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МАТЕМАТИЧЕСКОЕ МОДЕЛИРОВАНИЕ ДИНАМИКИ ЭПИДЕМИЙ И РАСПРОСТРАНЕНИЯ ЗАБОЛЕВАНИЙ С ИСПОЛЬЗОВАНИЕМ МОДЕЛИ SIR

MATHEMATICAL MODELING OF EPIDEMIC DYNAMICS AND DISEASE SPREAD USING THE SIR MODEL

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Аннотация. В данной статье рассматривается применение математического моделирования для анализа и прогнозирования распространения инфекционных заболеваний, с акцентом на модели SIR (Susceptible–Infectious–Recovered — восприимчивые, инфицированные, выздоровевшие), которая широко используется в эпидемиологии. Представлен общий обзор модели, после чего выводятся три ее фундаментальных дифференциальных уравнения, описывающие динамику изменения численности каждой из групп населения. Численные решения получены с использованием метода Эйлера для двух примерных случаев, результаты которых затем анализируются с целью выявления пика эпидемии и момента начала ее спада. Дополнительно исследуется взаимосвязь между величиной пика и исходным числом восприимчивых индивидов с помощью графического анализа. В статье обсуждаются ограничения как самой модели SIR, так и метода Эйлера, подчеркивается, что выбор параметров, таких как коэффициенты заражения и восстановления, существенно влияет на результаты моделирования. Цель работы — углубить понимание механизмов распространения заболеваний и

продемонстрировать, как математические методы могут способствовать разработке эффективных стратегий контроля и управления эпидемиями. Кроме того, исследование обращает внимание на практическое значение математического моделирования в эпидемиологии: применение подобных моделей позволяет прогнозировать возможные сценарии развития эпидемии, оценивать эффективность профилактических мер (вакцинации, изоляции, ограничения контактов) и оптимизировать распределение ресурсов здравоохранения. Использование модели SIR служит основой для построения более сложных моделей — SEIR, SIRS, SEIRD и других, что делает ее фундаментальным инструментом для дальнейших исследований в области динамики инфекционных процессов.
Abstract. This article examines the use of mathematical modeling for analyzing and predicting the spread of infectious diseases, focusing on the SIR (Susceptible–Infectious–Recovered) model, which is widely used in epidemiology. A general overview of the model is presented, followed by the derivation of its three fundamental differential equations that describe the dynamics of changes in each population group. Numerical solutions are obtained using Euler's method for two sample cases, and the results are then analyzed to determine the epidemic peak and the point at which it begins to decline. Additionally, the relationship between the height of the peak and the initial number of susceptible individuals is investigated through graphical analysis. The paper discusses the limitations of both the SIR model and Euler's method, emphasizing that the choice of parameters—such as infection and recovery rates—significantly affects the modeling results. The purpose of this work is to deepen the understanding of disease transmission mechanisms and to demonstrate how mathematical methods can support the development of effective epidemic control and management strategies. Furthermore, the study highlights the practical importance of mathematical modeling in epidemiology: such models make it possible to predict potential epidemic scenarios, assess the effectiveness of preventive measures (vaccination,

isolation, contact restrictions), and optimize the allocation of healthcare resources. The SIR model also serves as a foundation for constructing more complex models—such as SEIR, SIRS, SEIRD, and others—making it a fundamental tool for further research into the dynamics of infectious processes.
Ключевые слова: численный анализ, моделирование эпидемий, модель SIR, динамика заболевания, пиковая заболеваемость, инфекционные заболевания, метод Эйлера, восприимчивые и инфицированные, управление эпидемическим процессом

Keywords: numerical analysis, epidemic modeling, SIR model, disease dynamics, peak incidence, infectious diseases, Euler's method, susceptible and infected, epidemic management

Introduction

Epidemics have been a persistent challenge throughout human history, claiming millions of lives through diseases such as plague, cholera, and typhoid. Despite significant advances in medicine, new epidemics continue to emerge, some spreading at alarming rates and causing profound societal and economic disruptions. While many infections are relatively mild, others—such as COVID-19 and AIDS—have had catastrophic global effects. A common challenge in epidemic control is the rapid spread of diseases, often outpacing both population growth and timely interventions. Understanding the dynamics of disease transmission is therefore crucial for formulating effective strategies for containment and eradication.

The relevance of mathematical models in addressing these challenges has been well-documented across numerous studies. Anderson and May (1991) laid foundational work in understanding how diseases spread and how mathematical models, particularly the SIR model, can inform control strategies [1]. Keeling and Rohani (2008) further refined these models by exploring both theoretical frameworks and practical applications, making their insights indispensable for the current study [8]. The SIR model's development by Kermack and McKendrick

(1927) marked a pivotal moment in epidemic modeling, providing a simple yet powerful tool for understanding the progression of infectious diseases [7]. This model, as elaborated by Diekmann et al. (2013), remains central to epidemiological research today, with its influence extending to numerous subsequent developments in the field [4].

