

Epidemic Spread Models

Mathematische Modellierung

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1. Introduction

Since 2020, the topic that comes up and down in the media every day is related to Covid-19. Thanks to this, people become familiar with various technical terms, one of which is 'mathematical modeling'. This term was frequently mentioned when scholars analyzed and predicted the spread of Covid-19. Mathematical modeling is the mathematical description of the operating principle of a phenomenon or system in various fields such as nature, society, and technology using the language of mathematics. The history of mathematical modeling dates back to the 16th and 17th centuries. As mathematics, physics, and astronomy developed, scholars such as Johannes Kepler in Germany and Isaac Newton in England began to use mathematics to explain and predict natural phenomena, especially the motions of celestial bodies.

In the 18th century, about 80 years after Newton mathematically explained the movement of celestial bodies, mathematical modeling was introduced to the study of infectious diseases. Daniel Bernoulli, a Swiss mathematician and physicist, mathematically modeled the spread of smallpox. It can be said that the study of infectious diseases through mathematical modeling began in earnest in the 20th century. British pathologist Ronald Ross discovered the malaria parasite in the salivary glands of mosquitoes in 1893 and mathematically revealed the transmission route of malaria. In 1972, Scottish mathematician William, Culmick and epidemiologist Anderson McKendrick presented a unique mathematical model for explaining and predicting the spread of infectious diseases, the SIR model. This model is still used as a basis for studying the spread of various infectious diseases such as Covid-19, Ebola, and flu. In addition, many scholars study the transmission patterns of infectious diseases with various mathematical models derived from this model.

It is the role of epidemiologists and medical scientists to treat and prevent infectious diseases using various medical methods. However, it is necessary to model the spread of infectious diseases so that they can use timely methods. Just as the SIR model was created by mathematicians and epidemiologists together, it requires their collaboration. Various parameters required for modeling can be known from research by epidemiologists, and it is the role of mathematicians to complete modeling using these parameters.

In the project, mathematical modeling of epidemic spread is studied in two ways. The first is a model using a differential equation. Various models using differential equations are introduced, and parameter studies are conducted on the most basic

model. In addition, the complex model is constructed having similar shape with the graph of the actual graph. The second is a method using an agent-based model. We will introduce what is the agent-based model and how we apply and implement this model for epidemic spread using python. Various simulations with different conditions were executed and the parameter study is also conducted. Finally, the pros and cons of the two methods are compared.

2. Epidemic Spread Modelling with Differential Equation

2.1 Theory : Dynamic System and Differential Equation

To understand and control complex systems, quantitative mathematical models of these systems are essential. Therefore, it is necessary to interpret the relationship between system variables and obtain a mathematical model. In general, since dynamic systems are dealt with, differential equations are used to represent them. A dynamic system means usually a system that changes over time such as and the Lotka-Volterra scheme. The dynamic system only depends on the initial value and its states are connected to and depend on each other. In practice, the system becomes complex, and it is not possible to know all the related factors, so assumptions about the system's operation are necessary. After considering a physical system and stating the necessary assumptions, differential equations are obtained that represent the system. Approaches to the analysis of dynamic systems are listed as follows.

1. Define the system and its components.
2. Construct a mathematical model and list the necessary assumptions.
3. Find the differential equation describing the model.
4. Solve the equation for the desired output variable.
5. Review the equation solutions and assumptions.
6. If necessary, reinterpret or redesign the system

Since the spread of the epidemic to be modeled in the project is also a dynamic system over time, we use the differential equation and follow the approach above.

2.2 Method : Definition of the system and its components

1. Classes In the epidemic spread models with differential equations, the population is divided into different classes that describe the disease state of each individual.
 - N : Total population
This is the total population which is the sum of all other classes. It is mostly constant unless the research period is too long to be affected by birth, death and migration. In the project, it is assigned to Korea's total population of 51,740,000.
 - I : Infective or sick class

People in the class are infected and can pass the disease on to other people. For modeling, only the initial value of the class is needed and it is assigned to 1.

- S : Susceptible or healthy class

People in the class have not been contacted with the disease and are not immune yet. Since $S = N - I$, the initial value of the class is assigned to $51,740,000 - 1$.

- R : Removed or recovered class

People in the class are recovered and immune. People who are dead are also included in the class. They are no longer pass on the disease and contagious. However, there are some diseases that can still be transmitted after death. Depending on the disease, it should be taken into account. Its initial value is 0 since no one cannot have immune until they get a disease and recover.

- E : Exposed or latently infected class

People in the class are in the latent phase which means they are infected but they cannot yet pass on the disease. Since they don't have symptoms yet, it is hard to recognize the disease. Covid-19 is very difficult to record the people in this class because of an incubation period. Here, the incubation period and the latent period should be distinguished.

The incubation period is from the moment of infection with the virus

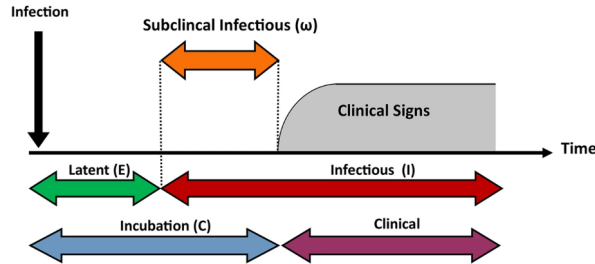


Figure 1: Latent and Incubation Period

until clinical symptoms appear. The latent period is from infection until contagious. Because of this complexity, it is difficult to record for the latent class. According to the Korean Centers for Disease Control and Prevention, the first confirmed case in Korea occurred on January 20, 2020, and the average latent period is 5 days. Therefore, 3 people who were contacted and confirmed within 5 days after the first confirmed person was set as the initial value for this class.

