Introduction to Computational Science
Assignment 1: SIR model (ODE)

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Abstract: This paper mainly focuses on the SIR model. By numerically integrating the theoretical equations, it gets the primary dynamic trend of spreading disease, followed by a phase space diagram analysis. And then, it fits a list of actual epidemic data and a vaccination plan into the model as evaluation. For a deeper exploration, demography and mortality have been considered, respectively, with additional Fourier analysis. In the rest part, it attempts to analyze the SEIR model similarly.

1 INTRODUCTION

These days, coronavirus disease 2019 (COVID-19) crisis on the labour market and social network has become a global issue, which has a huge impact on employment in Europe even world (Weber et al., 2021). So, it is critical to understand or predict the epidemic. It draws people attention to the interplay of various factors such as infectivity, recovery, and mortality.

To investigate the transmission and rationale, the SIR model (susceptible, infected and recovered) has re-emerged as a powerful tool for epidemiology research (Chowdhury et al., 2021). It was first introduced to find the solution of epidemic termination (Kermack and McKendrick, 1927). This model divides the epidemic process into three compartments: susceptible people, infected people who can spread disease, and safe people with immunity.

This paper expresses these factors as serious differential equations that can be numerically integrated into quantitive values. Additionally, a case of an influenza outbreak has been introduced to fit the model. Other factors like birth and death rate and mortality induced by illness will be added to the basic SIR model, followed by phase space and Fourier analysis.

Because in the objective case for some diseases, susceptible people will not become infected instantly before they enter into an exposed compartment. For example, brucellosis, HIV/AIDS, and leishmaniasis usually have long incubation periods, which cause a prolongation of clinical manifestations (WHO). In such a case, the SEIR model is devoted to addressing the additional factor, combined with the seasoning rate of infection. Due to practical constraints, this paper cannot comprehensively review other different models like MSIR, SEIS, MSEIR, MSEIRS etc.

2 METHODOLOGY

This paper uses the mathematical method mainly based on **Infectious Diseases in Humans and Animals** (Keeling and Rohani, 2008) to implement the theoretical model and utilize Python 3.6 to plot the results as 2D and 3D curve graphs. At the start, it needs to build a basic SIR model as a benchmark. Traditionally, it has been assumed that the small proportion of infected people within a large susceptible group initially. The susceptible people get the disease during a relatively short period. All infected people will get recovered at last and keep immunity forever.

A total population is a constant number: N. The product of the contact rates and transmission. Probability is defined as β. It also assumes a homogeneous mixing situation without a complex contact pattern. So βSI can be regarded as the transmission term, which represents the rate where new infectious individuals generate. Similarly, γ has been defined as the recovery rate (the inverse of the infectious period). The transmission from infected to recovered is equal to γI. Overall, Ordinary Differential Equations (ODE)

are listed as follows:

$$\frac{dS}{dt} = -\beta SI \tag{1}$$

$$\frac{dI}{dt} = \beta SI - \gamma I \tag{2}$$

$$\frac{dR}{dt} = \gamma I \tag{3}$$

For analysis, two useful values are extracted from those equations. One is γ/β, the relative removal rate. Another important product is called the basic reproductive ratio (R_0),

$$R_0 = \frac{\beta}{\gamma} \tag{4}$$

which can be understood as new second generation from the first infected generation in a wholly susceptible population.

2.1 Threshold Phenomenon

It should be noticed that not every disease can develop as an epidemic, which depends on if $\frac{dI}{dt} > 0$ or not. Two situations are listed in following:
Situation 1: When $\beta SI - \gamma I > 0$, it means the number of infected people will increase. That equls to S $> 1/R_0$. So a disease will only spread when the initial fraction of susceptible (S_0) is greater than $1/R_0$.
Situation 2: Additionally, if $S_0 = 1$, then the R_0 should be greater than 1 for epidemic happening.

2.2 Data fitting

To simulate the real disease spreading, it fits the actual infected data in a boy school from Table 1. This function is finished by python package: **scipy curve-fit**, which bases on a non-linear least-squares method. The original SIR differential equations attempt to optimize the constants β and γ until getting the minimized deviation with real cases.

