Dynamic behaviors of a modified SIRS model in epidemic diseases

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COVID-19 is a new infectious disease that has infected millions of people across the world. Early this year, the World Health Organization declared this disease a pandemic(1), raising fears that governments were not working aggressively and quickly enough to stop the disease's spread. The global response to end the further spread of the virus has involved the application of mathematical models. Although various models have been developed in relation to COVID-19, they all help fight the ongoing public health crisis by providing estimates of reproduction rates, analyzing changes in initial estimates following intervention implementation, predicting the potential for further global spread, and quantifying the burden and severity of the pandemic.

In this paper, we developed a modified form of the classical susceptible-infected-recovered (SIR) model for analyzing the spread and control of the COVID-19 pandemic. In recap, the SIR model is an epidemiological framework for analyzing the spread of an infectious disease within a closed population(2). This model applies ordinary differential equations (ODE) to track the number of exposed (E), susceptible (S), and recovered (R) individuals over a period of time. The SIR model's main assumptions are: the exposed develop permanent immunity to the disease after recovery; there is a homogenous mixing of the exposed and susceptible populations; and that the total population remains constant over time.

Based on the assumptions mentioned above, we extended the classical SIR model by including the dead individuals (D), and dividing the exposed into two categories, namely, reported infected compartment (V) and unreported infected compartment (U). The two groups of patients were assumed to have different recovery and death rates since they will be subjected to different interventions. For the reported infected compartment, we assumed that they are aware of their condition and follow CDC guidelines for the management of COVID-19. These guidelines include not having contact with the susceptible individuals and not spreading the disease. On the other hand, it was assumed that since the unreported infected compartment will still have the ability to expose a large number of the susceptible population as long as they are not quarantined or hospitalized.

Background

A common variant of the SIR model is the susceptible-infected-recovered-susceptible (SIRS) model. It can be remembered that the SIR model is based on the assumption that individuals develop lifelong immunity to diseases upon recovery. This is the case for several infectious diseases. However, for certain diseases such as seasonal influenza, immunity may wane over time, meaning individuals may become infected again(1). In this regard, the SIRS model allows recovered individuals to fail to develop immunity and rejoin the susceptible state.

The SIR model has been utilized in the study of the epidemiology of the COVID 19 pandemic to simulate real-life scenarios. However, most empirical studies show that the transmission of this disease does not correspond to the assumptions of the classic SIR model. Some research mentioned that not all individuals with travel history or severe symptoms had been tested to determine if they have been exposed to COVID-19(3). This is mainly due to the shortage of testing kits and systematic bureaucracy in hospitals. Therefore, there is a high likelihood that the number of reported COVID-19 infections is much lower than the actual number of infections. Discrepancies between reported and actual infections are exacerbated further because some patients experience mild or no symptoms at all, leading to a lack of testing.

Model Explanation

In this section, we developed the classic SIR model mentioned in the previous section to simulate the real-life scenario better.

Assumptions and Considerations. In reality, the transmission of an infectious disease is more likely to occur when there is adequate contact of a susceptible individual with an infected individual. However, people who get exposed to infectious diseases should not become infectious immediately. It will take some time for these exposed individuals to develop symptoms and become infectious. Therefore, we extend the classic model by including the individuals exposed to the infectious disease but have not yet become infectious (E).

Notice that the spread of disease happens on a much faster timescale than the population's birth and death. Therefore, we ignore the vitality of the population and only take the disease's deaths into account.

Due to the shortage of testing kits, hospitals, and systematic bureaucracy, only a sub-sample of people with severe symptoms or travel history was eventually tested. The number of reported infections, especially in the developing stages of the pandemic, is likely to be much lower than the actual number of infections. We assume these unreported infections were unrecognized because they often experience mild or no symptoms or misdiagnosed.

Based on this assumption, we then further extended the classical SIRS model structure by including the dead individuals and divided the infected compartment into the reported infected group and the unreported infected group. We set these two groups of patients to have different recovery rates and death rates since they will get different treatments. For the reported infected compartment, we assume they are self-aware and follow the CDC's instructions to protect themselves and others. Therefore, they will not have any contact with the susceptible individuals and spread the disease. On the other hand, if not hospitalized or quarantined, the unreported infected individuals will still have the ability to introduce the virus to the susceptible individuals they have contact with.

Model Construction. Our model can be explained in a network diagram, and the interaction between individual compartments was demonstrated in Fig.1 . The nodes represent the different stages in our model, and the edges represent the reactions.