2. Transition Coefficients

- β : Contagion or infection rate

It is the rate leaving the infectable/susceptible class S. Its size changes depending on the size of the departure class and the arrival class. The value announced by the Korean Centers for Disease Control and Prevention was used for the project. The initial value is 0.2161.

- γ : Recovery rate

It is the rate leaving the infected class. The average time that a person stays in class I is $1/\gamma$. It is known that it takes an average of 15 days from the first infection to recovery, but it was a little longer than this in the early days of the Covid-19 pandemic, so it is assumed to be 20 days. Therefore, this rate is $1/20 = 0.05$.

- ε : Contagious rate

It is the rate at which infected people become contagious. The average period of the latency period is $1/\varepsilon$. It is used only when there is class E in the model. Since the latent period is known to be 5 days, this rate was set to $1/5 = 0.2$.

- ξ : Rate of loss of immunity

It is the rate at which people lose their immunization from class R to class S. In the early days of Covid-19 pandemic, we could not consider the rate of reinfection, because it did not receive attention. The recently reported reinfection rate is known to be 26.11% and the infective rate is 0.2161, so we set ξ to be $0.2161 \cdot 0.2611$.

2.3 Method : Listing the necessary assumptions

The most important assumption is that the population is considered isolated, which means that no migration from out of the system is allowed. Since the target period is short, it is assumed that there are no changes from birth and death. Therefore, the total population is assumed to be kept constant. People who die of natural causes do not change N because they cancel out people born, whereas people who die of this disease do not change N because they belong to class R. In addition, the population is considered to be homogeneous. It means each individual has the same characteristics in relation to the disease. Everyone has the same probability to contact other people, to get infected, and to transmit the disease.

2.4 Method : Construction of mathematical models and differential equations

1. SI Model

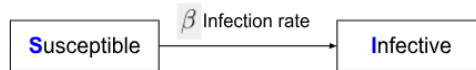


Figure 2: System of SI Model

It is the simplest of the epidemic spread models, with only transitions between infectable and infected classes. Once you have a disease, you are permanently infected. In this model, everyone will eventually get sick, which is not the case in reality. Since there is only movement of people leaving class S to class I, the rate of change of the two classes is the same magnitude but a different sign.

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

$$\frac{dI}{dt} = \frac{\beta}{N}SI$$

2. SIS Model

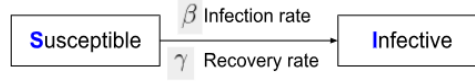


Figure 3: System of SIS Model

In the SIS model, unlike the SI model, once infected people are cured. However, since it is treated with no immunity, it is converted to an infectious class S again. The rate of recovery is also applied to the rate of change of the two classes with the same magnitude but different signs.

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

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

3. SIR Model

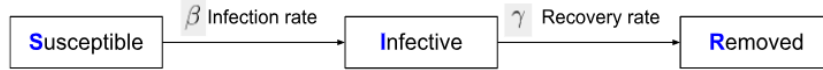


Figure 4: System of SIR Model

The SIR model is the most widely used model, and there are many studies on the spread of various infectious diseases using this model. Unlike the SIS model, the treated person has immunity and moves to class R. The change due to this recovery rate applies to class I and class R, so the magnitude is the same but the sign is different.

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

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

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

4. SIRS Model

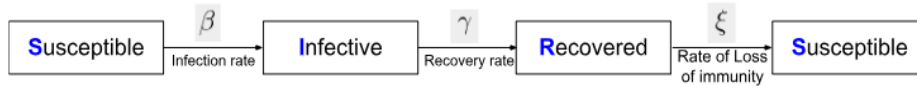


Figure 5: System of SIRS Model

In the SIRS model, after the same process as the SIR model, people in class R lose immunity someday and go back to class S again. The coefficient influencing this process is ξ which depends on the average duration. The rate of change is

related to ξ and the size of class R. It affects the rate of change of R and S, equal in magnitude and opposite in sign.

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\beta}{N}SI + \xi R \\ \frac{dI}{dt} &= \frac{\beta}{N}SI - \gamma I \\ \frac{dR}{dt} &= \gamma I - \xi R\end{aligned}$$

5. SEIS Model



Figure 6: System of SEIS Model

Unlike the previous models, the SEIS model has a latent period. When people in the healthy class S become infected with a disease, they are converted to the latent class E and stay for the latent period. The moment people show symptoms and become contagious, they switch to the infected class I. People in class E are difficult to be confirmed, recorded, and modeled. A transition from class S to E is equivalent to a transition from class S to I in the SIS model. The rate of change is the combination of the contagious rate ε and the size of class E and affects the change of class E and I.

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\beta}{N}SI + \gamma I \\ \frac{dE}{dt} &= \frac{\beta}{N}SI - \varepsilon E \\ \frac{dI}{dt} &= \varepsilon E - \gamma I\end{aligned}$$

6. SEIR Model



Figure 7: System of SEIR Model

Class E is added to the SIR model, and the rate of change of classes S, E, and I are defined as in the SEIS model. The difference is that recovered patients have immunity.