Day	1	2	3	4	5	6	7
Nmber	3	8	28	75	221	291	255
Day	8	9	10	11	12	13	14
Number	235	190	125	70	28	12	5

Table 1: School influenza outbreak

After acquiring β and γ, the rest steps are merely an integration of differential equations to show the whole epidemic trend.

2.3 Demography

Birth and death rates

The basic SIR model is based on the assumption that the disease spreads so quickly that not to be influenced by people born or who died. However, in reality, it should consider the new susceptible of birth and the reduced number of people of death. So, it is critical to observe the dynamics of an infectious disease in demographic processes. Because of the influx of new susceptible, a natural host lifespan, $1/\mu$ years, has been assumed. And then, the natural birth rate where individuals also suffer natural mortality is defined as μ. Considering all factors, it draws the SIR model with demography:

$$\frac{dS}{dt} = \mu - \beta SI - \mu S \tag{5}$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \tag{6}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{7}$$

where R_0, the secondary infectives per index case, has changed. Since the outflux transmission of infected is $\frac{1}{\gamma+\mu}$ time unit. Influx infectious rate β is stable. The R_0 becomes:

$$R_0 = \frac{\beta}{\gamma+\mu} \tag{8}$$

The increase of R_0 is understandable because some infected people died during the average period.

Another interesting idea is to explore when the system reachs a state of equilibrium after varring, with $\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$. It can set equations ((5)-(7)) as zero. After caculation, it gets the fixed point: $(S^*, I^*, R^*) = \left(\frac{1}{R_0}, \frac{\mu}{\beta}(R_0 - 1), 1 - \frac{1}{R_0} - \frac{\mu}{\beta}(R_0 - 1)\right)$. It means if $R_0 > 1$, the epidemic will evolve as such equilibrium. If $R_0 < 1$, the disease will die out.

Infection induced mortality

Similarly, it should notice that some disease has a high mortality on human, that may induce a decrease in infected people. When the mortality probability from the disease before recovering has been defined as ρ, it can get a more completed SIR model equations:

$$\frac{dS}{dt} = \mu - \beta SI - \mu S \tag{9}$$

$$\frac{dI}{dt} = \beta SI - \frac{\gamma+\mu}{1-\rho} I \tag{10}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{11}$$

where the reproductive ratio can be calculated by equation (10):

$$R_0 = \frac{\beta(1-\rho)}{\gamma+\mu} \tag{12}$$

2.4 SEIR model

Most infectious diseases have an incubation period, also called a hidden period, which refers to the period from the pathogen invading the body to the appearance of the earliest clinical symptoms. Different infectious diseases have different incubation periods, ranging from as short as a few hours to as long as several years. However, the same infectious disease has a fixed average incubation period. Such non-symptoms individuals are called Exposed and expressed by variable E in the SEIR model:

$$\frac{dS}{dt} = \mu - (\beta I + \mu)S \tag{13}$$

$$\frac{dE}{dt} = \beta SI - (\mu + \sigma)E \tag{14}$$

$$\frac{dI}{dt} = \sigma E - (\mu + \gamma)I \tag{15}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{16}$$

where the average duration of the latent period is given by $1/\sigma$. By setting equations (14) and (15) = 0, the R_0 could be expressed as:

$$R_0 = \frac{\sigma\beta}{(\sigma+\mu)(\gamma+\mu)} \tag{17}$$

Seasoning effect

Some contagious disease like cold, has a seasonal variation to spread pathogen. To examine the dynamical consequences of temporal changes in transmission, the new β has been re-defined. That is a function of time:

$$\beta(t) = \beta_0\,(1 + \beta_1 \cos(\omega t)) \tag{18}$$

where ω is used to adjust the total recurring period. $\omega = 2 \times \pi \div (365 \div 2)$, which means a whole cycle is a half year. And β_0 and β_1 serve as adjustment of amplitude.

3 RESULTS AND DISCUSSION

This section will discuss all the results from section 2, with phase space, Fourier, and control variates analysis, as a result of observing states dynamics, parameters interplay, and occasion behaviour.