Let us first consider the human population (N) separated into seven epidemiological states:

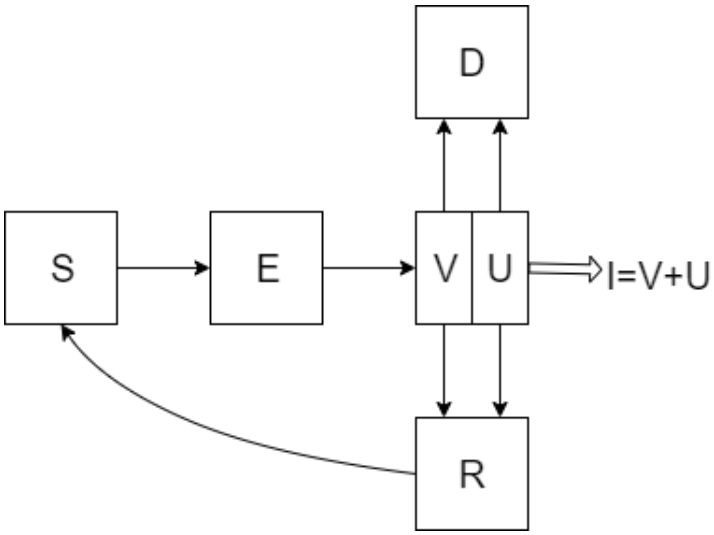

Fig. 1. The compartment interaction diagram for our epidemic model.

1. Susceptible (S). This presents a healthy individual that can be infected.
2. Exposed (E). This presents an individual exposed to the disease but not yet infectious.
3. Infected (I). This presents an individual who is infectious and will be in either one of the following states.

    - Unreported (U). This presents an individual who gets infected, having mild or no symptoms that did not get tested. We let P_V denote the proportion of the unreported population of infected people.
    - Reported (V). This presents an individual who gets infected, having disease symptoms, gets tested, and documented. We let P_U to be the proportion of the reported population of infected people.

4. Recovered (R). This presents an individual who gets recovered from the disease.
5. Dead (D). This presents an individual who did not survive the infection.

The actions taken between different states are as follows:

- Susceptible people (S) move into the exposed people (E) when they have contact with the infected people (I) and unreported infectious people (U) at a transition rate β.
- After that, exposed people (E) become infectious (I) at a transition rate ϵ.
- Then infected people were divided into two groups: reported (V) and unreported (U) with proportional transmission rates ϵP_V and ϵP_U respectively.
- In addition, the reported (V) and unreported (U) group will recover at a rate of γ_V and γ_U, respectively. Individuals in these two groups may die at a rate d_V and d_U, respectively. Thus the reported (V) and unreported (U) people move into recovery (R) and death (D) with a transition rate γ_U, γ_V and d_U, d_V, respectively.
- Finally, the individuals who are recovered from this disease (R) may fail to develop immunity and move to the susceptible group with a transition rate ξ.

Consider the statements above, the model dynamics can be described by the following systems of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= -\beta S(I-V) + \xi R \\
\frac{dE}{dt} &= \beta S(I-V) - \epsilon E \\
\frac{dI}{dt} &= \epsilon E - (d_V + \gamma_V)V - (d_U + \gamma_U)U \\
\frac{dV}{dt} &= \epsilon E P_V - (d_V + \gamma_V)V \\
\frac{dU}{dt} &= \epsilon E P_U - (d_U + \gamma_U)U \\
\frac{dR}{dt} &= \gamma_U U + \gamma_V V - \xi R \\
\frac{dD}{dt} &= d_U U + d_V V
\end{aligned} \tag{1}$$

Mathematical Analysis

Existence of equilibrium points. From Eq 1 in the model explanation section, we select the equations that are independent of the others.

$$\begin{aligned}
\frac{dS}{dt} &= -\beta S(I-V) + \xi R \\
&= -\beta SU + \xi R \\
&= -\beta S P_u I + \xi R \\
\frac{dE}{dt} &= \beta S(I-V) - \epsilon E \\
&= \beta SU - \epsilon E \\
&= \beta S P_u I - \epsilon E \\
\frac{dI}{dt} &= \epsilon E - (d_v + \gamma_v)V - (d_u + \gamma_u)U \\
&= \epsilon E - (d_v + \gamma_v)P_v I - (d_u + \gamma_u)P_u I \\
\frac{dR}{dt} &= \gamma_u U + \gamma_v V - \xi R \\
&= \gamma_u P_u I + \gamma_v P_v I - \xi R
\end{aligned}$$