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\beta}{N}SI \\ \frac{dE}{dt} &= \frac{\beta}{N}SI - \varepsilon E \\ \frac{dI}{dt} &= \varepsilon E - \gamma I \\ \frac{dR}{dt} &= \gamma I\end{aligned}$$

2.5 Result : Solution of the differential equation

1. Implementation

The initial values of the total population, each class, and the transition coefficients are set the same for all models as mentioned. The transition coefficients are kept constant during the modeling period. By dividing the value of the initial state by the total population N , this calculation provides a relative value, which makes it easy to compare different models. Differential equations should be solved numerically. There are various methods such as the Euler method or the Runge-Kutta method, but in the project, built-in functions were used.

2. SI Model

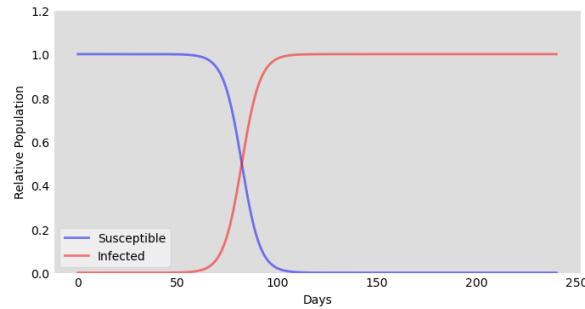


Figure 8: Relative Population in the Classes of SI Model

As healthy people become infected, the number of people in class S decreases and in class I increases. Since one day everyone will be infected, S will be 0, and I will be the entire population. Therefore, no peak (local maximum point) of I is not observed.

3. SIS Model

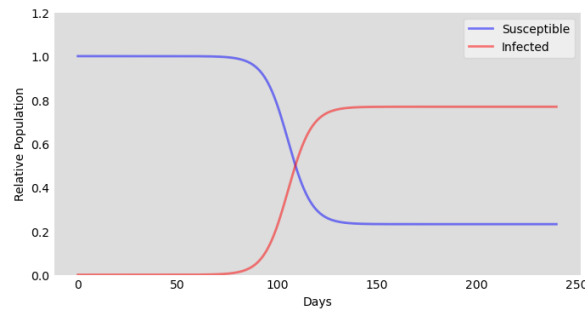


Figure 9: Relative Population in the Classes of SIS Model

Since the infected move from class I back to class S as they heal, S and I do not converge to 1 and 0. When I is slightly less than 1 and S is slightly greater than 0, the stationary state is reached. Therefore, no peak (local maximum point) of I is not observed.

4. SIR Model

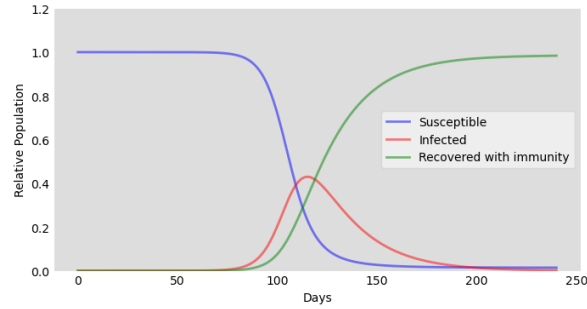


Figure 10: Relative Population in the Classes of SIR Model

Since this model also progresses in one direction, according to the coefficients we set, the first class S decreases to 0 and the last class R increases to 1. The middle class I increases to the maximum point and then decreases again to 0. In this model, everyone is immune and in the stationary state, the entire population is designed to stay in class R .

5. SIRS Model

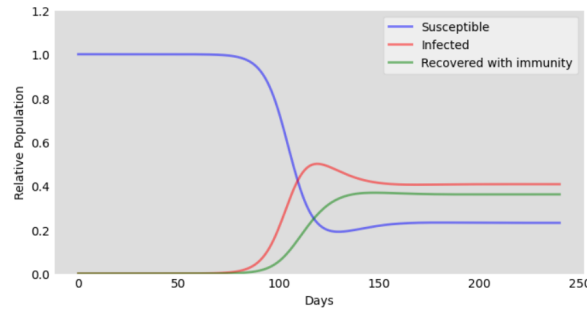


Figure 11: Relative Population in the Classes of SIRS Model

The shape of the graph is very different between the SIRS model and the SIR model, although the difference is only whether the immunity acquired by the recovered people is permanently retained or lost someday. In SIRS, people in class R can lose immunity, be converted back to class S , and become infected again. Class S does not converge to 0. After class I reaches the peak, it decreases slower than the SIR model to a value bigger than 0. Although the SIR model is the most used in many studies, we conclude that the SIRS model is most suitable for Covid-19. This is because even 3 years after the outbreak, there are still many reinfected cases.

6. SEIS Model

SEIS graphs are similar to SIS graphs in that classes S and I , having stationary states at somewhere between 0 and 1. As people in class I recover without immunity to class S , there is no permanent recovery. Therefore class I doesn't have the peak. Because of class E , the speed at which the classes change is slow, so it takes to reach the stationary state is slow, and the difference between the two classes is less.