3.1 Basic SIR model and threshold

Because there are two important parameters, the transmission rate of β and recovery rate of γ, associated with the epidemic. To know how two parameters interact with each other and impact on curve, there are totally two groups of experimental setting available: β = [1.5, 2.5, 3.5], γ = [0.5, 1.1, 1.7]. Then, assume the initial susceptible fraction is 0.95, and the infected belongs to 0.05 without recovered people. By numerical integration of SIR Ordinary differential equations, it produces nine combinations with different R_0. However, the content capacity restricts only one subfigure listed here. The full results exist in the APPENDIX section (Figure 11), with one of the subfigures as following:

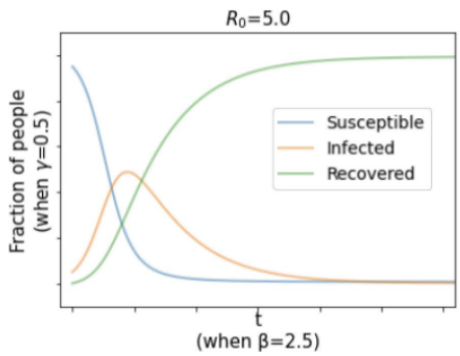

Figure 1: Basic SIR model

According to Figure 1, at the initial stage, the S (susceptible) is going down fast compared to the steady increase of I (infected) and R (recovered) people. The infected starts are reducing until it reaches a peak; meanwhile, the number of S equals I. Looking back to the rest graphs in Figure 11, the same rule still works for most situations, except for disease-free conditions when $R_0 \leq 1$.

Therefore, to demonstrate R_0 decides whether an epidemic occur as Section 2.1 threshold mentioned, another relation chart is provided: Figure 2 shows the total infected people including the recoverd has an exponential growing with R_0. The pathogen reachs a horriable infectious degree when R_0 = 5, because nearly whole people have got infected. Such a rule is also verified by the phase space analysis (Figure 3). When S_0 is less than $1/R_0$, the curve of the I directly drops down to 0. While S_0 is greater than $1/R_0$, I firstly have an increase and then decrease down 0. Additional, by comparing different R_0 in APPENDIX (Figure 12), as R_0 rising, the peak of I become higher.

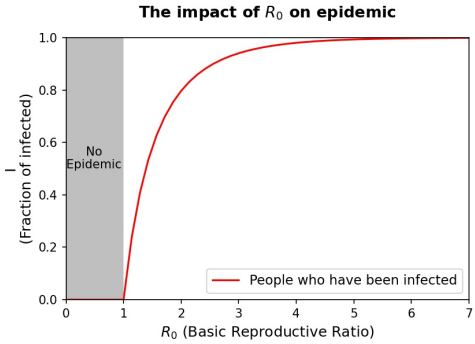

Figure 2: I and R_0 in SIR

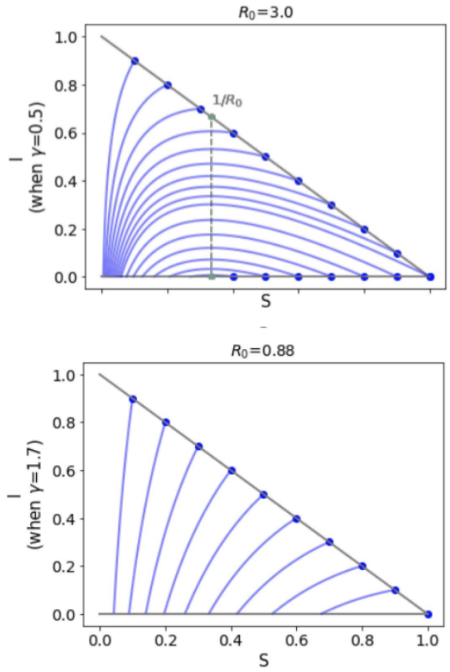

Figure 3: Phase space analysis in SIR

3.2 School epidemic

There is an influenza outbreak happening in a boys school, where the total number of students is 763. Every day, the specific infected number is recorded in Table 1. After using the non-linear square method, β (1.66) and γ (0.45) can be calculated to fit the basic SIR model. Then, the whole dynamics of S, I, and R are shown in Figure 4, where the I curve has a great fitting with historical data.