Murray (2002) contributes a broader understanding of mathematical biology, extending the foundational knowledge of epidemic modeling to a variety of biological systems, which helps contextualize the dynamics observed in human populations [9]. Brauer et al. (2019) offer additional insights into mathematical models in epidemiology, discussing the intricacies and limitations of models like the SIR in predicting real-world epidemic outcomes [2]. Ferguson et al. (2005) illustrate the practical application of such models in pandemic scenarios, particularly in assessing the effectiveness of mitigation strategies [5]. Colizza et al. (2007) take a different approach by incorporating network theory, showing how transportation systems can accelerate the global spread of epidemics, which further emphasizes the complexity of predicting disease dynamics [3].

In light of these contributions, this article explores the application of mathematical methods to the analysis and forecasting of epidemic behavior, with a particular focus on the SIR model. The discussion begins with an overview of the model and the mathematical derivation of its three fundamental differential equations. Euler’s method is then employed to obtain numerical solutions for two example scenarios. These results are analyzed to identify key points in the epidemic curve, such as the peak and the point of decline, with particular attention paid to how the initial number of susceptible individuals influences these outcomes. Graphical representations support the analysis, illustrating how different parameters can alter the trajectory of an epidemic. The article also critically examines the limitations of both the SIR model and Euler’s method, highlighting how variations in parameter choices can impact numerical solutions and the accuracy of predictions. By combining theoretical insights and practical applications, this article contributes to the ongoing dialogue on how mathematical models can enhance our understanding of epidemic dynamics and guide public health responses.

1. The SIR Model: Concepts and Mathematical Foundations

The SIR (Susceptible-Infectious-Removed) model belongs to the class of compartmental methods because it divides the entire population potentially involved in the spread of a disease into major groups (compartments).

In fact, the SIR model can be applied not only to people but also to animals and plants.

The basic SIR model divides the population into three compartments: susceptible, infected, and recovered [6].

The first compartment, S, comprises individuals susceptible to the disease—that is, those who can get sick. Quite often, at the onset of a disease, this group includes the entire population of a country, region, town, and similar areas, except for those already infected. Also, a portion of the population may be immune to the disease, such as through vaccination, so they won't be in the S group.

The second compartment, I, includes individuals who are already infectious—in other words, those who are currently sick and can transmit the disease.

The third compartment, R, consists of individuals who were either immune from the beginning, have recovered from the disease and thus acquired immunity, or have died from it. In the context of the basic SIR model, all are considered "removed" from the chain of transmission.

In the simplest SIR model, individuals can only move from the S group to the I group, and from the I group to the R group. The variant discussed in this paper does not account for factors such as repeated infections, asymptomatic carriers, birth and death dynamics, and other similar considerations (see the Limitations and Extensions of the SIR Model section for more detail).

Any SIR model has three variables: S, I and R, denoting the number of people in each respective group.

The total population is denoted as N. Obviously, $N = S + I + R$.

There are also two important constants (parameters) that are unique to each specific epidemic:

Infection transfer rate β

This constant shows how often the infection is transferred from one person to another, i.e., how often people move from the S group to the $,I$ group. This constant combines the rate of encounters between people and the probability of infection transfer during an encounter.

If, for instance, every infected person meets, on average, 10 people every day, then $10 \times S/N$ of these meetings are with susceptible people. Further, if 10%, or a 0.1 fraction, of such meetings, on average, result in infection transfer, then, on average, $0.1 \times 10 \times S/N$ susceptible people are infected every day by each infected person. Hence, the total number of susceptible people infected every day is $I \times 0.1 \times 10 \times S/N$. The constant β is defined as:

$$\beta = 0.1 \times 10 = 1$$

Recovery rate γ

This constant shows how often sick (infected) people recover, i.e., how often people move from the I group to the R group.

If, for example, the average duration of the disease is 10 days, then every day, on average, 1/10, or a 0.1 fraction of the sick people will recover, therefore:

$$\gamma = \frac{1}{10} = 0.1$$

Now, the relationship between S, I, and R is examined.

First, these numbers change over time, so they are functions of time: $S(t)$, $I(t)$ and $R(t)$.

As mentioned above, $I \times 1 \times S/N$ susceptible people will be infected every day, which means that the susceptible population will decrease every day by that number:

$$S(t + \Delta t) = S(t) - 1 \times \frac{S(t) \times I(t)}{N} \times \Delta t$$

$$S(t+\Delta t)-S(t)=-1\times\frac{S(t)\times I(t)}{N}\times\Delta t$$

$$\frac{S(t+\Delta t)-S(t)}{\Delta t}=-1\times\frac{S(t)\times I(t)}{N}$$

Where Δt appears in the formula above, it represents the small period of time (in days) over which the change in the S group is observed, assuming its size remains constan On the right-hand side, Δt is multiplied because $I\times 1$ $\times\, S/N$ is the daily number of infected people, while, for example, the hourly number w $\times\, 1/24\times S/N = I\times 0.0417\times S/N$.