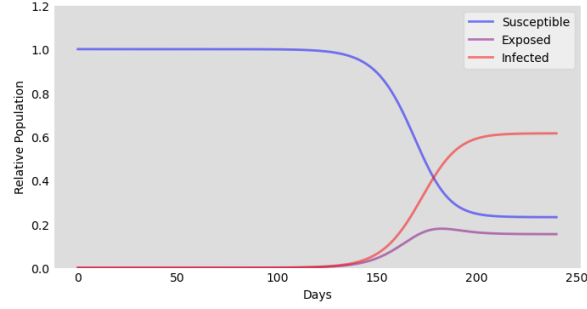


Figure 12: Relative Population in the Classes of SEIS Model

7. SEIR Model

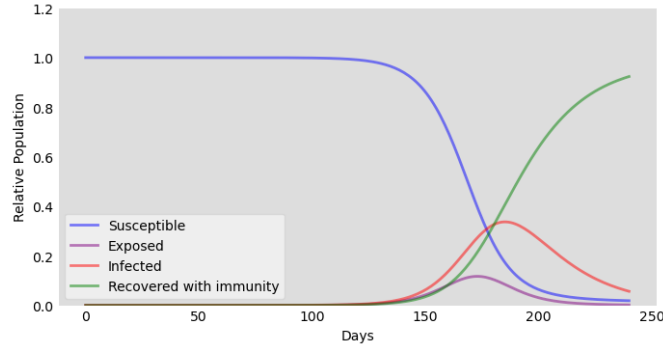


Figure 13: Relative Population in the Classes of SEIR Model

The SEIR model is the class I of the SIR model divided into two classes E and I. The stationary states of S and I are the same as SIR. Because of the latency period, the maximum of class I is reached later than the SIR. Since COVID-19 also has an incubation period, SEIS or SEIR models may be more appropriate. However, it is difficult to calculate accurately because people with no symptoms during the incubation period are hard to be recorded. Therefore, we thought that a model with a latent period was difficult to provide reliable modeling in this project.

2.6 Discussion : Review the solutions and assumptions

1. Reproduction Index and SIR Model

The primary purpose of modeling the spread of Covid-19 is to develop a response based on the prediction. For this purpose, the reproduction index is more important than the absolute number of infected people.

$$\mathcal{R}_0 = \frac{\beta}{\gamma}$$

Since this is a combination of transition coefficients β and γ of the SIR model, it is the most widely used in many studies. We also conducted a parameter study through the SIR model. The reproduction index with coefficients we used

in the models above is $\mathcal{R}_0 = \frac{\beta N}{\gamma} = \frac{0.2161}{0.05} = 4.322$. This number is pretty high, and the goal of the Korea Centers for Disease Control and Prevention is to keep this index below 1.

2. Parameter study of SIR model

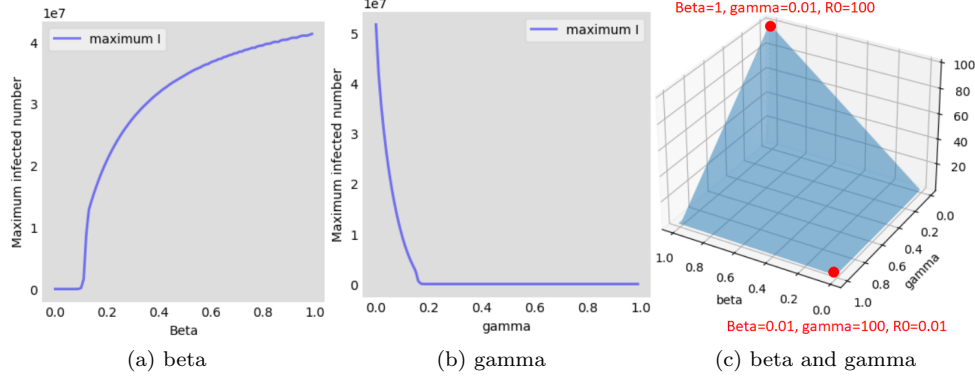


Figure 14: Maximum number of infected people (I) depending on parameters

As can be inferred from the formula of the reproduction index \mathcal{R}_0 , the smaller the infection rate β and the larger the recovery rate γ , the smaller the reproduction index \mathcal{R}_0 . However, γ is usually a characteristic of the disease and difficult to control. Since β can be reduced through policies such as lockdown and social distancing, SIR modeling is attempted with various β values.

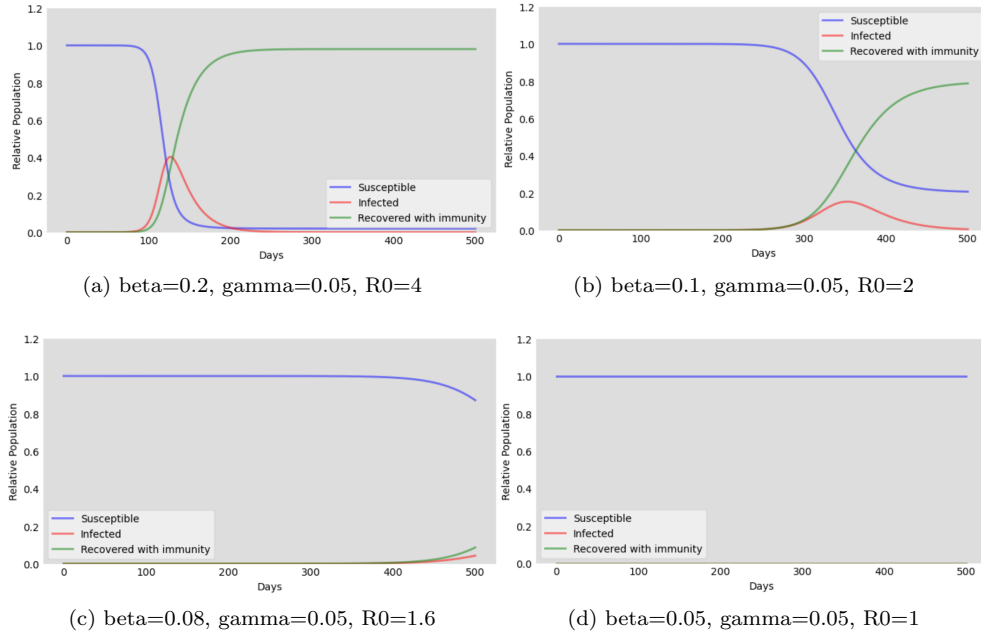


Figure 15: Model with various beta for fixed gamma=0.05

For the same recovery rate $\gamma = 0.05$, as the infection rate β decreases, that