In order to determine the existence of equilibrium point, we first need to set all four equations to zero. Notice, the infected population either report or withhold their medical conditions, i.e. $P_u + P_v = 1$. Thus, we have

$$-\beta S P_u I + \xi R = 0 \quad \text{[i]}$$
$$\beta S P_u I - \epsilon E = 0 \quad \text{[ii]}$$
$$\epsilon E - (d_v + \gamma_v) P_v I - (d_u + \gamma_u) P_u I = 0 \quad \text{[iii]}$$
$$\gamma_u P_u I + \gamma_v P_v I - \xi R = 0 \quad \text{[iv]}$$

As we can see from (i) and (ii), $\beta S P_u I = \xi R = \epsilon E$. Plugging this into (iii) gives us

$$\beta S P_u I = (d_v + \gamma_v) P_v I + (d_u + \gamma_u) P_u I$$

The trivial solution would be $S = 0$ and $I = 0$, a disease-free state, which, however, is not where our interests lay. Rearranging it gives us

$$S = \frac{(d_v + \gamma_V) P_v + (d_u + \gamma_u) P_u}{\beta P_u}$$

Thus, we have one equilibrium point worth discussing

$$(S*, I*) = (\frac{(d_v + \gamma_v) P_v + (d_u + \gamma_u) P_u}{\beta P_u}, 0)$$

Stability of equilibrium points. In order to determine the stability of our equilibrium point, let's first calculate the Jacobian Matrix.

$$\begin{aligned} J &= \begin{pmatrix} \frac{\partial \dot{S}}{\partial S} & \frac{\partial \dot{S}}{\partial E} & \frac{\partial \dot{S}}{\partial I} & \frac{\partial \dot{S}}{\partial R} \\ \frac{\partial \dot{E}}{\partial S} & \frac{\partial \dot{E}}{\partial E} & \frac{\partial \dot{E}}{\partial I} & \frac{\partial \dot{E}}{\partial R} \\ \frac{\partial \dot{I}}{\partial S} & \frac{\partial \dot{I}}{\partial E} & \frac{\partial \dot{I}}{\partial I} & \frac{\partial \dot{I}}{\partial R} \\ \frac{\partial \dot{R}}{\partial S} & \frac{\partial \dot{R}}{\partial E} & \frac{\partial \dot{R}}{\partial I} & \frac{\partial \dot{R}}{\partial R} \end{pmatrix}_{(S*, I*)} \\ &= \begin{pmatrix} -\beta P_u I & 0 & -\beta P_u S & \xi \\ \beta P_u I & -\epsilon & \beta P_u S & 0 \\ 0 & \epsilon & -(d_v + \gamma_v) P_v - (d_u + \gamma_u) P_u & 0 \\ 0 & 0 & \gamma_u P_u + \gamma_v P_v & -\xi \end{pmatrix}_{(\frac{(d_v + \gamma_v) P_v + (d_U + \gamma_U) P_u}{\beta P_u}, 0)} \\ &= \begin{pmatrix} 0 & 0 & -(d_v + \gamma_V) P_v + (d_u + \gamma_U) P_u & \xi \\ 0 & -\epsilon & (d_v + \gamma_v) P_v + (d_U + \gamma_u) P_u & 0 \\ 0 & \epsilon & -(d_v + \gamma_v) P_v - (d_u + \gamma_u) P_u & 0 \\ 0 & 0 & \gamma_u P_u + \gamma_v P_v & -\xi \end{pmatrix} \end{aligned}$$

Now, we find the eigenvalues.

$$\begin{aligned} det(J - \lambda I) &= det \begin{pmatrix} -\lambda & 0 & -(d_v + \gamma_v) P_v + (d_u + \gamma_u) P_u & \xi \\ 0 & -\epsilon - \lambda & (d_v + \gamma_v) P_v + (d_u + \gamma_u) P_u & 0 \\ 0 & \epsilon & -(d_v + \gamma_v) P_v - (d_u + \gamma_u) P_u - \lambda & 0 \\ 0 & 0 & \gamma_u P_u + \gamma_v P_v & -\xi - \lambda \end{pmatrix} \\ &= 0 \end{aligned}$$

We get

$$\begin{aligned} \lambda_1 &= 0 \\ \lambda_2 &= -\xi \\ \lambda_3 &= -\epsilon - (d_v + \gamma_v) P_v - (d_u + \gamma_u) P_u \end{aligned}$$

Since $\xi, \epsilon, d_v, d_u, \gamma_v, \gamma_u, P_v, P_u > 0$, we have non-positive λ_1, λ_2, and λ_3. Therefore, we have a stable equilibrium point at $(\frac{(d_v + \gamma_v) P_v + (d_u + \gamma_u) P_u}{\beta P_u}, 0)$.

