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CONTENT

The Appendix

In honor of the first Doctor of Mathematical Sciences Acad. Blagoj Popov, a mathematician dedicated to differential equations, the idea of holding the "Day of Differential Equations" was born, prompted by Prof. Ph.D. Boro Piperevski, Prof. Ph.D. Borko Ilievski, and Prof. Ph.D. Lazo Dimov. Acad. Blagoj Popov presented his doctoral dissertation on 05.05.1952 in the field of differential equations. This is the main reason for holding the " Day of Differential Equations" at the beginning of May.

This year on May 7th, the "Day of Differential Equations" was held for the sixth time under the auspices of the Faculty of Computer Sciences at "Goce Delcev" University in Stip and Dean Prof. Ph.D. Cveta Martinovska - Bande, organized by Prof. Ph.D. Biljana Zlatanovska. The event was organized online via the platform Microsoft Teams and with the selfless help and support of Prof. Ph.D. Natasa Stojkovik, Ass. Prof. Ph.D. Limonka Koceva Lazarova, Ass. Prof. Ph.D. Marija Miteva, Ass. Prof. Ph.D. Mirjana Kocaleva, Ass. Prof. Ph.D. Aleksandra Stojanova.

The participants of this event were:

1. Prof. Ph.D. Boro Piperevski, Prof. Ph.D. Sanja Atanasova and Stefan Boshkovski (student) from the Faculty of Electrical Engineering and Information Technology at Ss. Cyril and Methodius, University in Skopje;
2. Prof. Ph.D. Aleksa Malcheski from the Faculty of Mechanical engineering at Ss. Cyril and Methodius, University in Skopje;
3. Prof. Ph.D. Slagjana Brsakoska from the Faculty of Natural Sciences and Mathematics at Ss. Cyril and Methodius, University in Skopje;
4. Prof. Ph.D. Natasa Stojkovik, Prof. Ph.D. Martin Lukarevski, Ass. Prof. Ph.D. Limonka Koceva Lazarova, Ass. Prof. Ph.D. Marija Miteva, Ass. Prof. Ph.D. Mirjana Kocaleva, Ass. Prof. Ph.D. Aleksandra Stojanova, Ass. Prof. Ph.D. Jasmina Buralieva Veta, Ass. Prof. Ph.D. Elena Karamazova, Prof. Ph.D. Biljana Zlatanovska from the Faculty of Computer Sciences at "Goce Delcev" University in Stip.

Acknowledgments to Prof. Ph.D. Boro Piperevski, Prof. Ph.D. Borko Ilievski and Prof. Ph.D. Lazo Dimov for the wonderful idea and the successful realization of the event this year and in previous years.

Acknowledgments to the Dean of the Faculty of Computer Sciences, Prof. Ph.D. Cveta Martinovska - Bande for her overall support of the organization and implementation of the "Day of Differential Equations".

The papers that emerged from the "Day of Differential Equations" are in the appendix to this issue of BJAMI.

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APPLICATION OF DIFFERENTIAL EQUATIONS IN EPIDEMIOLOGICAL MODELS

LIMONKA KOCEVA LAZAROVA, NATASA STOJKOVIKJ, ALEKSANDRA STOJANOVA, MARIJA MITEVA

Abstract. Mathematical modelling is a tool for presenting objects and processes with mathematical language and mathematical rules. Mathematical models can be used for research in public health and epidemiology. Nowadays, during the COVID19 outbreak, mathematical modelling is playing a central role in controlling the spread of infection, making predictions that can help monitoring the epidemic and making adequate responses, in order to lower the number of new infections. In this paper, several models commonly used to control the spreading of infections are considered. These models can also help controlling the COVID 19 infections.

1. Introduction

Mathematical modelling describes processes and objects by using mathematical language, and computer simulation, on the other hand, presents a natural continuation of mathematical modelling. Computer simulation can be considered a kind of a computer experiment which corresponds to an experiment in the real world. When a mathematical model is made, mathematical analysis, combined with computer simulations, is used to investigate the global behavior of the model, thus obtaining the consequences of the assumptions [1-2].

Mathematical models can help to synthesize information from different sources into one consistent framework that allows an integrated analysis of complex problems. Mathematical models, also, can be used as a useful tool in research in the field of public health, especially epidemiology. These models in the field of public health can be used to simulate the impact of different interventions or strategies, and to provide quantitative predictions of how interventions can affect the population's health in the future, [2-3].