is, as the reproduction index \mathcal{R}_0 decreases, the rate of change of classes slows down. The maxima of infected class I also appear later, and the maxima are even lower. When \mathcal{R}_0 close to 1 which means that β and γ values are almost the same, Class I doesn't increase, so it can be said that Covid-19 has ended.

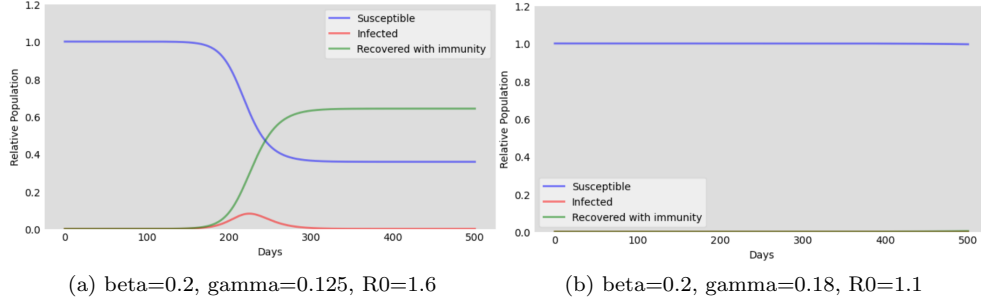


Figure 16: Model with various gamma for fixed $\beta=0.2$

For a disease with a high infection rate β , it is possible to increase the recovery rate γ if a good treatment is developed and the duration of infection is reduced. In order to compare with the graph in Fig.15(a) with $\beta = 2$ and $\gamma = 0.05$, β is maintained at 0.2 and γ is increased. As γ increases, the maximum value of infected class I gets lower and appears later. Both Fig.15(c) and Fig.16(a) show the graphs with $\mathcal{R}_0 = 1.6$, with smaller β and with larger γ respectively. The speed of change is faster with larger γ . When \mathcal{R}_0 becomes almost 1, Fig.16(b) can be considered to have ended at this time as Fig.15(d).

3. Review of Assumption

The assumptions of modeling are that there is no population change due to births, deaths, or immigration, and that all populations have homogeneous characteristics for Covid-19. However, the total population changes from time to time, but we do not reflect this and model the total population as constant. Above all, the reason why our models show a large error from the actual number of infected people is that people are not homogeneous to the infection of Covid-19. First of all, each person is inherently different in how vulnerable they are to COVID-19. Even if a confirmed case occurs in the same group, there are people who do not get it. In addition, external events dramatically increase or decrease the probability of contact with confirmed cases. This caused β to change time to time, which was not reflected in our model. It is too complicated to include changes in the total population in the modeling, but we can redesign the complex model to reflect the changing β .

2.7 Discussion : Redesign of the system

1. change in β

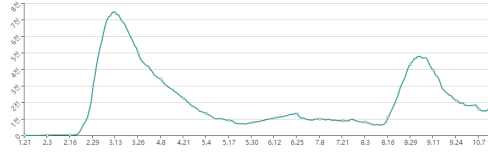
In Korea, in the first year of the corona outbreak, the number of confirmed cases did not exceed 10,000, and at this time, the human traffic of all confirmed cases was disclosed and controlled. Although it was relatively easy to manage compared to other countries, the number of confirmed cases dramatically jumped up and down due to several major incidents. For example, during the period when religious gatherings were banned, there were two incidents that large numbers of people gathered at heretical or Christian churches. There was also an incident

that a confirmed person visited a party at a large club and many people were infected. As a follow-up measure, social distancing was strengthened and the infected decreased again.

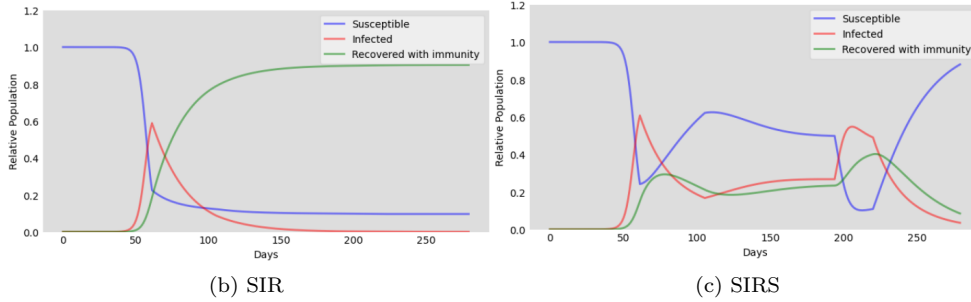
Time (Days)	Type	Incident	beta
[0,30]		First infected	0.3
[30,63]	Mass infection	Heresy's gathering	0.4
[63,108]	Social Distancing	Stage 1	0.05
[108,197]	Mass infection	Clubbing	0.1
[1967, 224]	Mass infection	Church service	0.4
[224, 284]	Social Distancing	Stage 2.5	0.01