Many epidemics can be avoided by a vaccination plan beforehand. So, based on section 2.1, the number of people who need vaccination to prevent an epidemic is calculated ($S_0 < 1/R_0$). The epidemic will not happen if the susceptible people are less than 207 (556 vaccinated) (Figure 5). This story tells that the

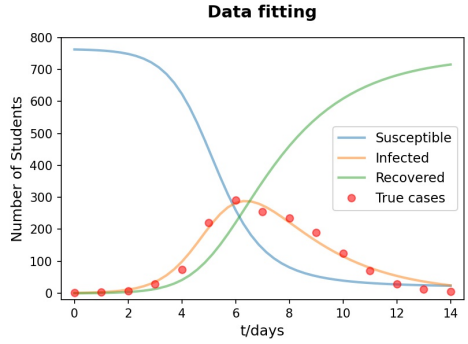

Figure 4: Influenza in school

effective way to deal with COVID-19 is vaccinated. Only if enough amount of people get vaccinated, the disease can burn out.

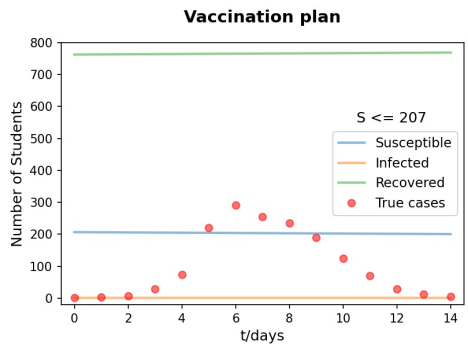

Figure 5: Vaccination

3.3 SIR model with demography

Assume the total population is stable, putting a natural birth and death rate (μ) into the basic SIR model. Furthermore, do the similar experimental parameters settings as Section 3.1 with different μ. The dynamics of SIR with demography are listed in the APPENDIX (Figure 13).

It shows that this model has a similar trend with a basic model at the beginning. However, I and R have to experience successively decreasing oscillation before reaching a state of equilibrium. The reason may relate to newborn people as a supply for the susceptible. Moreover, recovered people are constantly dying. In addition, it illustrates that the shorter the average lifespan is, the earlier an equilibrium happen, although they have approximately the same R_0 values. A possible explanation is that the people with a longer lifespan spend more average time on the whole changing process: S-I-R, whereas short-lived people experience less. So, high birth and death rates provide the model with fast replacement like metabolism

in biology.

One exciting part is that after reaching stable, S is more significant than R when R_0=1.33, 1.34, and 1.35. This phenomenon only appears when infectious disease ability is weak.

Compared with the primary SIR model phase space, the demography has a red stability point surrounded by a spiral if R_0 is large enough (Figure 6, from APPENDIX: Figure 14). S has a decline and follows a backwards tempered augment, which means the members of susceptible people are recharged by the new. Also, it can be found that when R_0 going up, the location of the stability point moves to the y-axis. Probably, if a disease has a robust infectious ability, it needs more recovered people to build a vaccination wall to prevent spreading.

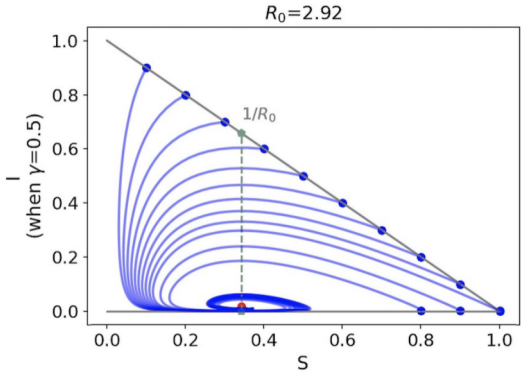

Figure 6: Phase space diagram of SIR with demography

To further explore the oscillation properties, Fourier analysis is used to analyze its amplitude and frequency (APPENDIX: Figure 15). Figure 6 only selects two graphs of those and illustrates that as R_0 going up, the oscillations has a huge amplitude and primarily concentrate on the early stage.