Now, if the limit of the left-hand side is taken as $\Delta t\to 0$, the following equality is fair:

$$\lim_{\Delta t\to 0}\frac{S(t+\Delta t)-S(t)}{\Delta t}=-1\times\frac{S(t)\times I(t)}{N}$$

The limit on the left-hand side represents the derivative of $S(t)$ by definition, leading to the following result:

$$\frac{dS}{dt}=-1\times\frac{S\times I}{N}=-\beta\frac{SI}{N}$$

Additionally, each day, the number of infected people will, on one hand, increase by the number of susceptible individuals who will become infected th rough contact, and, on the other hand, decrease by the number of infected individuals who will be removed, that is, by $0.1\times I(t)$.

$$I(t+\Delta t)=I(t)+1\times\frac{S(t)\times I(t)}{N}\times\Delta t-0.1\times I(t)\times\Delta t$$

$$I(t+\Delta t)-I(t)=1\times\frac{S(t)\times I(t)}{N}\times\Delta t-0.1\times I(t)\times\Delta t$$

$$\frac{I(t+\Delta t)-I(t)}{\Delta t}=1\times\frac{S(t)\times I(t)}{N}-0.1\times I(t)$$

$$\lim_{\Delta t \to 0} \frac{I(t + \Delta t) - I(t)}{\Delta t} = 1 \times \frac{S(t) \times I(t)}{N} - 0.1 \times I(t)$$

Using the definition of the derivative, the following is obtained:

$$\frac{dI}{dt} = 1 \times \frac{S \times I}{N} - 0.1 \times I = \beta \frac{SI}{N} - \gamma I$$

Finally, each day, the number of removed individuals will increase by the number of infected individuals who will be removed, that is, by $0.1 \times I(t)$.

$$R(t + \Delta t) = R(t) + 0.1 \times I(t) \times \Delta t$$

$$R(t + \Delta t) - R(t) = 0.1 \times I(t) \times \Delta t$$

$$\lim_{\Delta t \to 0} \frac{R(t + \Delta t) - R(t)}{\Delta t} = 0.1 \times I(t)$$

Here, by using the definition of the derivative, the following is obtained:

$$\frac{dR}{dt} = 0.1 \times I = \gamma I$$

Thus, a system of three differential equations is obtained, involving the independent variable t, the functions $S(t), I(t), R(t)$, and their derivatives dS/dt, $dI/$, dR/dt.

$$\frac{dS}{dt} = -\beta \frac{SI}{N}$$

$$\frac{dI}{dt} = \beta \frac{SI}{N} - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

The model assumes that the total population N is constant:

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

This follows from the fact that the derivative of N is zero:

$$\frac{dN}{dt} = \frac{d}{dt}\big(S(t) + I(t) + R(t)\big) = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = -\beta \frac{SI}{N} + \left(\beta \frac{SI}{N} - \gamma I\right) + \gamma I = 0$$

Hence, N is constant.

2. Applying the SIR Model: Solutions, Parameters, and Initial Conditions

The solution to the system of differential equations describing the evolution of $S(t)$, $I(t)$, $R(t)$ can be obtained through analytical methods in specific cases or more commonly through numerical techniques. One approach is to solve them numerically using the Euler method.

According to the Euler method, if a function is observed over a sufficiently small interval around a certain point x, the function values within that interval can be approximated with reasonable accuracy using the formula:

$$f(x+\Delta x) \approx f(x) + \frac{df}{dx}\Delta x$$

To solve the differential equations in the SIR model, it is necessary to know the total population N, the initial values of the three functions $S(0)$, $I(0)$, $R(0)$, as well as the constants β, γ.

Next, a time increment Δt must be selected, and the following equations can then be used:

$$S(t+\Delta t) = S(t) + \frac{dS}{dt}\Delta t = S(t) - \beta \times \frac{S(t)\times I(t)}{N} \times \Delta t$$

$$I(t+\Delta t) = I(t) + \frac{dI}{dt}\Delta t = I(t) + \beta \times \frac{S(t)\times I(t)}{N} \times \Delta t - \gamma \times I(t) \times \Delta t$$

$$R(t+\Delta t) = R(t) + \frac{dR}{dt}\Delta t = R(t) + \gamma \times I(t) \times \Delta t$$

It is most convenient to use $\Delta t = 1$, meaning one day. Then, for $t = 0$, the following holds:

$$S(1) = S(0+1) = S(0) - \beta \times \frac{S(0)\times I(0)}{N} \times 1$$

$$I(1) = I(0+1) = I(0) + \beta \times \frac{S(0)\times I(0)}{N} \times 1 - \gamma \times I(0) \times 1$$

$$R(1) = R(0+1) = R(0) + \gamma \times I(0) \times 1$$

Once the values of $S(1)$, $I(1)$ and $R(1)$ are obtained, the values of $S(2)$, $I(2)$ and $R(2)$ can be calculated in the same manner, followed by $S(3)$, $I(3)$ and $R(3)$, and so on.

However, $\Delta t = 1$ may not be sufficiently small and could produce results that differ significantly from the exact solution (the actual function).