Table 1: Value of Beta in the incidents

2. Redesign of Complex SIR and SIRS model



(a) Actual number of confirmed cases in Korea



(b) SIR

(c) SIRS

Figure 17: Complex model with various beta

As a result of modeling by changing β over time as in the table above, the SIR model is very different from the graph of the actual number of confirmed cases. In the actual graph, the number of confirmed cases increases and decreases, but the SIR graph has only one peak. We find that the SIR model is not the best because the recovered people have permanent immunity and there are not enough infectable people when β gets higher. Since the recovered people cannot be re-confirmed, the size of class I keeps decreasing after reaching the peak. We choose the SIRS model as the recovered people can be infectable again. It is better suited to reflect this changing β . If we look at the red line (infected class I) of the complex SIRS model graph, it is quite similar to the graph of the actual number of infected people. In this complex SIRS model, there are enough people who can be infected when β increases due to a specific event because of people from class R to S. Therefore, we conclude that the SIRS model is adequate to represent COVID-19 if the transition coefficients can be obtained more accurately.

3. Epidemic Spread Modelling with Agent-Based Model

3.1 Theory : Agent-Based Models

Let's start with the definition of the agent-based model(ABM). Agent-based models are computational simulation models that involve many discrete agents. Unlike the models using differential equations above, ABMs concentrate on the individual agent. We can set the internal states of agents, how the agents interact each other and even how the agents interact with environment. The following is typical properties generally assumed in agents and ABMs:

1. Agents are discrete entities.
2. Agents may have internal states.
3. Agents may be spatially localized.
4. Agents may perceive and interact with the environment.
5. Agents may behave based on predefined rules.
6. Agents may be able to learn and adapt.
7. Agents may interact with other agents.
8. ABMs often lack central supervisors/controllers.
9. ABMs often produce nontrivial "collective behavior" as a whole.

3.2 Implementation of ABM for epidemic spread model(COVID-19) using python

We are going to concentrate on the property 1, 2, 5 and 7. Python is a object-oriented language, so it is right choice to use python to implement ABM(property 1). We created a class 'Agent' and created as many objects as we want using this class 'Agent'. Each agent(in this case person) has internal states for epidemic spread model(property2): 'biostate', 'nextbiostate', 'biostatecountdown'. 'biostate' tells us which state is the agent in. We have total 7 states:

1. Unexposed
2. Asymptomatic but infected/contagious
3. Symptomatic and contagious
4. Symptomatic and not contagious
5. Post-COVID Immune
6. Naturally immune
7. Death

You can see the relationship among these states in the Figure 18. The other internal states play a role to change the 'biostate' of a agent over time and 'biostate' changes over time by the rule of COVID-19(Property 5). We implemented this rule of COVID-19 in the class 'Agent' as static variables, so that all the objects of this class can follow this rule. For example, there are static variables such as 'bioMin', 'bioMax' and 'bioTransition'. 'bioMin' indicates the minimal time of each state, 'bioMax' indicates the maximal time of each state, and 'bioTransition' is a matrix which indicates the probabilities to transfer from a state(row) to another state(column).

	state 1	state 2	state 3	state 4	state 5	state 6	state 7
bioMin	1	3	3	1	1	1	1
bioMax	1	10	8	7	1	1	1

Table 2: 'bioMin' and 'bioMax'

	state 1	state 2	state 3	state 4	state 5	state 6	state 7
state 1	0	0	0	0	0	0	0
state 2	0	0	0.5	0	0.5	0	0
state 3	0	0	0	0.95	0	0	0.05
state 4	0	0	0	0	1	0	0
state 5	0	0	0	0	0	0	0
state 6	0	0	0	0	0	0	0
state 7	0	0	0	0	0	0	0

Table 3: 'bioTransition'

When we look at the table 3: 'bioTransition', we can figure it out that the probability of transition from state 3 to state 4 is 0.95 and the probability of transition from state 3 to state 7 is 0.05(1-0.95). The first row of this matrix is all 0. It may look like it does not make sense, but we implemented this way, because the values of this row does not impact on the transition. State 1 can only transfer to state 2 and we use a variable 'contagionProb' directly, which is probability of contagion, instead of this matrix 'bioTransition'. We also implemented functions that make the agents interact each other. For example, when an agent with the state 'symptomatic and contagious' meet an agent with the state 'unexposed', the state of the latter agent could change to 'asymptomatic but infected/contagious'.

