ANALYSIS AND COMPARISON OF DIFFERENT MODELLING APPROACHES BASED ON AN SIS EPIDEMIC

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ABSTRACT
An SIS epidemic is a common approach to model a contagious disease. Three different modelling approaches are used to simulate an SIS epidemic. These methods are an ordinary differential equation model, an agent-based model and a stochastic model. The aim is to compare them in equivalent settings and analyse the qualitative and quantitative similarities and differences. Furthermore, they are extended to simulate diseases based on two serotypes. For the models with one serotype the results for all three approaches are very similar. For two serotypes there are some situations in which the three models agree and some in which they do not agree. Our conclusion is that the models are not completely equivalent. However it is not possible to determine which model is the best because this always depends on the actual situation. The comparison of the three modelling approaches can help to get a better understanding of SIS epidemics.

Keywords: SIS epidemic, ODE model, agent-based model, stochastic model

1. INTRODUCTION

Models of epidemics have been of interest for humanity for a long period of time. They are important to study the mechanism by which diseases spread, predict the courses of epidemics and to evaluate plans to control them. Common questions are interventions like vaccination strategies and isolation plans.
There are different types of epidemics and different concepts to model them. One of the first epidemic models was created by Kermack and McKendrick in the 1920s (Kermack and McKendrick 1927). They used ordinary differential equations (ODEs) to model SIS epidemics, the most simplified approach for contagious diseases. Today this serves as a classical model and is still used.
This concept can also be transferred to other modelling techniques. In this paper three approaches are considered: an agent-based model (AB model), an ODE model and a stochastic model (STOCH model). The purpose is to find out if these approaches are appropriate for epidemic simulation and to get a deeper understanding of the spread of epidemics. Since the basic characteristics of these techniques are different, it is important to have a closer look if the models are really equivalent and to compare the models in various scenarios.
The goal is to compare the three different approaches to model a simplified SIS epidemic with one and two serotypes in various scenarios and to analyse the differences if they exist.

2. SIMPLE SIS MODEL

An SIS model is a simplified concept of contagious diseases that describes the spread of an infectious disease within a constant and homogeneous population (denoted by N) of individuals. This means that there are no births, deaths and migration and all individuals are of the same type. The state of every person is either infected (denoted by I) or susceptible (denoted by S).
Since the population is constant and there are only two states Equation (1) holds at every point in time t:

$$S(t) + I(t) = N \tag{1}$$

The susceptible individuals can be infected through contact with an infected individual and stay infected for a specified constant period of time. After this time, they change their state from infected to susceptible and they can potentially be infected again (see Figure 1).

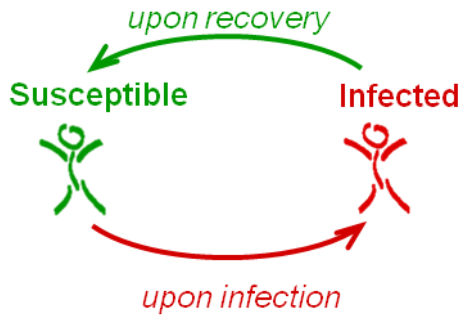

Figure 1: Illustration of the States and Transitions of the Simple SIS Model

There is no immunity or any other disease states. The transmission of infection is direct. There is no period of latency, this means the time period between the transmission of infection and the initiation of the infectiosity does not exist.

2.1. Ordinary Differential Equation Model

The ODE model splits the population in two parts, the infected and the susceptible part; these are the two state variables of the model. The infected part is denoted by I, the susceptible part by S. The size of the states varies in time, because susceptible can become infected and vice versa. Figure 2 shows the two states S and I and the transitions between them. The transition rate from S to I is α, from I to S it is described by β.

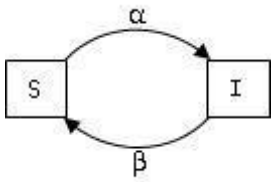

Figure 2: Illustration of the States and Transitions of the ODE Model

The parameters of the ODE model are α and β. α is the average number of transmissions per person per time unit caused by contacts between susceptible and infected individuals. α also depends on the infection probability. β is the ratio of recovery.
The ODE model consists of two ODEs (Equation (2) and (3)) with initial conditions (4) and (5) which describe the shift of the susceptible and the infected part of the population. The ODE model is similar to the model of McKendrick and Kermack.

$$\dot{I}(t) = \alpha I(t)S(t) - \beta I(t) \quad (2)$$

$$\dot{S}(t) = -\alpha I(t)S(t) + \beta I(t) \quad (3)$$

$$I(0) = I_0 > 0 \quad (4)$$

$$S(0) = N - I_0 \quad (5)$$

Equation (2) describes the change of the number of the infected part in time. The first term on the right hand side of Equation (2) represents the newly infected and the second term the recovered. Equation (3) describes the change of the number of the susceptible part in time. Since the population is constant the right hand side of Equation (3) varies only in the sign from the right hand side of Equation (2).
The two ODEs are analytically solvable and the solution of the ODE system is unique.

