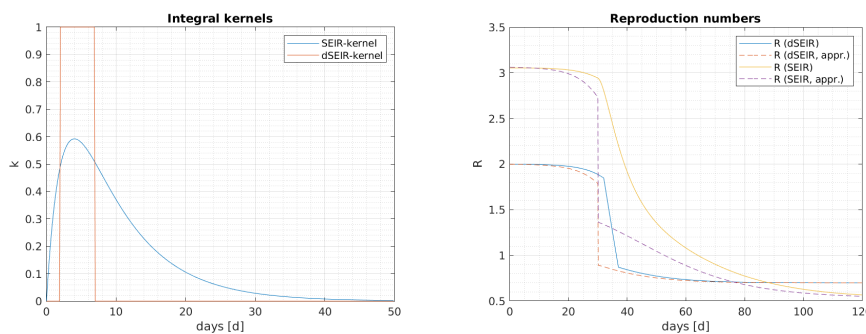
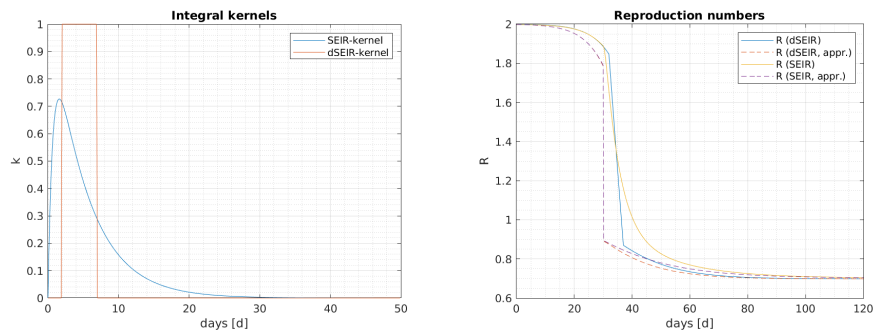


Fig. 9: Reproduction numbers with γ -jump: $\gamma = 0.4 \rightarrow 0.2$ (method (1)).



Conclusion

Fig. 10: Reproduction numbers with γ -jump: $\gamma = 0.4 \rightarrow 0.2$ (method (2)).

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A. Eigenvalues of the dSEIR model

We will deduce some information about the number and position of eigenvalues, i.e. the zeroes of f in the complex half plane $Re \lambda > 0$.

On any compact set $\Omega \subset \mathbf{R}^2$ which is diffeomorphic to the 2-disc with $\mathbf{f}|_{\Omega^{-1}(0)} \subset \text{int}(\Omega)$ and \mathbf{f} twice differentiable the following index formula holds

$$\sum_{\lambda \in \mathbf{f}|_{\Omega^{-1}(0)}} \text{ind}(\mathbf{f}, \lambda) = \text{deg}(\mathbf{f}|\Omega) = 1 + (\#\mathcal{I} - \#\mathcal{E})/2, \quad (49)$$

with \mathcal{I} and \mathcal{E} being the set of interior and exterior tangencies, respectively. Thereby, the set of tangencies is $\mathcal{T} \subset \partial\Omega$ consists of the zeroes of the (outer) normal component of \mathbf{f} and \mathcal{I} and \mathcal{E} are the subsets where the derivative of this component in the direction of \mathbf{f} is negative or positive, respectively. In case it is zero, we demand the zero to be a transversal one, which at the same time implies that \mathcal{I} and \mathcal{E} are finite sets. The formula then relates the numbers of interior and exterior tangencies on $\partial\Omega_r$ to the indices $\text{ind}(\mathbf{f}, \lambda)$ of the zeroes of \mathbf{f} in Ω_r , counted with their multiplicity each. Since f is holomorphic, the latter are positive numbers, i.e. $\text{ind}(\mathbf{f}, \lambda) \in \mathbf{N} = \{1, 2, \dots\}$. Moreover, in the generic case, the zeroes are of first order which implies $\text{ind}(\mathbf{f}, \lambda) = 1$.

Rescaling the time, we may assume $\gamma = 1$ and the characteristic equation of the dSEIR model becomes

$$f(\lambda) = \lambda + \exp(-\tau_i \lambda) - \exp(-\tau_e \lambda) \quad (50)$$

which simplifies the notation.

In the following, we often separate the real and imaginary parts, using $\lambda = \eta + i\omega$ and $f(\lambda) = u(\eta, \omega) + i v(\eta, \omega)$. Accordingly, f can be considered as a function $\mathbf{f} = (u, v) : \mathbf{R}^2 \rightarrow \mathbf{R}^2$, $(\eta, \omega) \mapsto (u(\eta, \omega), v(\eta, \omega))$ on 2-space. Let $\Omega_r = \{(\eta, \omega) \in \mathbf{R} \mid \eta > 0, \eta^2 + \omega^2 \leq r^2\}$ with the two corners being suitably smoothed (later on it will show up what this means).