Numerical Simulations

In this section, we calculated the approximate numerical solutions of the model equations 1 for different parameters and initial populations using the SciPy library for Python. Since we do not have COVID-19 related data available, we will test our model with self-derived initial conditions listed in table 1.

Symbols	Definitions	Initial values
S(0)	Initial susceptible individuals	10^6
E(0)	Initial exposed individuals	0
I(0)	Initial infected individuals	5
U(0)	Initial reported infected individuals	4
V(0)	Initial unreported infected individuals	1
R(0)	Initial recovered individuals	0
D(0)	Initial dead individuals	0
β	Transmission rate between S and E	0.000003
ϵ	Transmission rate between E and I	0.05
P_V	Transmission rate between E and V	0.5
P_U	Transmission rate between E and U	0.5
d_V	Transmission rate between V and D	0.05
d_U	Transmission rate between U and D	0.08
γ_V	Transmission rate between V and R	0.1
γ_U	Transmission rate between U and R	0.05
ξ	Transmission rate between R and S	0.2

Table 1

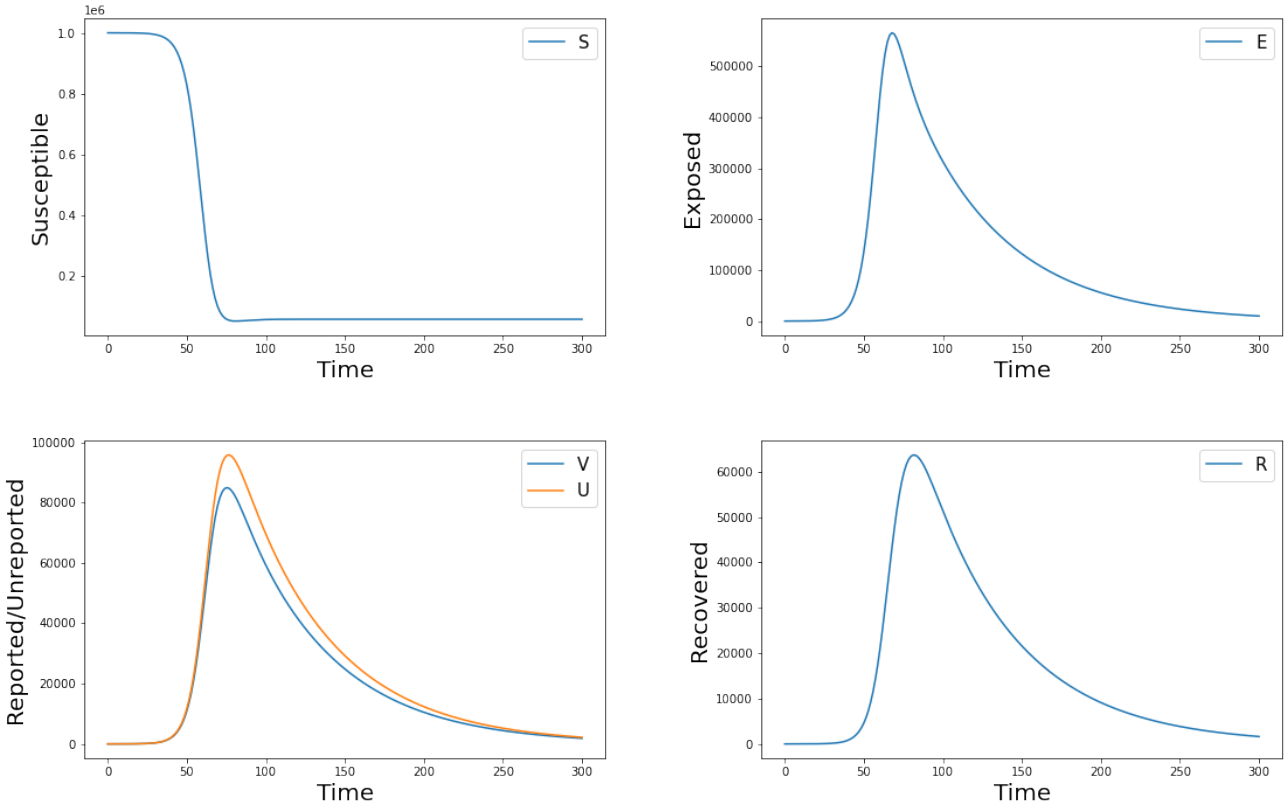

Fig. 2. Computational simulations for the dynamics of the model Eq. (1) using Python; there are model dynamics of: susceptible individuals, exposed individuals, reported/unreported symptomatic infected individuals, and recovered individuals.

