Modeling the Transmission of Covid-19

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1. Introduction

The progress of an epidemic through the population is highly amenable to mathematical modelling. In particular, the first attempt to model and hence predict or explain patterns dates back over 100 years, although it was the work of Kermack and McKendrick that established the basic foundations of the subject. These early models, and many subsequent revisions and improvements, operated on the principle that individuals can be classified by their epidemiological status—most simply susceptible to the infection, infected and therefore infectious, and recovered and hence no longer infectious. (We stress that this classification is based upon an individual's ability to host and transmit a pathogen, and may be relatively unconnected to their medical status.) In this review, we focus on how such models can be used to predict the future outcome of an epidemic process (or the impact of control measures); however, models may also have a more theoretical use as explanatory tools elucidating fundamental principles of transmission and the factors driving epidemic behavior.

Coronavirus disease 2019 (COVID-19) is a contagious disease caused by a virus, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The first known case was identified in Wuhan, China, in December 2019.The disease

quickly spread worldwide, resulting in the COVID-19 pandemic. The symptoms of COVID-19 are variable but often include fever, cough, headache,fatigue, breathing difficulties, loss of smell, and loss of taste.Symptoms may begin one to fourteen days after exposure to the virus. At least a third of people who are infected do not develop noticeable symptoms.of those who develop symptoms noticeable enough to be classified as patients, most (81%) develop mild to moderate symptoms (up to mild pneumonia), while 14% develop severe symptoms (dyspnea, hypoxia, or more than 50% lung involvement on imaging), and 5% develop critical symptoms (respiratory failure, shock, or multiorgan dysfunction).Older people are at a higher risk of developing severe symptoms. Some people continue to experience a range of effects (long COVID) for months after recovery, and damage to organs has been observed.Multi-year studies are underway to further investigate the long-term effects of the disease.COVID-19 transmits when people breathe air contaminated by droplets and small airborne particles containing the virus. The risk of breathing these is highest when people are in close proximity, but they can be inhaled over longer distances, particularly indoors. Transmission can also occur if contaminated fluids are splashed or sprayed in the eyes, nose, or mouth, or, more rarely, via contaminated surfaces. People remain contagious for up to 20 days and can spread the virus even if they do not develop symptoms.

2. The Kermack-McKendriek Model

Considered as one of the first compartmental models, Kermack-McKendrick epidemic model was developed in the late 1920s with the pioneering work of Kermack and McKendrick.The model is described as the SIR model for the spread of disease, which consists of a system of three ordinary differential equations characterizing the changes in the number of susceptible (*S*), infected (*I*), and recovered (*R*) individuals in a given population. The model is a good one for many infectious diseases, despite its simplicity. Ever since, numerous and more complex compartmental mathematical models have been developed. For instance, in biology, modeling is particularly useful in studying organs like the lungs, heart, intestinal edema and cancer, etc. Almost all these models take their source on Kermack-McKendrick's model and serve to help gain insights into the transmission and control mechanisms of diseases like HIV, TB, malaria and their interactions with others. Then most of the works done on modeling the dynamics of epidemiological diseases have been limited only to models based on (a system of) classical first-order differential equations. However, there is a growing interest in applying fractional calculus to mathematical epidemiology since it has turned out recently that many phenomena in different fields, including sciences, engineering, and technology, can be described very successfully by the models using fractional-order differential equations. In this model, a population of size

N(t) is divided into different classes, disjoint and based on their disease status. At time *t*, *S=S(t)* is the part of population representing individuals susceptible to a disease, *I=I(t)* is the part of population representing infectious individuals, *R=R(t)* is the part representing individuals that recovered from the disease. One of the most famous epidemic models is Kermack-McKendrick SIR model. Let *N(t)=S(t)+I(t)+R(t)*, the Kermack-McKendrick then is

$$\frac{ds}{dt} = -\beta SI \qquad (1)$$

$$\frac{dI}{dt} = \beta SI - vI \qquad (2)$$

$$\frac{dR}{dt} = vI \qquad (3)$$

subject to the initial condition: $S(0) = S_0, I(0) = I_0,$ and $R(0) = R_0,$ and therefore, $N(0) = S_0 + I_0 + R_0$. The basic reproduction number $R_0 = \beta S_0/\mu$ is the threshold that completely determines the dynamics of transmission of the epidemic. We have three cases:

- If R_0 >1, then *I(t)* increases (disease will spread, epidemic case).
- If R_0 <1, then *I(t)* decreases (disease will disappear).
- If R_0 =1, then *I(t)* will remain the same.