The idea that the transmission and spread of infectious diseases can follow laws that can be presented and formulated in a mathematical language is not new. Nowadays, living in the COVID 19 pandemic, finding some mathematical models for epidemiology uses is a key tool for guiding public health measures, [2]. The main idea in transmission epidemiology models is opposed to statistical models. Epidemiology models present a mechanistic description of infection transmission among individuals. This mechanistic description can describe the time evolution of an epidemic in mathematical terms, and, in this way, it can connect the individual level process of transmission with the population level of incidence and prevalence of an infectious disease. For obtaining a mathematical formulation of these dependencies it is necessary to analyze all dynamic processes that contribute to disease transmission.

Mathematical modelling, for this purpose, can be integrated with knowledge from different disciplines like social sciences, clinical sciences, or microbiology. [4-6]

In the last several decades, mathematical modelling used in public health and infectious disease control has been proven to be a standard practice and a useful tool in decision-making. During the outbreak of the influenza pandemic in 2009, mathematical modelling helped researchers to make decisions in order to obtain an adequate national response to control outbreak in the early phase of the pandemic. The mathematical model also helped to adopt vaccination strategies in the later phase of pandemic. These models were also used for estimating the key parameters for outbreak control during the Ebola Epidemic [6-9].

During the pandemic of Covid 19, mathematical modelling, according to previous experiences, can also be used and has been used for taking outbreak control, and taking appropriate measures such as lock down, case isolation, contact-tracing with quarantine, and sanitary funeral practices in order to lower the numbers of new infections. As vaccines have become available, mathematical models can be used for adopting vaccination strategies and thus monitor and predict the impact of vaccination on the epidemic [5-6, 10].

In this paper, we are considering the mathematical models that are commonly used for the spread of infectious diseases. These models are SI, SIS, SIR, SIRS, and SEIR model without vital dynamics. These models can be used for prediction, control, and treatment of infectious diseases.

2. Mathematical models

For all models that are considered in this paper, it is assumed that the initial population is N individuals. The susceptible group is formed from the total population that is at risk of a disease. Over time, out of this population some susceptible individuals will become infected. All infected people are forming the infectious group. The member of the infected group will contribute to onwards transmission.

The infected individuals may recover and acquire life immunity or transient immunity. the recovered people will form the recovered group. On the other side, some of the infected persons die. Those who die will form the death group, [11-14].

2.1. SI model – without vital dynamics

The simplest model for the spread of an infection is the SI model, which tracks the fraction of a population in each of two groups: susceptible and infected. The sizes of these groups are functions of time t. They we will be denoted with S(t) – susceptible group and I(t) – infected group.

In the SI model without vital dynamics, any change (births and deaths) in the population is ignored. Because of that, this model is also called "closed epidemic model" with a fixed size of population, N = S + I.
In Figure 1 the SI model is represented.

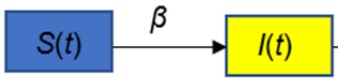


Figure 1. *SI Model*

From Figure 1 we can conclude that susceptible individuals become infected with rate β. A pair of ordinary differential equations describes this model:

$$\frac{dS}{dt} = -\frac{\beta SI}{N}, \quad \frac{dI}{dt} = \frac{\beta SI}{N} = \beta I\left(1 - \frac{I}{N}\right). \tag{2.1}$$

β is the transmission (infection) rate, i.e. the rate of the virus spread that represents the probability for disease transmission between a susceptible individual and an infected individual.

The expressions $dS/dt, dI/dt$ denote the change in the susceptible and infected groups during time, respectively.

Because the total population $N = I(t) + S(t) \Rightarrow S(t) = N - I(t)$, it follows that

$$\frac{dI}{dt} = \frac{\beta\left(N - I(t)\right)I(t)}{N}.$$

The differential equation is known as the logistic growth equation, proposed by Verhulst (1845) for population growth.

$$\frac{1}{\left(N - I(t)\right)I(t)}\frac{dI}{dt} = \frac{\beta}{N} \Rightarrow \quad \int_0^t \frac{1}{\left(N - I(t)\right)I(t)}\frac{dI}{dt}dt = \int_0^t \frac{\beta}{N}dt \ .$$

For the solution of the differential equation the logistic curve is obtained

$$I(t) = \frac{I(0)N}{I(0) + \left(N - I(0)\right)e^{-\frac{\beta}{N}t}}.$$

Figure 2 represents the number of suspectable and infected individuals. When $t \to \infty, I \to N$, everyone becomes infected.