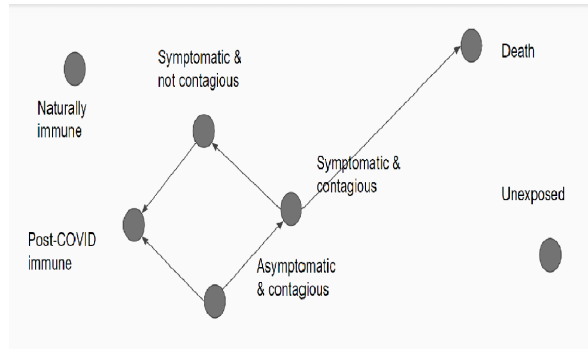


Figure 18: The network of the biological states

3.3 Simulation with basic social model

We are going to execute various simulations with various conditions. In the first simulation, we assume that the people(agents) meet some randomly chosen people and we call it as basic social model. The following is the parameters for this simulation:

- numAgents = 500 : int, the number of agents
- naturalImmunity = 0.01 : float, the proportion of naturally immuned people
- numInteractions = 10 : int, how many interactions per day per agent on average
- numDays = 50 : int, the number of days
- contagionProb = 0.1 : float, normal contagion probability
- numInfected = 3 : int, the number of infected at the starting point

We executed this simulation 100 times and the following(Figure 19) is the result using the mean values:

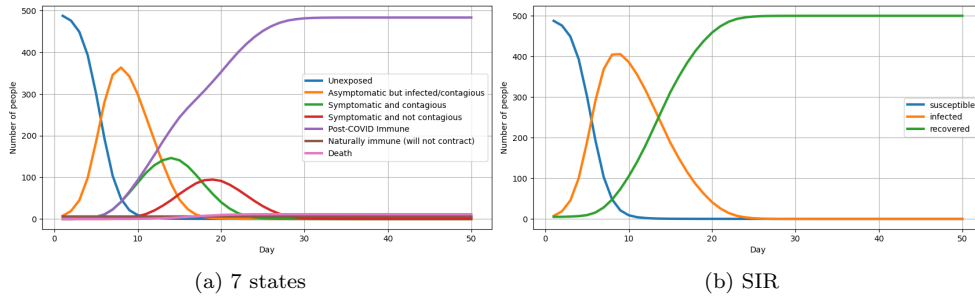


Figure 19: Result of the simulations with basic social model

The graphs of Figure 19 look fine. The shape of the SIR graph in Figure 19 is as same as the known shape of the SIR graph.

3.4 Simulation with realistic social network

The basic social model, which we just used, is not realistic, because people have their own social network and usually meet someone in that social network. So we implemented the Erdos-Renyi model on a social network, which is more realistic than the basic social model, and we executed simulations with this social network. All parameters, which are used for the simulation with basic social model, are also used here with same values and there are two more parameters:

- socialNetwork = social network created by Erdos-Renyi model
- sampleFromNetwork = 0.98: float, how much this social network effects on whom an agent meet

For example, when the parameter 'sampleFromNetwork' is 0.90, it means that the probability of a person meets another person, who is in his/her social network, is 0.90 and the probability of the person meets another person, who is randomly chosen, is 0.10. We executed this simulation 5 times, which is a lot less than last time, because this simulation takes too long. You can see the result in Figure 20.

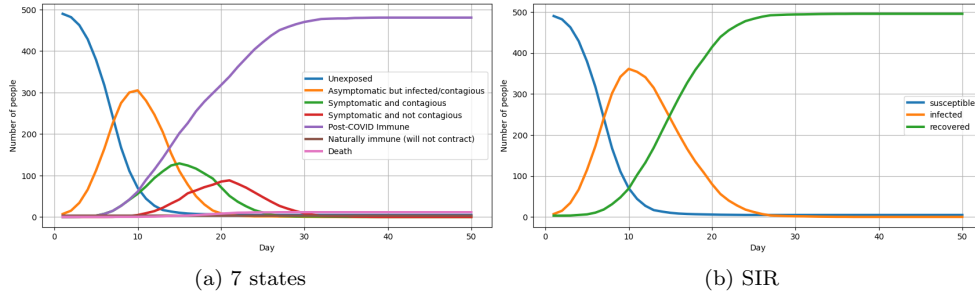


Figure 20: Result of the simulations with realistic social network

When we compare the yellow graphs of (a) in Figure 20 and in Figure 19, the peak of the yellow graph in Figure 20 is lower and the x value of the peak of the yellow graph in Figure 20 is larger. When we compare the yellow graphs of (b) in Figure 20 and in Figure 19, we can get a similar information. Therefore we can conclude that the speed of spread of COVID-19 in the simulation with realistic social network is slower than the speed of spread of COVID-19 in the simulation with basic social model.

3.5 Simulation with realistic social network and quarantine

On the top of the simulation with realistic social network(3.4) we added quarantine, so that the people who have symptoms cannot meet other people. All parameters, which are used for the simulation with realistic social network(3.4), are used here with same values. We executed this simulation also 5 times because of the same reason. The result is following:

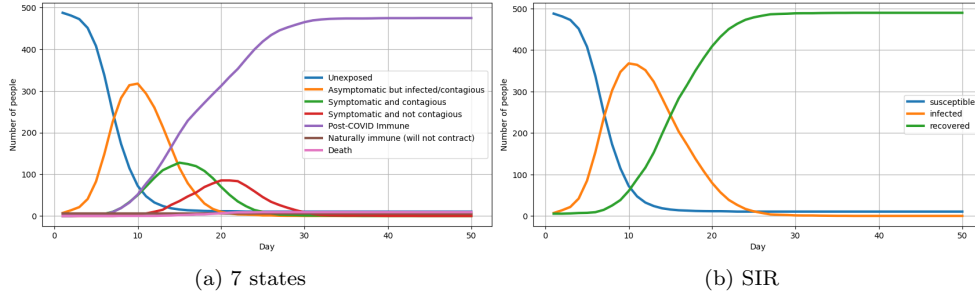


Figure 21: Result of the simulations with realistic social network and quarantine

When we compare the graphs of Figure 20 and Figure 21, there is no significant difference. That is an interesting result, because it means that the quarantine does not work as much as we expect. I could have not found any error of my code yet. Maybe we can think about it deeper later.