3. More complex model to study the spread of Covid-19

Let us define the following parameters:

β = infection rate
μ = death rate , the same for all individuals
v = recovery rate
α = death rate caused by Covid-19
γ = rate by which recovered individuals have lost their immunity and became susceptible the disease

We assume relationships between $S, I,$ and R as showing in the following diagram:

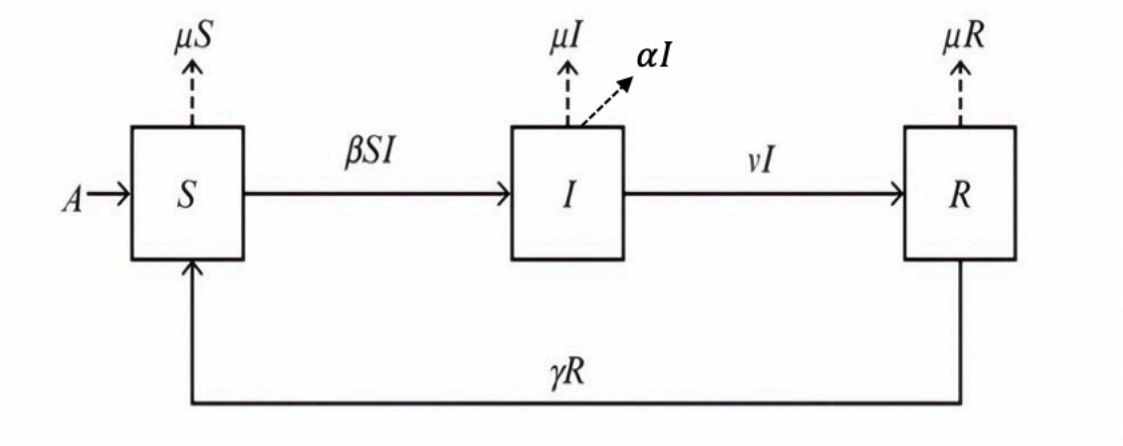

Figure 1: Diagram shown relationships between S, I, and R.

We thus have the following system of differential equations:

$$\frac{dS}{dt} = A - \beta SI + \gamma R - \mu S \qquad (4)$$

$$\frac{dI}{dt} = \beta SI - vI - \alpha I - \mu I \qquad (5)$$

$$\frac{dR}{dt} = vI - \gamma R - \mu R \qquad (6)$$

To show that the solutions are bounded, we introduce a differential equation for N(t), which is obtained by adding $N(t) = S(t) + I(t) + R(t)$. Thus,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt}.$$

We know $N(0) = N_{\circ}$, where $N_{\circ}$ is a constant.

$$\frac{dN}{dt} = A - \mu\,(S + I + R) - \alpha I$$

$$= A - \mu N - \alpha I.$$

We know that

$$\frac{dN}{dt} = A - \mu N - \alpha I < A - \mu N.$$

Therefore, if
$\frac{dN}{dt} = A - \mu N$,

bounded then

$$\frac{dN}{dt} = A - \mu N - \alpha I,$$

is also bounded. We thus have

$$\frac{dN(t)}{A - \mu N} = dt$$

$$\int \mu \frac{dN}{A - \mu N} = -\mu \int dt$$

$$\ln(A - \mu N) = -\mu t + c$$

$$-\mu N = e^{-\mu+c} - A$$

$$N = \frac{-e^{\mu+c}}{\mu} + \frac{A}{\mu}$$

$$N(0) = -\frac{e^{0+c}}{\mu} + \frac{A}{\mu} = N_o$$

$$\frac{e^{0+C}}{\mu} = N_\circ - \frac{A}{\mu}$$

$$e^{C} = -\mu N_\circ + A$$

$$C = \ln|-\mu N_\circ + A|$$

$$N(T) = \frac{A}{\mu} - \frac{e^{-\mu t}}{\mu} \times e^{\ln|-\mu N_\circ + A|}$$

$$= \frac{-1}{\mu} e^{-\mu t}[-\mu N_\circ + A] + \frac{A}{\mu}$$

$$= N_\circ e^{-\mu t} - \frac{A}{\mu} e^{-\mu t} + \frac{A}{\mu}$$

$$N(t) = N_\circ e^{-\mu t} + \frac{A}{\mu}[1 - e^{-\mu t}]$$

$N(t) \to \frac{A}{\mu}$ as $t \to \infty$.