This is illustrated in Figure 1, where the blue line represents the actual function, and the red line shows the solution obtained using the Euler method.

The Euler method with $\Delta t = 0.25$ and Microsoft Excel was used to compute numerical solutions for all the models discussed later in this paper.

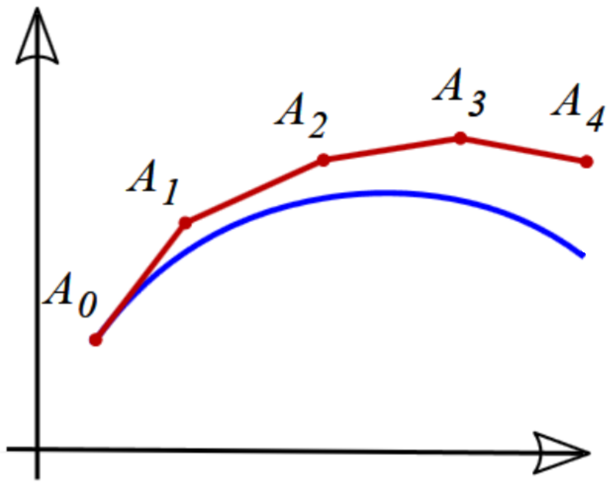


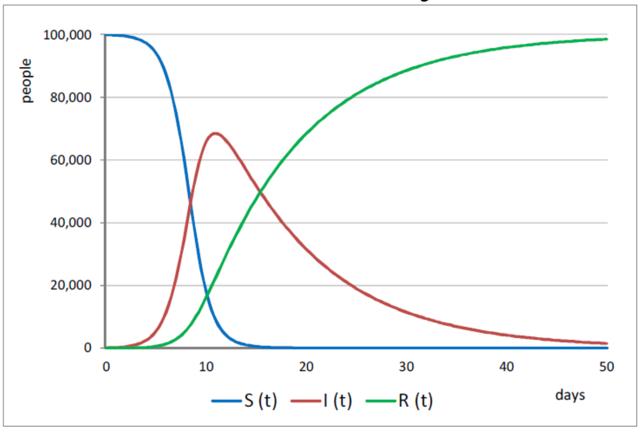
Figure 1. Graphical illustration of the Euler method. The blue line represents the exact function, while the red line shows the approximate solution.

Sample Model 1 has the following parameters:

$N = 100000 \quad S(0) = 99900 \quad I(0) = 100 \quad R(0) = 0$

$\beta = 1.00 \quad \gamma = 0.10$

This means that, in a population of 100000 people, there were initially 100 infected individuals, with all others susceptible. The infection transfer rate was 1.00, and the recovery rate was 0.10.



N =	100000	Δt =	0.25
S (0) =	99900		
I (0) =	100		
R (0) =	0		
β =	1.00		
γ =	0.10		
t	S (t)	I (t)	R (t)
0.00	99900	100	0
0.25	99875	122	3
0.50	99844	150	6
0.75	99807	184	9
...	...	...	...
49.25	3	1637	98360
49.50	3	1596	98401
49.75	3	1556	98441
50.00	3	1517	98479

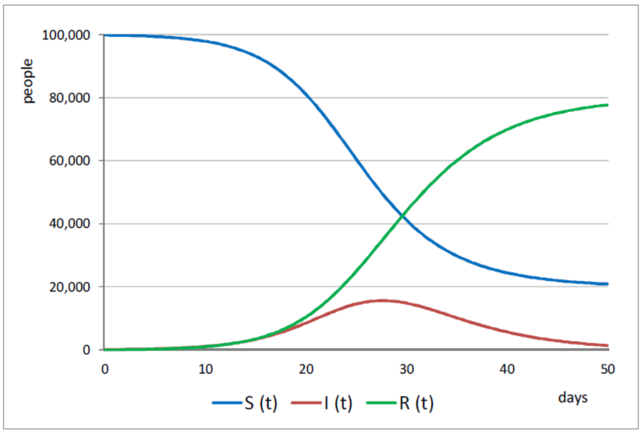
Figure 2. Initial values, parameters, and solution for Model 1 using the Euler method.

Sample Model 2 has the following parameters:

$$N = 100000 \qquad S(0) = 99900 \qquad I(0) = 100 \qquad R(0) = 0$$

$$\beta = 0.50 \qquad \gamma = 0.25$$

This means that, in a population of 100000 people, there were initially 100 infected individuals, with all others susceptible. The infection transfer rate was 0.50, and the recovery rate was 0.25.



N =	100000	Δt =	0.25
S (0) =	99900		
I (0) =	100		
R (0) =	0		
β =	0.50		
γ =	0.25		
t	S (t)	I (t)	R (t)
0.00	99900	100	0
0.25	99888	106	6
0.50	99874	113	13
0.75	99860	120	20
...	...	...	...
49.25	21003	1544	77453
49.50	20963	1488	77549
49.75	20924	1434	77642
50.00	20886	1382	77732

Figure 3. Initial values, parameters, and solution for Model 2 using the Euler method.