Another key factor is disease-induced mortality. It is defined as ρ. The same experimental procedure work as well (APPENDIX: Figure 16), and the obvious difference is that the horizontal centerline between S and R moves downward. It can be explained as some dead people due to illness.

Figure 8 shows that mortality is bad for disease spreading. If the pathogen is too fatal, the host will quickly die before infecting the others.

3.4 SEIR model

A new category for infected but not yet infectious is added, referred to as Exposed and represented by the variable E, to build the SEIR model. Defining the average duration of the latent period is 1/σ. Through the

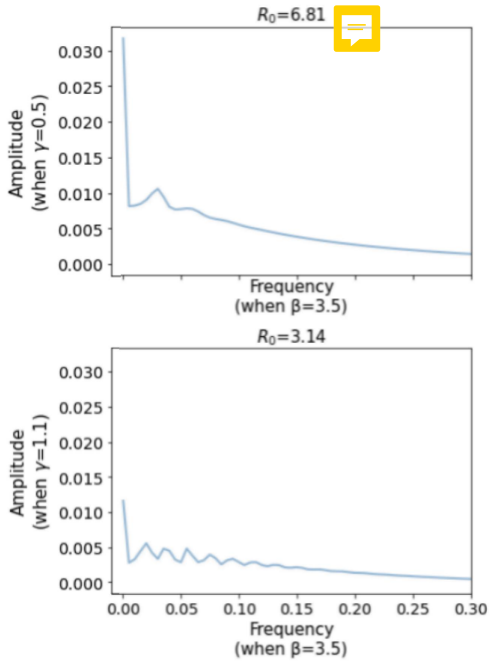

Figure 7: Fourier analysis towards SIR with demography

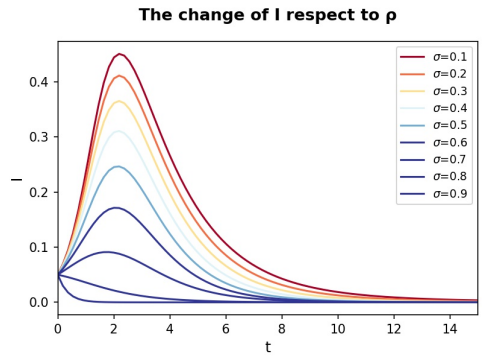

Figure 8: Impact of mortality on I

simulation of different initial conditions of the proportion of patients and latent people under different parameters (APPENDIX: Figure 17), it can be seen that the initial conditions of the proportion of σ have a direct impact on the time of occurrence, peak, and end of the epidemic. However, it has little effect on the shape and characteristics of the epidemic curve. The epidemic curve under different initial value conditions almost shifts along with the time index.

In these figures, the exposed decreases after reaching the peak, until it approaches a low level but higher than infected people; the susceptible decreases rapidly after the outbreak.

Figure 9 explores the impact of contact rate on I. A single factor analysis of various σ under similar conditions shows that as the contact rate increases, the in-

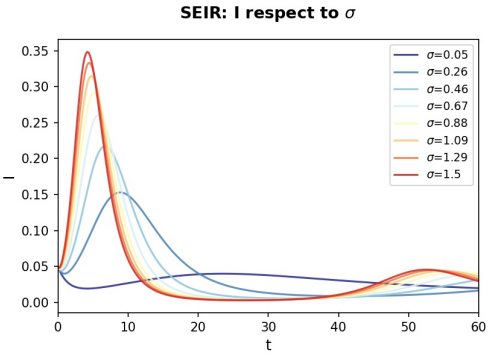

Figure 9: Impact of mortality on I

fected peaks earlier and more robust, but eventually, they all stabilize.