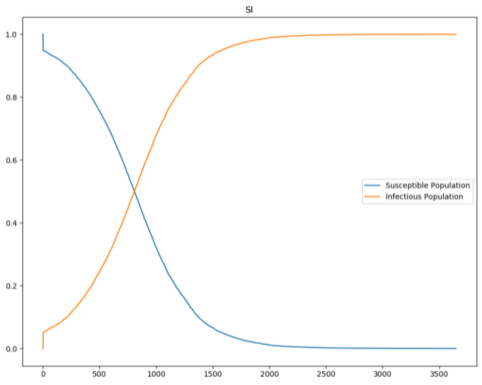


Figure 2. *Suspectable and infected individuals in the SI model*

2.2. SIS model

An infected individual can recover from infection and return unprotected to the suscectible group. After that, these individuals can be infected again. These cases can be modelled by using the SIS model that is presented in Figure 3.

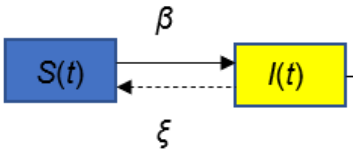


Figure 3. *SIS Model*

For a fixed population, where there are no births or deaths and persons recover from the disease with a recovery rate ξ, the simplest form of the model in Figure 3 is given with the following differential equations:

$$\frac{dS}{dt} = -\frac{\beta SI}{N} + \xi I, \quad \frac{dI}{dt} = \frac{\beta SI}{N} - \xi I \tag{2.2}$$

where $\xi = \frac{1}{\tau_{immunity}}$, and $\tau_{immunity}$ (immunization time) represents the duration of the temporal immune response of the recovered population. After this period, the persons return unprotected to the suscectible group.

The basic reproduction number $R_0 = \frac{\beta}{\xi}$ is the average number of infected contacts from an infected individual. R_0 is a very important parameter in the model because it allows

us to find out the secondary cases produced by one person in a field. If $R_0 \leq 1$, the pandemic will disappear spontaneously, while with $R_0 > 1$ it will continue spreading.

Because $S(t) = N - I(t)$, it follows

$$\frac{dI}{dt} = (\beta - \xi) I - \frac{\beta}{N} I^2. \tag{2.3}$$

By solving of the equation (2.3), with the initial condition $I(0) = I_0$, the following solution is obtained:

$$I(t) = \frac{(\beta - \xi) N I_0}{(\beta - \xi) N e^{-(\beta-\xi)t} + \beta I_0 \left[1 - e^{-(\beta-\xi)t}\right]}. \tag{2.4}$$

The long-term behaviour of the system can be estimated considering the possible values of $\beta - \xi$.

If $(\beta - \xi) > 0$, $e^{-(\beta-\xi)t} \to 0$ as $t \to \infty$, then the following equilibrium state is obtained:

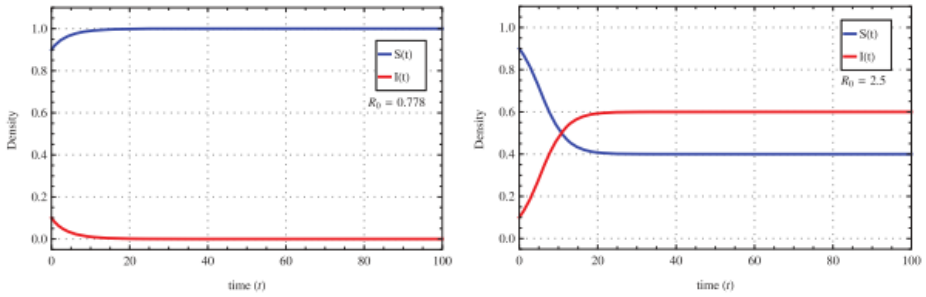
$$\lim_{t\to\infty} I(t) = \frac{(\beta - \xi) N I_0}{\beta I_0} = \left(1 - \frac{\xi}{\beta}\right) N.$$

When $(\beta - \xi) < 0$, $e^{-(\beta-\xi)t} \to \infty$ as $t \to \infty$, and then $\lim_{t\to\infty} I(t) = 0$.