The Euler method provides more accurate results (that is, a numerical solution closer to the actual or exact solution) when smaller time increments are used.

Please refer to the Limitations of the Euler Method section later in the paper for further details.

An in-depth examination of Model 1 and Model 2 reveals important insights.

The dynamics of the number of infected individuals in these models are influenced by factors such as the transmission rate, recovery rate, initial population distribution, and contact patterns within the population.

Model 1 shows that:

1. The maximum fraction of infected people is 68% (68445), which represents the peak of the red line.
2. The majority of the population became infected, as evidenced by the fact that the blue line (representing susceptible individuals) nearly reaches zero (3).

Model 2 shows that:

1. The maximum fraction of infected people was approximately 16% (15622).
2. A significant portion of the population (79%) became infected, as the blue line (representing susceptible individuals) falls to 21% (20886).

It is evident that the infection transfer rate is lower, and the recovery rate is higher in Model 2, which explains the difference.

However, the formal criterion for determining when the epidemic reaches its peak is when the rate of change of infected individuals becomes zero.

Considering the second differential equation, it can be transformed:

$$\frac{dI}{dt} = \beta\frac{SI}{N} - \gamma I = I \times \left(\beta\frac{S}{N} - \gamma\right)$$

Since $I \geq 0$, the sign of dI/dt depends on the sign of $\beta S/N - \gamma$. This means that dI/dt is positive (and hence $I(t)$ increases) when $\beta S/N - \gamma$ is positive:

$$\beta\frac{S}{N} - \gamma > 0$$

$$\frac{\beta}{\gamma} > \frac{N}{S}$$

Therefore, the number of infected people $I(t)$ continues to grow as long as $S(t)$ remains large enough to satisfy the above inequality. Accordingly, the epidemic will begin to subside once $S(t)$ becomes small enough to satisfy:

$$\frac{\beta}{\gamma} \leq \frac{N}{S}$$

Therefore, the critical point of the epidemic spread is characterized by the following equation:

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

Indeed, this can be observed in both the graphs and the data tables used to plot them. In Model 1, the peak of the epidemic occurs at $t = 11$, when $S(11) = 8611$ and:

$$\frac{\beta}{\gamma} = \frac{1.00}{0.10} = 10 \approx \frac{N}{S(11)} = \frac{100000}{8611} = 11.6$$

Obviously, $10 \approx 11.6$ is too rough an approximation; however, a more precise solution ($\Delta t = 0.025$), discussed in the Limitations of the Euler Method section later in the paper, shows that the peak occurs at $t = 10.375$, with $S(10.375) = 9991$. Therefore, the condition is more accurately met:

$$\frac{\beta}{\gamma} = \frac{1.00}{0.10} = 10 \approx \frac{N}{S(10.375)} = \frac{100000}{9991} \approx 10$$

In Model 2, at the peak of the epidemic is at $t = 27.5$, $S(27.5) = 49982$ and

$$\frac{\beta}{\gamma} = \frac{0.50}{0.25} = 2 \approx \frac{N}{S(27.5)} = \frac{100000}{49982} \approx 2$$

The ratio above is known as the basic reproduction number:

$$R_0 = \frac{\beta}{\gamma}$$

Howard (Howie) Weiss of the Georgia Institute of Technology (Atlanta, GA, USA), in his paper The SIR Model and the Foundations of Public Health (Weiss, 2013), proves the Epidemic Threshold Theorem, which fully supports this idea [10].

H. Weiss introduces another ratio known as the effective reproduction number:

$$R_e = \frac{S(0)}{N} \times \frac{\beta}{\gamma}$$

The theorem states that if $R_e \leq 1$ at the onset of an epidemic, then $I(t)$ decreases to zero as $t \to \infty$ however, if $R_e > 1$, then $I(t)$ increases, reaches a maximum, and subsequently decreases to zero as $t \to \infty$. Furthermore, $R_e = S(0)/N \times \beta/\gamma \leq 1$ implies that $\beta/\gamma \leq N/S(0)$, exactly as previously deduced. H. Weiss also derives a formula for the maximum number of infected individuals in the case where the entire population is initially susceptible [10]:

$$I_{max} = N \times \left(1 - \frac{1 + \ln R_0}{R_0}\right)$$

For Model 1, $I_{max} = 100000 \times (1 - (1 + \ln 10)/10) \approx 66974$

For Model 2, $I_{max} = 100000 \times (1 - (1 + \ln 2)/2) \approx 15343$

These values are quite close to those obtained using the Euler method with $\Delta t = 0.25$ (68445 for Model 1 and 15622 for Model 2). The differences can be attributed to the approximate nature of the Euler method results and the fact that the initial number of susceptible individuals (99900) is slightly less than the total population.

Based on the formula for I_{max}, the dependence of the I_{max}/N ratio (the peak fraction of the infected population) on R_0 was graphed (Figure 4).