3.6 Simulation with basic social model and regulation by government - Parameter study

We wanted to add a regulation by government, so that People cannot meet other people in a group, which consists of more than 4 people. To implement this regu-

lation easier we made an assumption that each person meets 3 people a day after the regulation is enforced. To sum up, each person meets 10 people a day before the regulation is enforced and 3 people a day after the regulation is enforced. We decided to use basic social model here instead of realistic social network, because simulation with the realistic social network takes too much time. We also decided not to apply quarantine here to see the effect of the regulation well.

We were curious about how important it is that the government enforces the regulation early. So we conducted a parameter study. Of course the parameter is when the government enforces the regulation after people are infected. We chose 15,12,9,8,7,6 and 3 as the value of this parameter. For example, the value 15 of this parameter means that the government enforces the regulation 15 days after people are infected. We executed 100 simulations on each parameter value. The result is following:

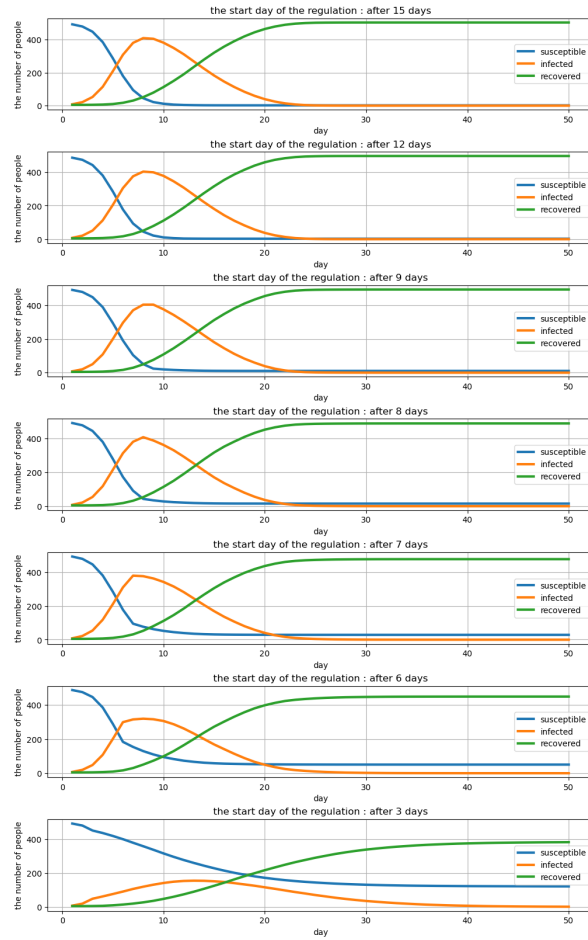


Figure 22: Simulation with basic social model and regulation by government

When we see the last 2 graphs(values of parameter: 6, 3), we can easily find that the regulation works. Especially when the value of the parameter is 3, the effect of the regulation is huge. When we compare the first 5 graphs(values of parameter: 15, 12, 9, 8, 7) to the graph of (b) in Figure 19, there is no significant difference. It means that the regulation does not work well, when the government starts to enforce the regulation after 7 days. It is interesting that the regulation works really well, when

the value of parameter is 6. We thought it is because the regulation is enforced before the number of infected people reaches a peak. That is the reason why one day earlier makes a big difference. It is of course hard to say that one day earlier makes a big difference in the real world, because only 500 agents are created in these simulations. But we can at least say that the government needs to enforce the regulation before the number of infected people reaches a peak to make the spread of COVID-19 slow down. We can also conclude that the earlier the regulation is enforced before the peak, the dramatically slower COVID-19 spreads.

4. Discussion and Conclusion

Among the 6 models using differential equations, the model we selected is a complex SIRS model with various β . Of 4 classes, the latent class E and its coefficient ε are hard to note in magnitude. In addition, Covid 19 is re-infectable losing immunity, so the last transition must be from R to S. Even for the same model, it is found that the model changed when the transition coefficient are changed. As long as we can get reliable initial values and transition coefficients, our complex SIRS model is quite accurate and responsive.

The advantage of models using differential equations is that, as a dynamic system, we can see the big picture over time. It also includes the most important factors, infection and recovery rate. However, there is a disadvantage in that it cannot include various factors other than the infection rate and recovery rate.

In contrast to models using differential equations, ABMs are focusing on each agent. There are some rules in a agent-based model and agents live, communicate each other and interact with environment by those rules. Those rules and the property of agents can be implemented as you want. ABMs are so open-ended and flexible that ABMs are nice playgrounds to try testing scenarios. This trait is a great advantage of ABMs, but it can also lead us to a bad direction. ABMs are so flexible that we can add too many details, which makes it difficult to analyze the result.

It is also possible that the user of ABMs concludes that a scenario is right, even though it is not. For example, the user can get the same result as the result of the real world while testing a false scenario and it can confuse the user. If a user have a deep knowledge about the system he/she created, the user can make good scenarios and then the probability that the user conclude the wrong scenario is right would be much lower. Therefore the deep knowledge of the system is very helpful for the users of ABMs.

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6. Appendix

1. Projektarbeit_Epidemic_Spread_Model_DGL_Soul_Suh.py : Submit attachment as file
2. Projektarbeit_Epidemic_Spread_Model_ABM_Soul_Suh.ipynb : Submit attachment as file
3. abm.py : Submit attachment as file