There are two equilibrium states for the SIS model. The equilibrium points for I which can be obtained by setting $\frac{dI}{dt} = \beta I\left(1 - \frac{I}{N}\right) - \xi I = 0$. The first is $I^*=0$ (disease free state) and for $S^* = N - I^* = N$. The second equilibrium point can be obtained from $\frac{dI}{dt} = \frac{(\beta - \xi) N}{\beta} = \left(1 - \frac{\xi}{\beta}\right) N = \left(1 - \frac{1}{R_0}\right) N = \frac{N}{R_0}(R_0 - 1)$. From this, the second fixed point is $I^* = \frac{N}{R_0}(R_0 - 1)$ and $S^* = \frac{N}{R_0}$.

The point (N, 0) represents the disease-free equilibrium points (DFE) and the point $\left(\frac{N}{R_0}, \frac{N}{R_0}(R_0 - 1)\right)$ represents the endemic equilibrium (EE) points. Figure 4 a)

represents the case when $R_0 \leq 1$ and the system is in a disease-free steady state. Figure 4 b) represents the case when $R_0 > 1$ and the infected individual leads to more than one infection, thus spreading the pathogen in the population, ξ.



a) $R_0 = 0.78, \beta = 0.7, \xi = 0.9.$ b) $R_0 = 2.5, \beta = 0.5, \xi = 0.2.$

Figure 4. *Density versus time for the SIS model without vital dynamics where N = 1, S(0) = 0.9, and I(0) = 0.1, [12].*

2.3. The General Epidemic Model – SIR Model

The SIR model was proposed by Kermack and McKendrick in 1927. The KermackMcKendrick model is based on several assumptions: there are no births and deaths in the population, the population is closed, all recovered individuals have complete immunity and cannot be infected again. In the SIR model, the population is divided into three groups: susceptible individuals - S(t), infective individuals - I(t) and recovered individuals R(t).

The number of individuals in each class changes during time, since $S(t)$, I(t), and $R(t)$ are functions of time t. The total population size N is the sum of the sizes of these three classes: $S(t) + I(t) + R(t) = N$, where N is the total population.

First, the SIR model without vital dynamics is considered. This model is not a dynamic model, i.e., the rate of birth and the death rate are not included in the model. The SIR without dynamics model is represented in Figure 5.

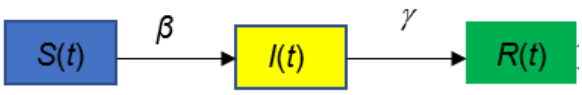


Figure 5. *SIR model without vital dynamics.*

The SIR model is described by the system of the following differential equations:

$$\frac{dS}{dt} = -\frac{\beta SI}{N}, \quad \frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I, \quad \frac{dR}{dt} = \gamma I \tag{2.5}$$

and $I(0) = I_0$, $S(0) = S_0$ and $R(0) = R_0$ are the initial conditions.

The parameter β is the transmissions rate, and the parameter γ is the recovery rate. The recovery time is $\tau_{recovery} = \frac{1}{\gamma}$.

Because $\frac{dS}{dt} < 0$, the number of susceptible individuals is always decrementing, independently of the initial condition S(0). Since $S(t)$ is monotonous and positive t, $\lim_{t\to\infty} S(t) = S(\infty)$. The number of recovered R(t) is monotone and bounded by N, and $\frac{dR}{dt} > 0$ for all t (the number of recovered individuals always incrementing), $\lim_{t\to\infty} R(t) = R(\infty)$.

On the other side, the number of infected individuals can be either monotonically decreasing to zero or it may have non-monotone behaviour by first increasing to some maximum value, and after this decreasing to zero. The number will firstly be increasing if $\frac{dI_0}{dt} = (\frac{\beta S(0)}{N} - \gamma)I(0) > 0$. So, the number of infected individuals will be increasing initially if $\frac{\beta S(0)}{N} - \gamma > 0$ from where $\frac{\beta S(0)}{N\gamma} > 1$.

To determine S_∞ and R_∞, the first equation is divided by the third equitation from (2.5):

$$\frac{dS}{dR} = -\frac{\beta S}{\gamma N} \tag{2.6}$$

which is a separable equation, and its solution is:

$$S(t) = S(0)e^{-\frac{\beta}{\gamma N}(R(t)-R(0))} \geq S(0)e^{-\frac{\beta}{\gamma N}} > 0. \tag{2.7}$$

So, it follows that $S_\infty > 0$ and this variable is called the final size of the epidemic.