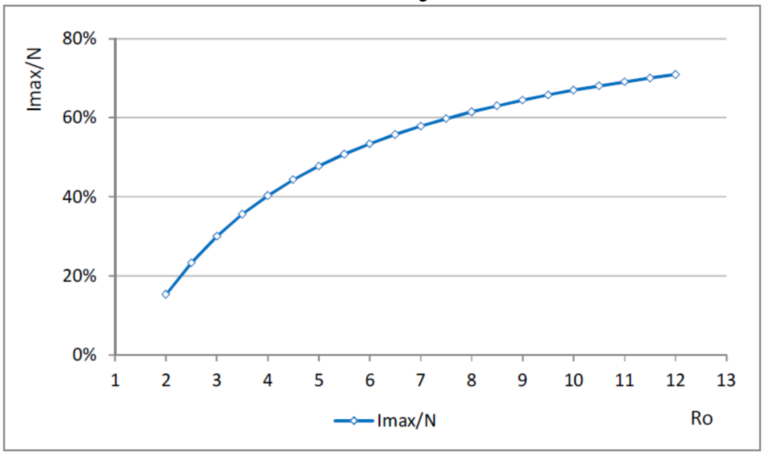
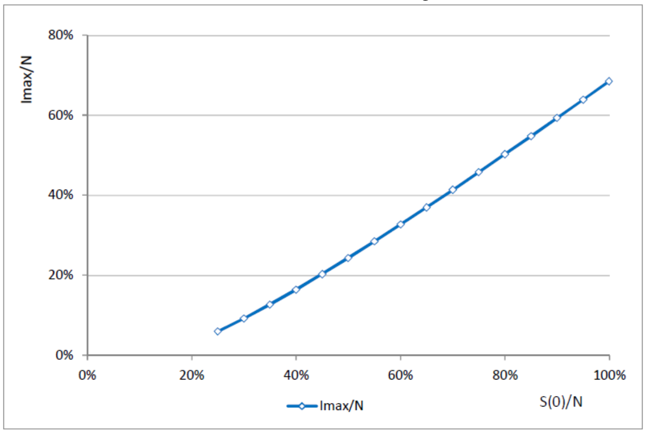


Figure 4. Graph illustrating the relationship between the peak fraction of the infected population (I_{max}/N) and the basic reproduction number (R_0).

As R_0 increases, the peak fraction of infected individuals grows at an increasingly slower pace. This implies, for example, that decreasing R_0 from 4 to 3 impacts I_{max}/N more significantly than reducing R_0 from 10 to 9.

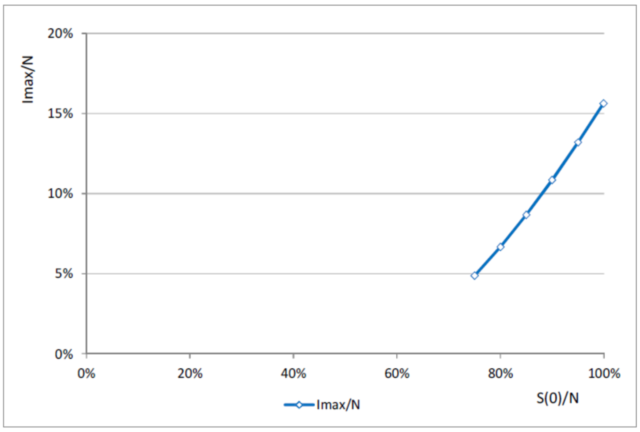
To reduce $R_0 = \beta/\gamma$, β can be lowered by implementing precautionary measures such as staying at home when sick to decrease encounter rates, wearing masks to reduce the probability of transmission during encounters, etc. On the other hand, γ can be increased by shortening the average duration of illness through timely prophylactics and proper medical treatment.

The Euler method was also used in MS Excel to explore how I_{max}/N depends on $S(0)/N$, which refers to how the peak fraction of infected individuals is influenced by the initial number of susceptible people. The following graphs display the relationship between I_{max}/N and $S(0)/N$ for both Model 1 and Model 2 (Figure 5 and Figure 6).



S(0)/N	Imax/N
99.9%	68.4%
95%	63.9%
90%	59.3%
85%	54.8%
80%	50.3%
75%	45.8%
70%	41.4%
65%	37.0%
60%	32.7%
55%	28.5%
50%	24.4%
45%	20.3%
40%	16.4%
35%	12.7%
30%	9.2%
25%	6.0%
20%	
15%	
10%	
5%	

Figure 5. Relationship between the peak fraction of the infected population (I_{max}/N) and the initial fraction of susceptible individuals ($S(0)/N$) for Model 1, where the infection transfer rate $\beta = 1.0$ and the recovery rate $\gamma = 0.10$.