Thus

$\frac{dN}{dt} = A - \mu N - \alpha I$ is also bounded.

That means the solutions (total population density) increase with time until they reach the value $\frac{A}{\mu}$, which means the solutions are bounded by this value. Therefore, the model is biologically reasonable.

3.1 Equilibrium points

Equilibrium is a state of a system which does not change. If the dynamics of a system is described by a differential equation (or a system of differential equations), then equilibria can be estimated by setting a derivative (all derivatives) to zero.

Example:

$$\frac{dN}{dt}=f(N)$$

To find equilibria we have to solve the equation f(N)=0.

Now we apply that to our system in equations (4-6). The SIR model has a disease-free equilibrium point (DFE) which is

$$(\boldsymbol{S_0}, \boldsymbol{I_0}, \boldsymbol{R_0}) = \left(\frac{\boldsymbol{A}}{\boldsymbol{\mu}}, \mathbf{0}, \mathbf{0}\right)$$

3.2 Linear stability analysis

We now analyze the local stability of the system (4-6) around the DFE point.

The Jacobian matrix of the system

$$\frac{dS}{dt} = A - \beta SI + \gamma R - \mu S = f(S, I, R)$$

$$\frac{dI}{dt} = \beta SI - vI - \alpha I - \mu I = g(S, I, R)$$

$$\frac{dR}{dt} = vI - \gamma R - \mu R = z(S, I, R)$$

is as follow:

$$\mathrm{J} = \begin{pmatrix} \frac{\partial f}{\partial S} & \frac{\partial f}{\partial I} & \frac{\partial f}{\partial R} \\ \frac{\partial g}{\partial S} & \frac{\partial g}{\partial I} & \frac{\partial g}{\partial R} \\ \frac{\partial Z}{\partial S} & \frac{\partial Z}{\partial I} & \frac{\partial Z}{\partial R} \end{pmatrix} = \begin{pmatrix} -\beta I - \mu & -\beta S & \gamma \\ \beta I & \beta S - v - \alpha - \mu & 0 \\ 0 & v & -\gamma - \mu \end{pmatrix}.$$

We then find the Jacobian matrix at the disease-free equilibrium (DFE):

$$\mathrm{J}\left(\frac{A}{\mu}, 0, 0\right) = \begin{pmatrix} -\mu & -\beta\frac{A}{\mu} & \gamma \\ 0 & \beta\frac{A}{\mu} - (v + \alpha + \mu) & 0 \\ 0 & v & -\mu - \gamma \end{pmatrix}$$

$$|J - \lambda I| = \left| \begin{pmatrix} -\mu & -\beta\frac{A}{\mu} & \gamma \\ 0 & \beta\frac{A}{\mu} - (v + \alpha + \mu) & 0 \\ 0 & v & -\mu - \gamma \end{pmatrix} - \begin{pmatrix} \lambda & 0 & 0 \\ 0 & \lambda & 0 \\ 0 & 0 & \lambda \end{pmatrix} \right|$$

$$= \begin{vmatrix} -\mu - \lambda & -\beta\frac{A}{\mu} & \gamma \\ 0 & \beta\frac{A}{\mu} - (v + \alpha + \mu) - \lambda & 0 \\ 0 & v & -\gamma - \mu - \lambda \end{vmatrix}$$

The characteristic polynomial, therefore, is

$$(\mu + \lambda)(-\beta\frac{A}{\mu} - (v + \alpha + \mu) - \lambda)(\mu + \gamma + \lambda)$$

The eigenvalues are:

$\lambda_1 = -\mu, \qquad \lambda_2 = -\mu - \alpha - \gamma, \qquad \lambda_3 = \beta\frac{A}{\mu} - (v + \alpha + \mu).$