2.2. Agent-Based Model

The AB model describes the behaviour of the single individuals. Therefore it is an intuitive approach. An individual can either be infected or susceptible. All agents are identical except for their states. Individuals have contacts with other individuals and these contacts are created randomly within the population. Susceptible individuals can be infected when they meet infected individuals. This process represents the contagion. For one susceptible agent the contacts and the contagion are illustrated in Figure 3.

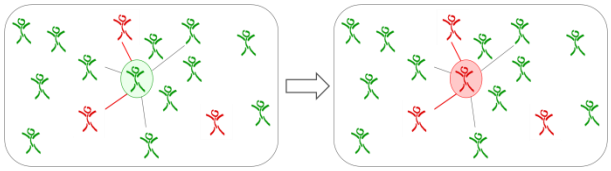

Figure 3: Illustration of the Contagion of a Susceptible Agent

After a fixed period of time an infected agent becomes susceptible again (see Figure 4).

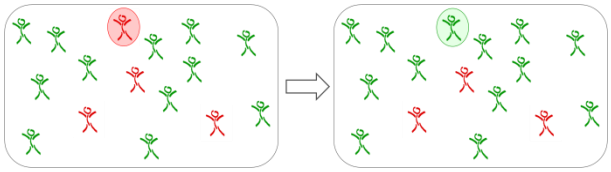

Figure 4: Illustration of the Recovery of an Infected Agent

The spread of the disease occurs upon the above-mentioned rules and is not defined explicitly.
The parameters of the model are the probability of infection ω, the number of contacts per agent κ and the duration of disease γ.
For the comparison with the other models, the numbers of infected and susceptible individuals are aggregated.

2.3. Stochastic Model

The STOCH model treats the two states susceptible and infected. The model consists of two equations, one for the number of infected individuals and one for the number of susceptible individuals.
The parameters of the model are identical to the parameters of the AB model, namely the probability of infection ω, the number of contacts per individual κ and the duration of disease γ.
The number of infected individuals for the present time step is determined dependent on the number of infected and susceptible individuals of the previous time steps (Equation (6)). In Equation (6) the newly infected individuals of time step t are represented by ni(t) and r(t) is the number of the recovered at time step t. The number of the susceptible individuals results from the difference of the size of the population and the number of infected individuals (Equation (7)).

$$I(t) = ni(t-1) + I(t-1) - r(t-1) \quad (6)$$

$$S(t) = N - I(t) \quad (7)$$

The part of the population which changes the state is calculated in discrete time steps.

2.4. Comparison of the Models

The three models use different parameters (see Table 1).

Table 1: Parameters of the Models

ODE Model	AB Model and STOCH Model
α	ω
β	γ
	κ

In order to compare the models in equivalent settings it is important to know the relations between the parameters.

$$\alpha = \frac{\kappa \cdot \omega}{N} \tag{8}$$

$$\beta = \frac{1}{\gamma} \tag{9}$$

The Equations (8) and (9) show the relation of the parameters and therefore the models became comparable by parameters that represent the same situation.

These relations have been found by the following considerations: In the STOCH model the number of contacts between I and S individuals with transmission of infection per time unit is $\frac{\kappa \cdot I \cdot S \cdot \omega}{N}$. To obtain the parameter α from the ODE model the last expression has to be divided by $I \cdot S$ (see Equation (8)). The duration of the disease is the reciprocal of the ratio of recovery (see Equation (9)).

In general, the results of the models show that in each model a steady state is reached after an adaption phase in the beginning of the simulation.

The steady states of the three models are independent of the initial value of the number of the infected individuals and only depend on parameters that represent infection probability, recovery time and contact number.

The AB model and the STOCH model have similar adaption phases, while the adaption phase of the ODE model is different. The ODE model and the STOCH model reach the same steady states and the steady states of the AB model are slightly different.

During the adaption phase of the AB model and the STOCH model a so-called "overshooting" occurs (see Figure 6 and 7). The ODE model has no "memory" and therefore there exists no "overshooting" (see Figure 5). That means the number of the infected depend only on the number of the infected of the previous time step. The AB model and the STOCH model use numbers from earlier time steps too.