The epidemic will be finished, if $\lim_{t\to\infty} I(t) = I_\infty$ then $I_\infty = 0$. The last can be obtained by integrating the first equation in (2.5).

On the beginning of an epidemic, it is assumed that the number of infected individuals $I(0)$ is from $\lim_{t\to\infty} I(t) = 0$ and $\lim_{t\to\infty} S(t) = S(\infty)$, the number of infected individuals is $S(0) - S(\infty)$.

With integrating of the equation (2.6) the total number of infected people from the beginning of the epidemic up to its end is obtained:

$$I(\infty) - I(0) = \frac{\gamma N}{\beta} \ln\left(\frac{S(\infty)}{S(0)}\right) - S(\infty) + S(0) + c \tag{2.8}$$

where c is a constant and $S(\infty)$ is the number of susceptible individuals at the end of the pandemic. Since the number of the initially infected individuals is small, the following is obtained:

$$\ln\left(\frac{S(\infty)}{S(0)}\right) = \frac{\beta}{\gamma}\left(\frac{S(\infty)}{N} - 1\right) + c', \tag{2.9}$$

where c' is a constant.

The basic reproduction number $R_0 = \frac{\beta}{\gamma}$ is an average number of secondary infections caused by a single infectious individual. This notation is a general notation in literature for the basic reproduction number, and it is different from the initial number of recovered individuals $R(0) = R_0$. The importance of the basic reproduction number can be seen if second equation in (2.5) is rewritten in the following way:

$$\frac{dI}{dt} = \left(\frac{\beta S}{\gamma N} - 1\right)\gamma I = \left(R_0 \frac{S}{N} - 1\right)\gamma I. \tag{2.10}$$

If (2.10) is not positive, then $R_0 S(0) \le N$ the epidemic will be avoided. Otherwise, if (2.10) is positive $R_0 S(0) > N$ the epidemic will occur.

Figure 6 represents the limiting values of the *S*, *I*, and R groups for different values of R_0 where N is normalized to 1.

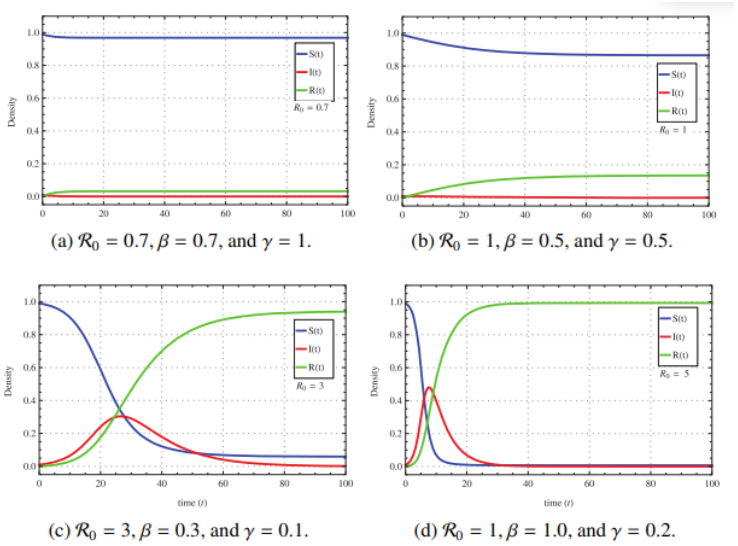


Figure 6. *Density versus time for the SIR model without vital dynamics where N = 1, S(0) = 0.99, I(0) = 0.01 and R(0) = 0, [12].*

If in the SIR model the birth and death rates are included, then the SIR model is with vital dynamics.

2.4. SIRS model

The SIRS model is obtained from the basic SIR model, but in this model the infected individuals can recover from infection and return unprotected to the suscpectible group. After that, these persons can be infected again. The SIRS model is represented in Figure 7.