Hence the DFE is stable (as in case **B** in Figure 2) if

$$\beta \frac{A}{\mu} < v + \alpha + \mu, \quad (7)$$

and the DFE is unstable (as in case **C** in Figure 2) if

$$\beta \frac{A}{\mu} > v + \alpha + \mu. \quad (8)$$

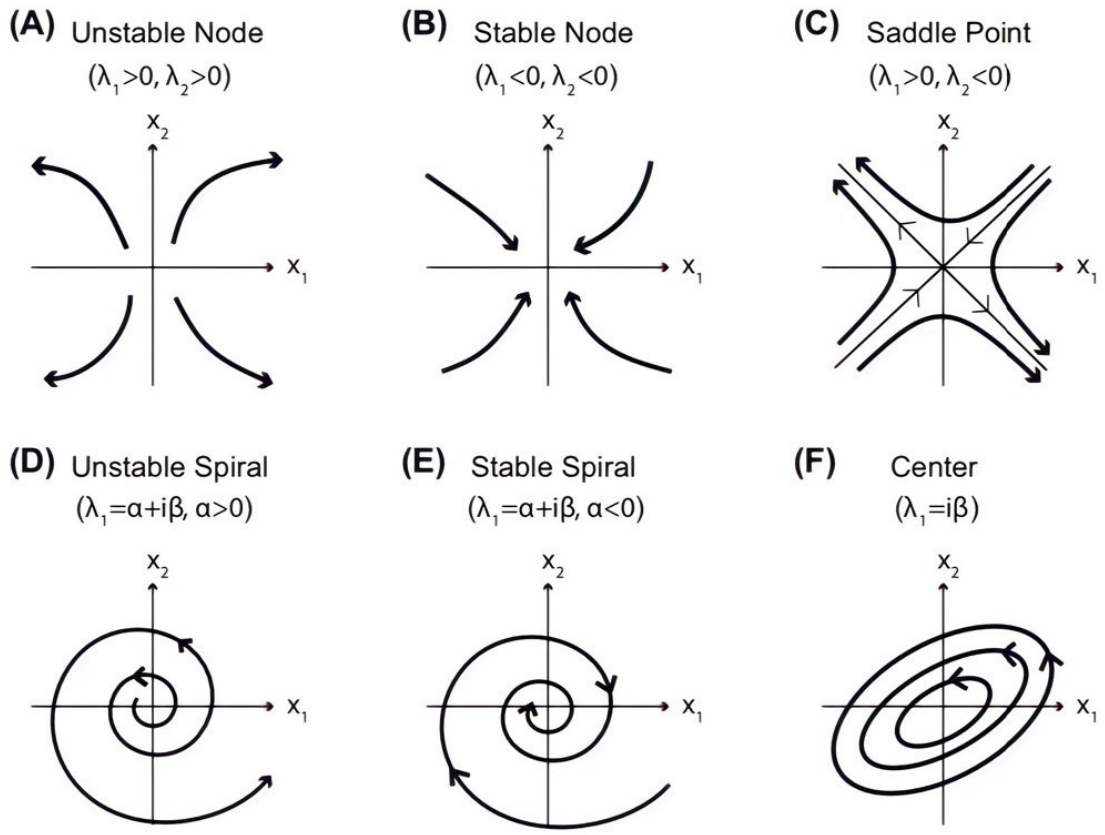

Figure 2: Phase portrait for the system (4-6).

3.3 The basic reproduction number (R_0)

An important concept in epidemiology is the basic reproduction number, defined as follows: In a healthy population we introduce one infection and compute the

expected infection among the susceptibles caused by this single infection. We call it the expected secondary infection, or basic reproduction number, and denote it by R_0.

Since both sides of the inequalities (7-8) are positive, we can divide both sides of the inequalities by $\beta\frac{A}{\mu}$ to obtain

$$1 < \frac{v+\alpha+\mu}{\beta\frac{A}{\mu}}, \quad (9)$$

and the DFE is unstable if

$$1 > \frac{v+\alpha+\mu}{\beta\frac{A}{\mu}}. \quad (10)$$

We can clearly see that the disease will disappear when

$$1 < \frac{v+\alpha+\mu}{\beta\frac{A}{\mu}}$$

and the disease will spread when

$$1 > \frac{v+\alpha+\mu}{\beta\frac{A}{\mu}}$$

We thus take the basic reproduction number (R_0) to be

$$R_0 = \frac{\beta A}{\mu(v+\alpha+\mu)}.$$

This is because we know the disease disappear when the DFE is stable ($R_0 < 1$) and the disease will spread when the DFE is unstable ($R_0 > 1$).