For the results shown in Figure 5, 6 and 7 the size of the population N is 10000 and the initial value of the number of infected individuals I_0 is 1000.

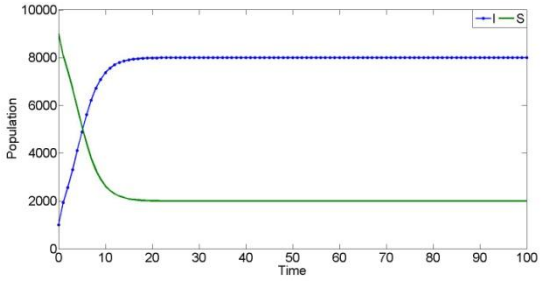

Figure 5: ODE Model with Parameters: $\alpha = 5 \cdot 10^{-5}$; $\beta = 0.1$

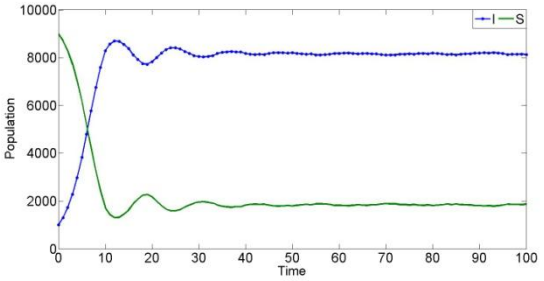

Figure 6: AB Model with Parameters: $\omega = 0.05$; $\gamma = 10$; $\kappa = 10$

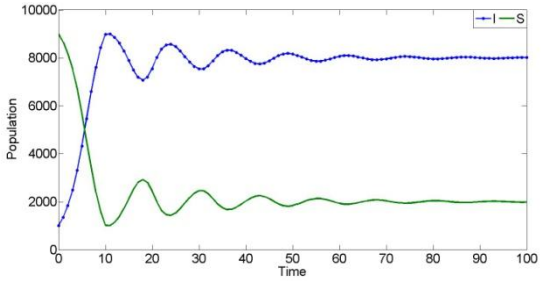

Figure 7: STOCH Model with Parameters: $\omega = 0.05$; $\gamma = 10$; $\kappa = 10$

The quantitative discrepancy between the results of the models is small and therefore the three models are appropriate methods to simulate an SIS epidemic.

3. SIS MODEL WITH TWO SEROTYPES

In contrast to the simple SIS model there are three states: individuals can either be susceptible or infected with serotype 1 or infected with serotype 2. A serotype is a variation of the pathogen of a contagious disease. The assumption for the simulated diseases is that an infected person can only be infected by one serotype at one time. An infected individual can become infected with the other serotype and then it loses the original serotype (see Figure 8). This property of the model is necessary, because models become instable when infected individuals are not susceptible for the other serotype (Urach 2009; Bauer, Pöll, and Winterer 2011) and therefore the model is unusable.

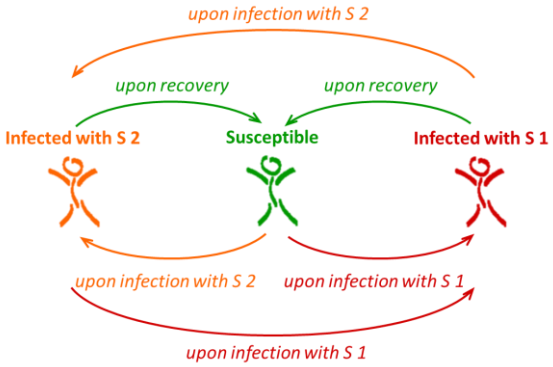

Figure 8: Illustration of the States and Transitions of the SIS Model with Two Serotypes

Further assumptions are the same as for the simple SIS model.

3.1. Ordinary Differential Equation Model

The ODE model splits the population in three state variables: I (infected with serotype 1), J (infected with serotype 2) and S (susceptible). Figure 9 shows the three states S, I and J and the transitions between them.