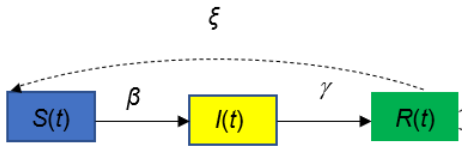


Figure 7. *SIRS model*

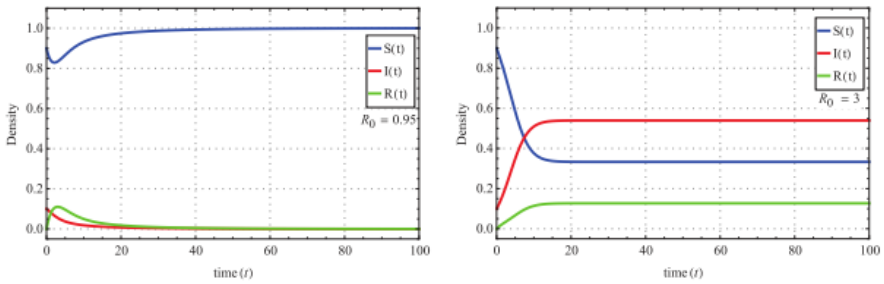
The model is described by the following system of differential equations:

$$\frac{dS}{dt} = -\frac{\beta SI}{N} + \xi R, \quad \frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I, \quad \frac{dR}{dt} = \gamma I - \xi R$$

Where the parameter ξ is the rate by which the recovered people return to the susceptible group due the loss of immunity and $\tau_{immunity} = \frac{1}{\xi}$ is the time of immunization.

This system has two equilibrium points DFE, $(S^*, I^*, R^*) = (N, 0, 0)$, and EE equilibrium point $(S^*, I^*, R^*) = \left(\frac{N}{R_0}, \frac{N}{R_0}\frac{\xi}{\gamma+\xi}(R_0-1), \frac{N}{R_0}\frac{\gamma}{\gamma+\xi}(R_0-1)\right)$.

Figure 8 a) represents the case when $R_0 \leq 1$ and the system is in a disease-free steady state. Figure 8 b) represents the case when $R_0 > 1$.



a) $R_0 = 0.95$, $\beta = 0.95$, $\gamma = 1$, $\xi = 0.5$. b) $R_0 = 3$, $\beta = 0.6$, $\gamma = 0.2$, $\xi = 0.85$

Figure 8. *Density versus time for the SIR model without vital dynamics where N = 1, S(0) = 0.9, I(0) = 0.1 and R(0) = 0, [12].*

If in the SIRS model the birth and death rates are included, then the SIRS model is with vital dynamics.

2.5. SEIR Epidemic Model

The total population is divided into four groups: susceptible individuals - *S*(t), exposed individuals - *E*(t), infective individuals - *I*(t) and recovered individuals - *R*(t).

In the SEIR model, a new group of individuals – the exposed group is added. Exposed individuals are the people who are infected, but they are not yet able to transmit the disease to other individuals.

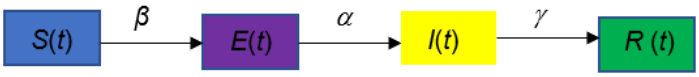


Figure 9. *SEIR model*

The size of population is constant and $N = I(t) + S(t) + R(t) + E(t)$. For this model, the ordinary differential equations are:

$$\frac{dS}{dt} = -\frac{\beta SI}{N}, \quad \frac{dE}{dt} = \frac{\beta SI}{N} - \alpha E, \quad \frac{dI}{dt} = \alpha E - \gamma I, \quad \frac{dR}{dt} = \gamma I$$

where $\alpha = \dfrac{1}{\tau_{incubation}}$ is the incubation rate, i.e., the rate by which the latent patients become infectious and $\tau_{incubation}$ is the time of incubation.

Since latency delays the start of the individual's infectious period, the secondary spread from an infected individual will occur at a later time compared to an SIR model, which has no latency. Therefore, including a longer latency period will result in a slower initial growth of the outbreak. However, since the model does not include mortality, the basic reproductive number, $R_0 = \dfrac{\beta}{\gamma}$, does not change.

3. Conclusion

SI, SIS, SIR, SIRS, and SEIR are mathematical models that can be used for spreading of different infectious diseases. The advantages of using mathematical models in epidemiology are in the fact that mathematical representation of biological processes enables transparency and accuracy regarding the epidemiological assumptions. This allows researchers to test understanding of the disease epidemiology by comparing model results and results obtained from observation. Also, mathematical models can help predict outcomes of taking measures for stopping the spread of infections, as well as taking new appropriate measures.

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