S(0)/N	Imax/N
99.9%	15.62%
95%	13.19%
90%	10.85%
85%	8.67%
80%	6.67%
75%	4.87%
70%	
65%	
60%	
55%	
50%	
45%	
40%	
35%	
30%	
25%	
20%	
15%	
10%	
5%	

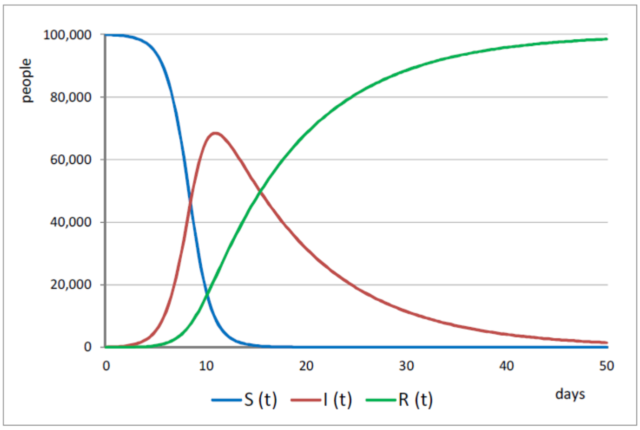
Figure 6. Relationship between the peak fraction of the infected population (I_{max}/N) and the initial fraction of susceptible individuals ($S(0)/N$) for Model 2, where the infection transfer rate $\beta = 0.5$ and the recovery rate $\gamma = 0.25$.

The graphs appear incomplete because, for certain values of $S(0)$, the number of infected individuals $I(t)$ does not reach its maximum within the 50-day period. However, since the curves closely resemble straight lines, the relationship between the variables is nearly linear. To illustrate this, the solution for Model 1 with an initial susceptible fraction of $S(0)/N = 60\%$ is shown below:

$N = 100000 \quad S(0) = 60000 \quad I(0) = 100 \quad R(0) = 40000$

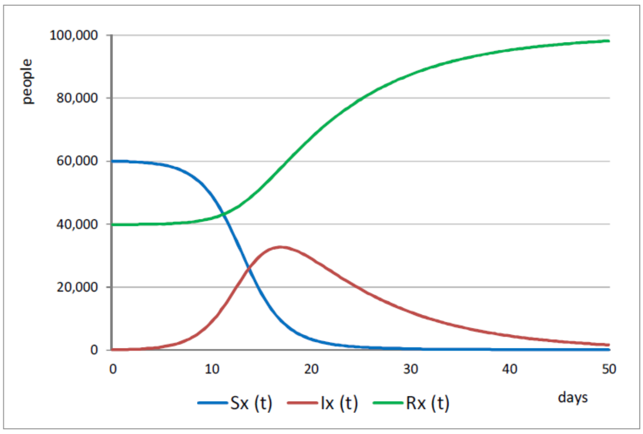
$\beta = 1.00 \qquad \gamma = 0.10$

In other words, within a population of 100000 individuals, there were initially 100 infected, 60000 susceptible, and 39900 immune (removed). The infection transmission rate was 1.00, and the recovery rate was 0.10. For easier comparison, the solution for Model 1 with S(0)/N = 99.9%—previously shown in Figure 2—is repeated below (Figure 7).



N =	100000	Δt =	0.25
S (0) =	99900		
I (0) =	100		
R (0) =	0		
β =	1.00		
γ =	0.10		
t	S (t)	I (t)	R (t)
0.00	99900	100	0
0.25	99875	122	3
0.50	99844	150	6
0.75	99807	184	9
...	...	...	...
49.25	3	1637	98360
49.50	3	1596	98401
49.75	3	1556	98441
50.00	3	1517	98479

Figure 7. Initial values, parameters, and numerical solution for Model 1 with $S(0)/N = 99.9\%$



N =	100000	Δt =	0.25
S (0) =	60000		
I (0) =	100		
R (0) =	39900		
β =	1.00		
γ =	0.10		
t	S (t)	I (t)	R (t)
0.00	60000	100	39900
0.25	59985	113	39903
0.50	59968	127	39905
0.75	59949	142	39908
...	...	...	...
49.25	153	1803	98044
49.50	152	1759	98089
49.75	151	1716	98133
50.00	151	1673	98176

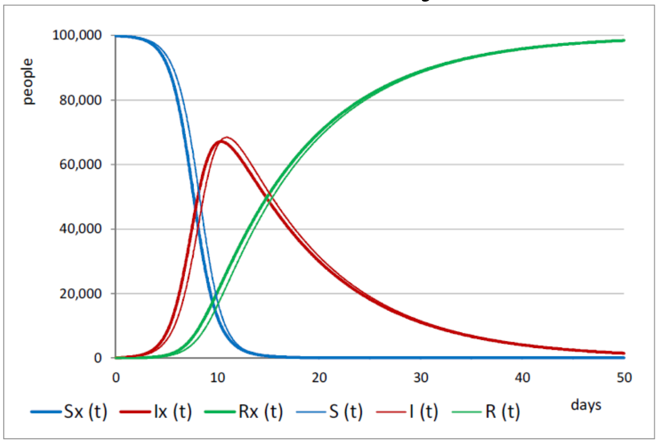
Figure 8. Initial values, parameters, and numerical solution for Model 1 with $S(0)/N = 60\%$