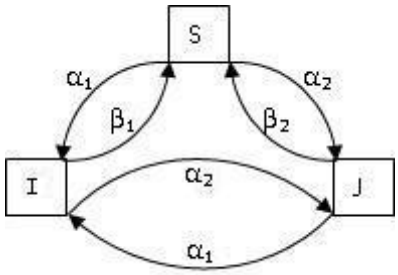

Figure 9: Illustration of the States and Transitions of the ODE Model with Two Serotypes

The parameters of the ODE model are α_1, α_2, β_1 and β_2. α_1 and α_2 are the average numbers of transmissions per person per time unit caused by contacts between susceptible and infected individuals. α_1 and α_2 also depend on the infection probability of each serotype. β_1 and β_2 are the ratios of recovery of serotype 1 and 2.
The ODE model consists of three ODEs (Equation (10), (11) and (12)) with initial conditions (13), (14) and (15). Every ODE represents the change of the number of individuals of one state. The ODEs are more complex than the ODEs of the simple SIS model.

$$\dot{I}(t) = \alpha_1 I(t)S(t) - \beta_1 I(t) - \alpha_2 I(t)J(t) + \alpha_1 I(t)J(t) \quad (10)$$

$$\dot{J}(t) = \alpha_2 J(t)S(t) - \beta_2 J(t) + \alpha_2 I(t)J(t) - \alpha_1 I(t)J(t) \quad (11)$$

$$\dot{S}(t) = -\alpha_1 I(t)S(t) - \alpha_2 J(t)S(t) + \beta_1 I(t) + \beta_2 J(t) \quad (12)$$

$$I(0) = I_0 > 0 \quad (13)$$

$$J(0) = J_0 > 0 \quad (14)$$

$$S(0) = N - I_0 - J_0 \quad (15)$$

The ODE system cannot be solved analytically, but at least the solution of the ODE system is unique.

3.2. Agent-Based Model

The AB model is an extension of the AB model for one serotype. The rules for the agents are very similar. It is easy to extend the AB model, because in the model only the phase of the transmission of infection has to be adjusted, because now an infected can be infected with the other serotype.
The parameters of the model are the probabilities of infection with serotype 1 ω_1 and serotype 2 ω_2, the number of contacts per agent κ and the duration of the diseases γ_1 and γ_2.

3.3. Stochastic Model

The STOCH model consists of three equations, one for the number of individuals infected with serotype 1, one for the number of individuals infected with serotype 2 and one for susceptible.
The parameters of the model are identical to the parameters of the AB model, namely the probabilities of infection with serotype 1 ω_1 and serotype 2 ω_2, the number of contacts per individual κ and the duration of the diseases γ_1 and γ_2.
The number of infected individuals of each serotype for the present time step is determined dependent on the number of infected and susceptible individuals of the previous time steps (Equation (16) and (17)). In Equation (16) and (17) the newly infected of time step t are represented by $ni_1(t)$ and $ni_2(t)$. $r_1(t)$ and $r_2(t)$ are the numbers of the recovered at time step t. c_1 (respectively c_2) is the part of the infected individuals with serotype 1 (respectively 2) which becomes infected by serotype 2 (respectively 1). The number of the susceptible individuals results from the difference of the size of the population and the number of infected individuals (Equation (18)).

$$I(t) = ni_1(t-1) + I(t-1) - r_1(t-1) - c_1(t-1) \quad (16)$$

$$J(t) = ni_2(t-1) + J(t-1) - r_2(t-1) - c_2(t-1) \quad (17)$$

$$S(t) = N - I(t) - J(t) \quad (18)$$

The equations are more complex than the equation in the STOCH model for one serotype.
The numbers of I, J and S are calculated in discrete time steps.

3.4. Comparison of the Models

The three models use different parameters (see Table 2).

Table 2: Parameters of the Models

ODE Model	AB Model and STOCH Model
α_1	ω_1
α_2	ω_2
β_1	γ_1
β_2	γ_2
	κ

The relations of the parameters of the three models are like the relations of the parameters of the simple SIS model (see Equation (19) and (20)).

$$\alpha_1 = \frac{\kappa \cdot \omega_1}{N}, \alpha_2 = \frac{\kappa \cdot \omega_2}{N} \quad (19)$$

$$\beta_1 = \frac{1}{\gamma_1}, \beta_2 = \frac{1}{\gamma_2} \quad (20)$$

This strategy enables further comparability of the three models.

There are two different cases for the parameterisation of the three models. On the one hand, the parameters for the two serotypes are equal (equally strong serotypes), on the other hand, there is a difference in at least one parameter of the serotypes (unequally strong serotypes). The models always reach a steady state. The steady states of the AB model and the STOCH model are independent of the initial settings. In the ODE model in case of equally strong serotypes the steady states are dependent on the initial settings, for unequally strong serotypes they are independent.

The main difference between the models is that coexistence of both serotypes is more often found for the AB model (see Figure 11). The STOCH model and the ODE model have almost no situations of coexistence for unequally strong serotypes. This means that the weaker serotype usually dies out (see Figure 10 and 12).

For the results shown in Figure 10, 11 and 12 the size of the population N is 10000 and the initial value of the number of infected individuals with serotype 1 I_0 and serotype 2 J_0 is both 1000.