4. Numerical simulations

In this section, we numerically using Matlab and *ode45 solver* examine the impact of the basic reproduction number R_0 on the model solutions. We analytically showed that the disease will disappear when $R_0 < 1$ and the disease will spread when $R_0 > 1$. Here, we confirm that numerically. In Table 1, we picked random values that make $R_0 = 0.5 < 1$ and show in Figure 3 that the disease disappeared. In Figure 3 when $R_0 = 1.5 > 1$ the infectious disease continues spreading. We see

in Figure 3 the solution (S) moves towards the stable equilibrium point

$$(S_0, I_0, R_0) = \left(\frac{A}{\mu}, 0,0\right) = (2,0,0),$$

since the parameters in Table 1 make it locally stable ($R_0 = 0.5 < 1$). While the solutions move away from this point when $R_0 = 1.5 > 1$ since it becomes unstable. We thus confirmed our analytical result.

Table 1: Parameters used in Figure 3.

Parameter	Description	Value
S_0	Initial susceptible population (at $t = 0$)	0.9
I_0	Initial infected population (at $t = 0$)	0.1
R_0	Initial recovered population (at $t = 0$)	0
A	Birth rate	2
β	Effective contact rate	1
v	Recovery rate	1
μ	Normal death rate	1
α	Death rate caused by Covid-19	1
γ	Rate by which recovered individuals have lost their immunity and became susceptible the disease	1
R_0	Basic reproduction number ($R_0 = \frac{\beta A}{\mu(v+\alpha+\mu)}$)	0.6667

Table 2: Parameters used in Figure 4.

Parameter	Description	Value
S_0	Initial susceptible population (at $t = 0$)	0.9
I_0	Initial infected population (at $t = 0$)	0.1
R_0	Initial recovered population (at $t = 0$)	0
A	Birth rate	2
β	Effective contact rate	3
v	Recovery rate	1
μ	Normal death rate	1
α	Death rate caused by Covid-19	1
γ	Rate by which recovered individuals have lost their immunity and became susceptible the disease	1

R_0	Basic reproduction number $(R_0 = \frac{\beta A}{\mu(v+\alpha+\mu)})$	2

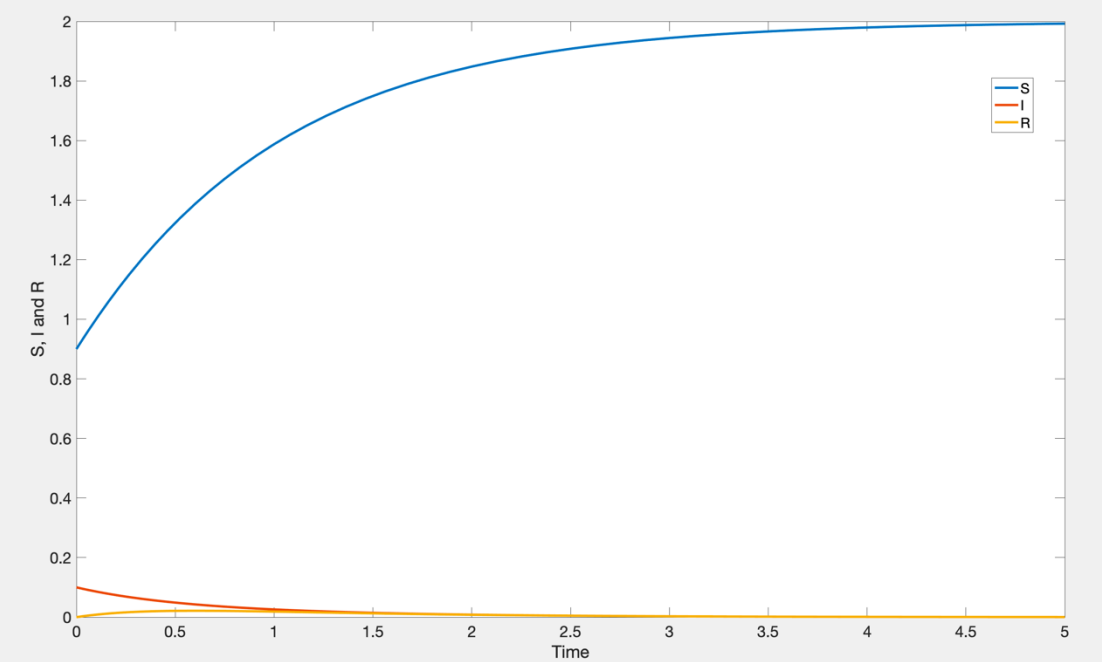

Figure 3: The solution of the system (4-6) with parameters from Table 1when R0<1. The disease disappeared.