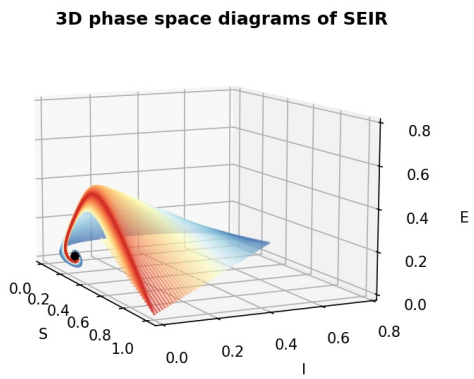

Figure 10: 3D phase space analysis

Because the SEIR model has an additional compartment, 3D phase space analysis of the SEIR model is introduced. From the S-I 2D dimension, it looks the same as the SIR model with demography. In E division, the exposed has a smooth growing sphere converge with other compartments on the top. Furthermore, decrease steadily towards the stability point as a spiral way.

After introducing the seasonal effect, the curves become periodic oscillations repetitively (APPENDIX: Figure 18). The time-varying transmission rate is expressed by the up and down movement of the four compartments.

4 CONCLUSION

In summary, this paper investigates phenomena in real life, summarises them as an abstract system, lists mathematical equations, and builds a vivid model on a computer, strengthening the importance of computational science. It does much work of analysis. From the basic SIR, SIR with demography, SIR with mortality, and SEIR, it academically obeys a strict research way.

SIR model gives insight into how different compartments vary. Based on its observation, a key factor: R_0, has been introduced. This factor determines almost every curve in all graphs, including the following ones. The school influenza outbreak can be deterred by 73% vaccinated people beforehand. For the SIR model with demography, there are constant oscillations in curves, followed by Fourier analysis stating R_0 influence their amplitude and frequency at a certain level. Mortality performs an inverse effect on disease spreading.

SEIR model has an additional incubation status but has similar dynamics with the SIR model. However, after adding seasonal force, the model starts a periodicity. S, E, I, R peak one by one with various amplitude. These results are practical to predict and control the epidemic.

In the future, more work could be done to analyze the 3D phase diagram in Section 3.4. The model becomes too complex to find some valuable clues. Moreover, design a better way to evaluate our models. To simulate the seasonal effect, the β sinusoidal expression is too simple to reflect actual events, is there a way to investigate a more complex case using Taylor Formula?

REFERENCES

Chowdhury, S., Roychowdhury, S., and Chaudhuri, I. (2021). Universality and herd immunity threshold : Revisiting the sir model for covid-19.

Keeling, M. and Rohani, P. (2008). *Modeling Infectious Diseases in Humans and Animals*. Princeton University Press.

Kermack, W. O. and McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London. Series A, Containing papers of a mathematical and physical character*, 115(772):700–721.

Weber, T., Hurley, J., and Adăscăliței, D. (2021). *COVID-19: implications for employment and working life*. Employment and labour markets. Publications Office, Luxembourg.