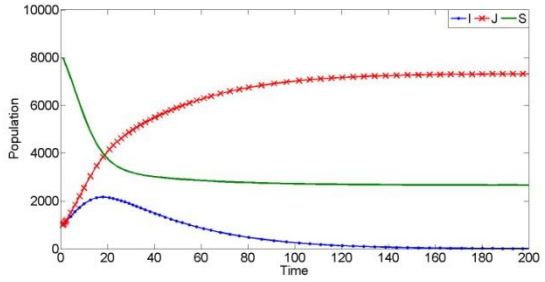

Figure 10: ODE Model with Two Serotypes with Parameters: $\alpha_1 = \alpha_2 = 2.5 \cdot 10^{-5}$; $\beta_1 = 0.1$; $\beta_2 = 1/15$

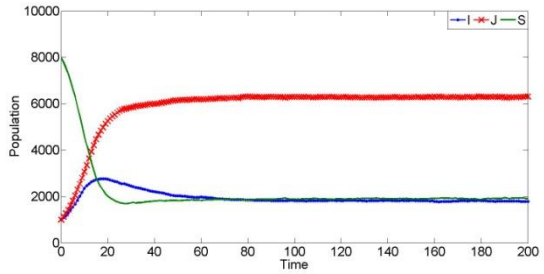

Figure 11: AB Model with Two Serotypes with Parameters: $\omega_1 = \omega_2 = 0.05$; $\gamma_1 = 10$; $\gamma_2 = 15$; $\kappa = 5$

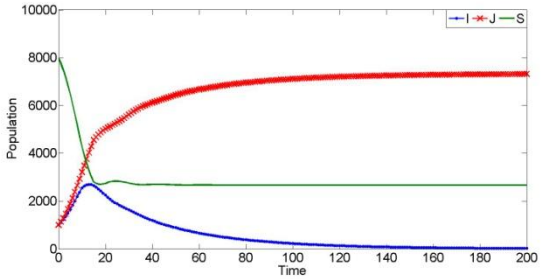

Figure 12: STOCH Model with Two Serotypes with Parameters: $\omega_1 = \omega_2 = 0.05$; $\gamma_1 = 10$; $\gamma_2 = 15$; $\kappa = 5$

In case of two equally strong serotypes, the steady states are different for all three models. In the cases where one serotype dies out in all three models, the steady states of the STOCH model and the ODE model are the same and the steady state of the AB model is very similar, while the adaption phase is similar for the STOCH model and the AB model and different for the ODE model. These results are obvious, because after extinct of one serotype the models are equivalent to the simple SIS models. There are two problems: First, for the ODE model and the STOCH model there is coexistence of both serotypes only in special cases. Second, the steady states of the models are quantitatively different. These problems show that further research for concurrent serotypes is required.

4. CONCLUSIONS

There are many possibilities to model a real problem. In this paper, three modelling approaches with different properties for an SIS epidemic have been compared in equivalent settings and there are some situations in which the three models agree and some in which they do not agree. In the latter case, a special validation of the results is needed to see which model gives the most realistic results. It is not possible to say only by comparison which modelling approach is the best, however it clearly shows qualitative and quantitative differences and similarities of these approaches. It always depends on the given situation which approach is right for the problem. In the cases where all three models agree, the models are considered to be valid.

Cross-validation is an important method in the field of modelling (Bharathy and Silverman 2010). It describes the attempt to solve the question with an alternative model and compare it with the actual model. If the

alternative model provides the same results the actual model might be considered to be more reliable, and consequently more valid.

In this study we show that the three modelling approaches all result in the same steady states for the simple SIS model, even though they have different adaption phases. The adaption phases can be explained by structural differences of the approaches. However, it seems that all three models are suitable to model SIS epidemics.

The situation is more complex for two competitive serotypes. In that case only the agent based model produces a stable coexistence of both serotypes while the ODE model and the STOCH model lead to an unstable behaviour. This means that the models simulate different situations caused by different structures, although their assumptions are all the same. We conclude that the choice of a model for SIS epidemics with competitive serotypes highly depends on the actual situation. It is important to examine structures and values in the real world crucially, compare them with the model and choose the right approach upon this knowledge. However, only the AB model is able to produce stable coexistence of serotypes. Usage of this model will be subject to further validation methods depending on the actual problem. This work might include further examination and revision of the ODE and STOCH model to find out more about consequences of structural differences.

Finally, the comparison of the modelling approaches can help to show in which situations models are valid and to get a better understanding of SIS epidemics.

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