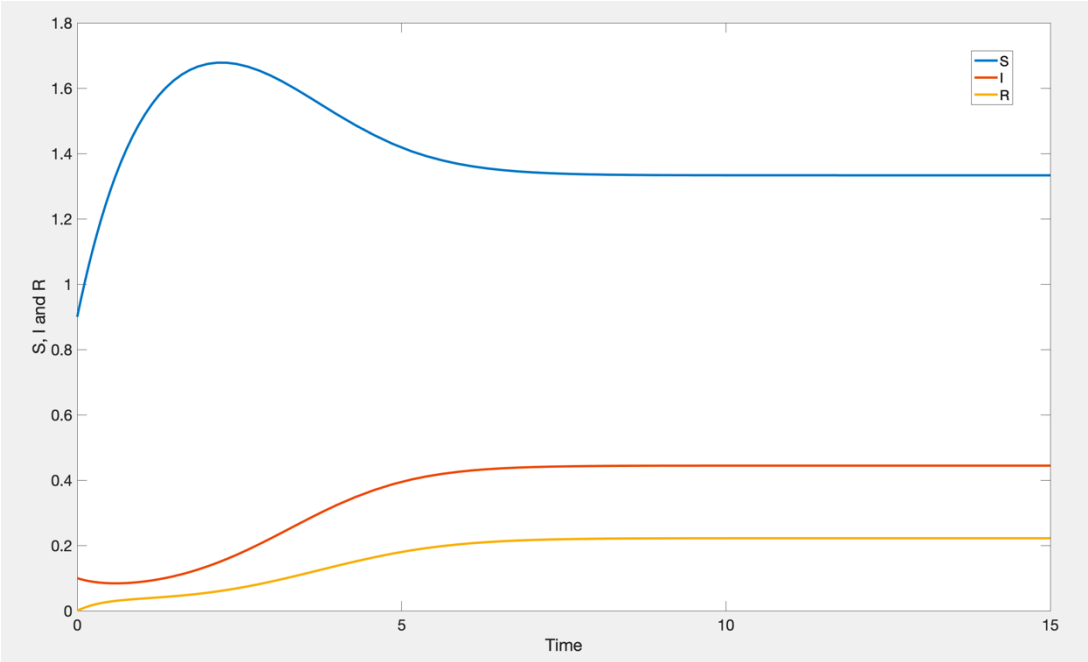

Figure 4: The solution of the system (4-6) with parameters from Table 1when R0>1. The disease continues spreading.

References:

[1] WO. Kermack, AG. McKendrick, A Contribution to the Mathematical Theory of Epidemics . Proceedings of the Royal Society A, Mathematical, Physical and Engineering Sciences,700, 1927
[2] Avner Fridman, Introduction to mathematical biology, springer 2016.

Appendix

```
function dv = fun_SIR(t,v)
global A gamma mu beta V alpha
S = v(1); % susceptible
I = v(2); % infected
R = v(3); % recovered
dv = zeros(3,1);
dv(1) = A - beta*S*I +gamma*R- mu*S;
dv(2) = beta*S*I - V*I-alpha*I - mu*I;
dv(3) = V*I- gamma*R - mu*R;
```

```
%-------------------------------------------------------------------------------
```

```
global A V gamma mu beta alpha
%% parameters
A = 2;
V = 1;
gamma = 1;
mu = 1;
beta = 1;
alpha = 1;
R0 = beta*A/(mu*(V+alpha+mu))
%% initial conditions
S0 = 0.9; % susceptible
I0 = 0.1; % infected
R0 = 0; % recovered
init = [S0; I0; R0];
tspan = [0,15];
[t,v] = ode45('fun_SIR',tspan,init);
plot(t,v(:,1),'LineWidth',3), hold on
plot(t,v(:,2),'LineWidth',3), hold on
plot(t,v(:,3),'LineWidth',3), hold on
```