Choosing $r > 2$ implies $|\exp(-\tau_i \lambda) - \exp(-\tau_e \lambda)| \leq 2$ on Ω_r . Therefore, f obviously points outwards everywhere on the half circle $\partial\Omega_r \setminus (\{0\} \times \mathbf{R})$ and thus has no tangencies or zeroes there. Thus, they can only occur on $\partial\Omega_r \cap (\{0\} \times \mathbf{R}) = \{0\} \times [-r, r]$ as a subset of the zero set \mathcal{Z} of $u(0, \cdot)$ on the imaginary axis. Since on the imaginary axis $\mathbf{f} = (u, v)$ (s. above) becomes

$$\begin{aligned} u(0, \omega) &= \cos(\tau_i \omega) - \cos(\tau_e \omega) \\ &= -2 \sin\left(\frac{\tau_i + \tau_e}{2} \omega\right) \sin\left(\frac{\tau_i - \tau_e}{2} \omega\right) \end{aligned} \quad (51)$$

$$v(0, \omega) = \omega - \sin(\tau_i \omega) + \sin(\tau_e \omega) \quad (52)$$

the zero set \mathcal{Z} of $u(0, \cdot)$ is discrete and given by

$$\mathcal{Z} = \frac{2\pi}{\tau_i + \tau_e} \mathbf{Z} \cup \frac{2\pi}{\tau_i - \tau_e} \mathbf{Z}. \quad (53)$$

The index formula only applies if \mathbf{f} has no zeroes on $\partial\Omega_r$, which means that $v(0, \cdot)$ must not have zeroes on $\mathcal{Z} \cap [-r, r]$. However, we have $v(0, 0) = u(0, 0) = 0$.

Generically this is true except for the zero at $(0, 0)$. Otherwise, this can be overcome by means of arbitrarily small changes of τ_i or τ_e .

Generically, all zeroes are of first order, however there also might occur double zeroes. According to our discussion above, they will not contribute to \mathcal{I} or \mathcal{E} and can be ignored. Thus, $\mathcal{I} \cup \mathcal{E} = \{0\} \times \mathcal{Z}_r^1$ with \mathcal{Z}_r^1 denoting the subset of first order zeroes in $\mathcal{Z} \cap [-r, r]$. W. l. o. g. we may use a numbering

$$\mathcal{Z}_r^1 = \{\omega_k \mid k = -K, -K + 1, \dots, K - 1, K\} \quad (54)$$

with $\omega_{k-1} < \omega_k$ and $\omega_0 = 0$ and observe that At this point we can already calculate R in case $\omega_{-k} = \omega_k$. Moreover, we deduce that the zero crossings are alternating, more precisely

$$\text{sign}(\partial_\omega u(0, \omega_k)) = (-1)^{k-1} \text{sign}(\omega_k). \quad (55)$$

Now we use that

$$\begin{aligned} \omega_k \in \mathcal{I} &\Leftrightarrow \text{sign}(\partial_\omega u(0, \omega_k)) \text{sign}(v(0, \omega_k)) > 0 \\ \omega_k \in \mathcal{E} &\Leftrightarrow \text{sign}(\partial_\omega u(0, \omega_k)) \text{sign}(v(0, \omega_k)) < 0 \end{aligned} \quad (56)$$

according to the definition. Since

$$\partial_\omega u(0, \omega_k) = \begin{cases} -(\tau_i + \tau_e) \sin(\tau_i \omega_k) & \text{if } \omega_k \in \frac{2\pi}{\tau_i - \tau_e} \mathbf{Z} \\ -(\tau_i - \tau_e) \sin(\tau_i \omega_k) & \text{if } \omega_k \in \frac{2\pi}{\tau_i + \tau_e} \mathbf{Z} \end{cases} \quad (57)$$

$$v(0, \omega_k) = \begin{cases} \omega_k & \text{if } \omega_k \in \frac{2\pi}{\tau_i - \tau_e} \mathbf{Z} \\ \omega_k - 2 \sin(\tau_i \omega_k) & \text{if } \omega_k \in \frac{2\pi}{\tau_i + \tau_e} \mathbf{Z} \end{cases} \quad (58)$$

we observe that

$$\partial_\omega u(0, \omega_{-k}) = -\partial_\omega u(0, \omega_k) \quad (59)$$

$$v(0, \omega_{-k}) = -v(0, \omega_k) \quad (60)$$

$$\omega_k > 0, \text{sign}(\partial_\omega u(0, \omega_k)) = 1 \Rightarrow \text{sign}(v(0, \omega_k)) = 1 \quad (61)$$

whereas in case $\text{sign}(\partial_\omega u(0, \omega_k)) = -\text{sign}(\omega_k)$ the conclusion is more complicated. To this end we define

$$\mathcal{K}^- = \{k \mid 0 < k \leq K, \text{sign}(v(0, \omega_k)) = -1\} \quad (62)$$

$$\chi(k) = \begin{cases} 1 & k \in \mathcal{K}^- \\ 0 & \text{else} \end{cases} \quad (63)$$

Finally, we are ready to compute

$$\begin{aligned} \#\mathcal{I} - \#\mathcal{E} &= \sum_{0 \neq k = -K}^K \text{sign}(\partial_\omega u(0, \omega_k)) \text{sign}(v(0, \omega_k)) \\ &= 2 \sum_{k=1}^K \text{sign}(\partial_\omega u(0, \omega_k)) \text{sign}(v(0, \omega_k)) \\ &= 2 \sum_{k=1,3,\dots,K} \left(\text{sign}(\partial_\omega u(0, \omega_k)) \text{sign}(v(0, \omega_k)) \right. \\ &\quad \left. + \text{sign}(\partial_\omega u(0, \omega_{k+1})) \text{sign}(v(0, \omega_{k+1})) \right) \\ &= 2 \sum_{k=1,3,\dots,K} \left(1 \cdot 1 + (-1) \cdot (-1)^{\chi(k)} \right) \\ &= 4 \#\mathcal{K}^- \end{aligned} \quad (64)$$

The resulting index formula then serves for the computation of the number of eigenvalues in the positive half plane by means of:

$$\#(\mathbf{f}|\Omega_r)^{-1}(0) = \sum_{\lambda \in (\mathbf{f}|\Omega_r)^{-1}(0)} \text{ind}(\mathbf{f}, \lambda) = 1 + 2 \#\mathcal{K}^- \quad (66)$$

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