Fig.2 shows the model dynamics of susceptible, infected, reported&unreported, and recovered individuals. The number of susceptible populations decreases gradually and becomes stable after 80 days while the dynamics of exposed and recovered people increase and get flat again after 300 days. The reported and unreported populations share a

similar pattern but have different curves due to their different populational death and recovery rates. According to Fig 2, our system will eventually reach equilibrium. This observation matches the discovery of the stable equilibrium point in the mathematical analysis section. This result suggests that our model is a reasonable approach to reality.

Another goal of this section is to study the dependency between the solutions and specific parameters. In this case, we will focus on the expose rate and the report rate. See fig.3.

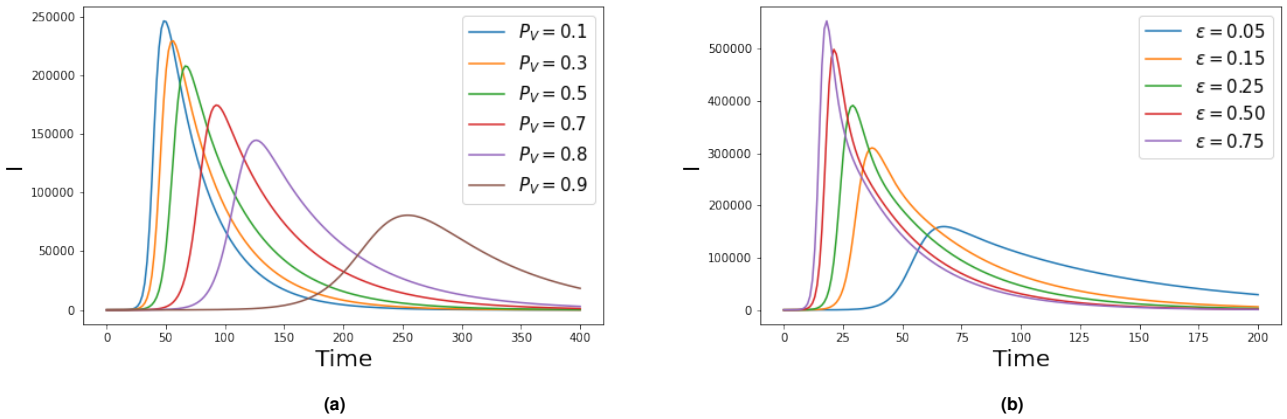

Fig. 3. The effect of transition rate ϵ and reported proportion rate P_V on infected individuals in computational simulations using Python. Parameters used ϵ = 0.05, 0.15, 0.25, 0.50, 0.75, and P_V = 0.2, 0.3, 0.5, 0.7, 0.9.

Figure 3a shows that the impact of the report rate P_V on the infected population. As we can see in the figure, a higher report rate will result in a generally smaller epidemic peak but will take a longer time to reach the system's equilibrium. Although this might sound counterintuitive, we can interpret it this way: the higher report rate limits the disease's spread, but the effect is not strong enough to suppress the spread and force it to die out.

Figure 3b explains that the impact of the transition rate ϵ on the infected population.As the figure suggests, a lower transition rate ϵ will result in a generally smaller epidemic peak. However, it will also take a longer time for the system to reach equilibrium, which may not be a desirable property. This is reasonable since ϵ is a crucial rate of determining the infected population.

Conclusion

In our model, we found one non-trivial stable equilibrium point, which signals that Covid-19 will die out on its own eventually. However, the spreading of Covid-19 cannot be well-understood by mathematical models since other unpredictable factors also influence the pandemic. Namely, the mutation of coronavirus has yet to be thoroughly studied and government policy on mandatory quarantine. How people will react to this kind of policy also varies from country to country.

We used Python to conduct simulations using different parameters and initial conditions, which helped us identify the crucial variables that impact how the coronavirus plays out. We mainly focused on the exposed rate ϵ and the report rate of the infected population P_v, which are our key parameters different from other models. It makes sense that a lower exposed rate and high report rate both result in a smaller epidemic peak. It can be strongly suggested that people should stay at home to cut off the transmission of COVID-19. People should get Covid tests and report their symptoms as early as possible to receive corresponding treatments and quarantines.

Future improvements in our model could include coronavirus mutation, medical supplies, vaccination, and government intervention with the public reaction. More research and data are needed for a more accurate COVID-19 model and a more effective way to control the disease.

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