Limitations and extensions of the SIR model

The basic form of the SIR model presented in this paper has several limitations. It does not consider the possibility of reinfection, which is relevant for diseases like influenza where immunity may be short-lived—such cases are better modeled without the “Removed” (R) category. The model also excludes asymptomatic carriers who can transmit the disease without showing symptoms; this can be addressed by adding a “Carrier” (C) compartment. Additionally, it overlooks diseases with a significant incubation period during which infected individuals are not yet infectious, incorporating an “Exposed” (E) group addresses this. As noted in the section on the mathematics of the SIR model, the total population is treated as constant, meaning vital dynamics such as births and deaths are not considered. While this may be acceptable for short-term outbreaks, it becomes problematic for long-lasting diseases like COVID-19 or AIDS, where demographic changes are significant. Including vital statistics may also require accounting for maternal immunity in newborns, which can be modeled by adding a “Maternal Immunity” (M) compartment. Vaccination effects are also not included in the basic model but can be represented by introducing a “Vaccinated” (V) group. These and other enhancements can be incorporated into extended versions of the SIR model.

Limitations of the Euler method

The Euler method, being a numerical approach, produces approximate results that can differ from the exact analytical solution. Greater accuracy is achieved when smaller increments of the independent variable are used. As an example, Figure 9 displays two solutions for Model 1 calculated with different time steps: $\Delta t = 0.25$ and $\Delta t = 0.025$. The appropriate choice of Δt depends on the desired level of precision. While the discrepancy may not be immediately apparent in the graph, a portion of the corresponding data table reveals notable differences in the values of $S(t)$, $I(t)$ and $R(t)$, with deviations reaching nearly 35%.



t	Sx (t)	Ix (t)	Rx (t)	S (t)	I (t)	R (t)
...	...	...	...	...	...	...
7.000	73643	23312	3045	81705	16304	1991
7.025	73214	23683	3103	81372	16596	2032
7.050	72780	24057	3162	81039	16889	2073
7.075	72343	24435	3222	80706	17181	2113
7.100	71901	24816	3283	80373	17473	2154
7.125	71455	25200	3345	80040	17765	2195
7.150	71005	25587	3408	79707	18058	2236
7.175	70550	25977	3472	79374	18350	2276
7.200	70092	26370	3537	79041	18642	2317
7.225	69630	26767	3603	78708	18934	2358
7.250	69164	27166	3670	78375	19227	2399
7.275	68694	27567	3738	77998	19555	2447
7.300	68221	27972	3807	77621	19884	2495
7.325	67744	28379	3877	77245	20213	2543
7.350	67263	28789	3948	76868	20541	2591
7.375	66779	29201	4020	76491	20870	2639
7.400	66292	29615	4093	76114	21199	2687
7.425	65801	30032	4167	75738	21527	2735
7.450	65307	30451	4242	75361	21856	2783
7.475	64810	30872	4318	74984	22185	2831
7.500	64310	31295	4395	74607	22513	2879
7.525	63806	31720	4474	74188	22877	2936
7.550	63300	32147	4553	73768	23241	2992
7.575	62792	32575	4633	73348	23604	3048
7.600	62280	33005	4715	72928	23968	3104
7.625	61766	33436	4797	72508	24331	3161
7.650	61250	33869	4881	72088	24695	3217
7.675	60731	34303	4965	71668	25059	3273
7.700	60211	34738	5051	71248	25422	3330
7.725	59688	35174	5138	70828	25786	3386
7.750	59163	35611	5226	70408	26150	3442
...	...	...	...	...	...	...

Figure 9. Two solutions for Model 1 using different time increments (thinner lines correspond to the less precise solution with $\Delta t = 0.25$). Missing values for the $\Delta t = 0.25$ solution were estimated using linear interpolation.

Conclusions

The SIR model plays a pivotal role in understanding epidemic dynamics by compartmentalizing the population into susceptible, infectious, and removed groups. By utilizing mathematical principles, particularly Euler's method for solving differential equations, this model offers valuable insights into how epidemics unfold over time. The analysis of different sample models demonstrates that both the infection transfer rate (β) and the recovery rate (γ) significantly influence the peak and the eventual decline of an epidemic. Key findings show that a higher infection transfer rate leads to a greater proportion of the population becoming infected, while a higher recovery rate accelerates the epidemic's resolution.

The critical point where the epidemic peaks can be determined by the balance between the infection transfer rate and the susceptible population, as highlighted by the basic reproduction number (R_0). Furthermore, the model underscores the importance of early intervention measures aimed at lowering R_0, such as reducing encounter rates or increasing recovery rates, to mitigate the spread and impact of an epidemic.

Despite the utility of the SIR model, it has limitations, including assumptions about constant population size, homogeneity of the population, and exclusion of other factors like re-infection or asymptomatic carriers. Nevertheless, the insights gained through this model can inform strategies for epidemic control and help prepare for future outbreaks. By understanding the mathematical foundations and the influence of various parameters, policymakers and health professionals can better predict and manage the spread of infectious diseases.

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