APPENDIX

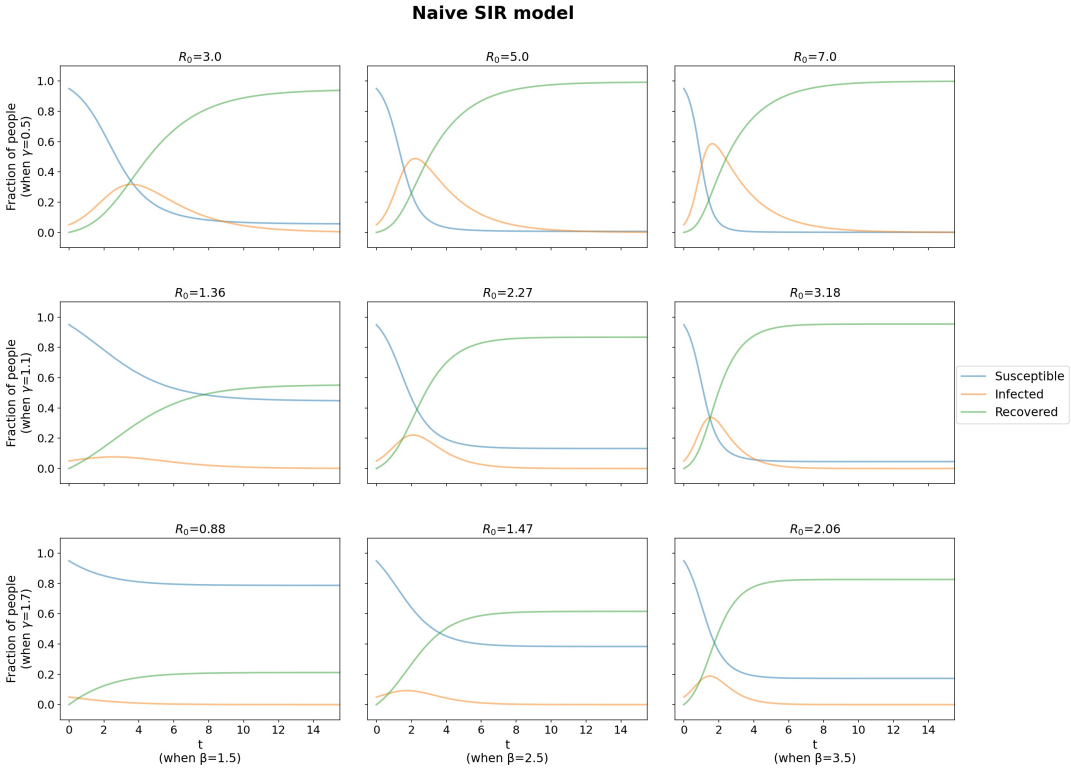

Figure 11: Initialization: $S_0 = 0.95$, $I_0 = 0.05$, $Recoverd_0 = 0$

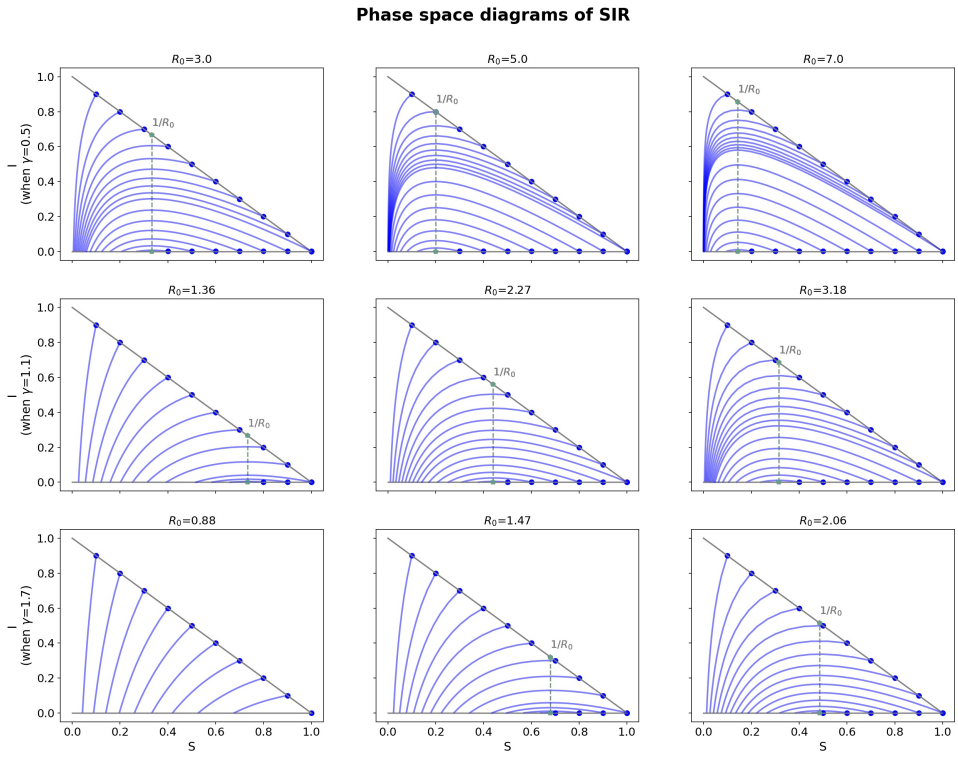

Figure 12: Initialization: S_0 = [from 0.1 to 1 increases by 0.1], I_0 = 1 - S_0, $Recoverd_0$ = 0

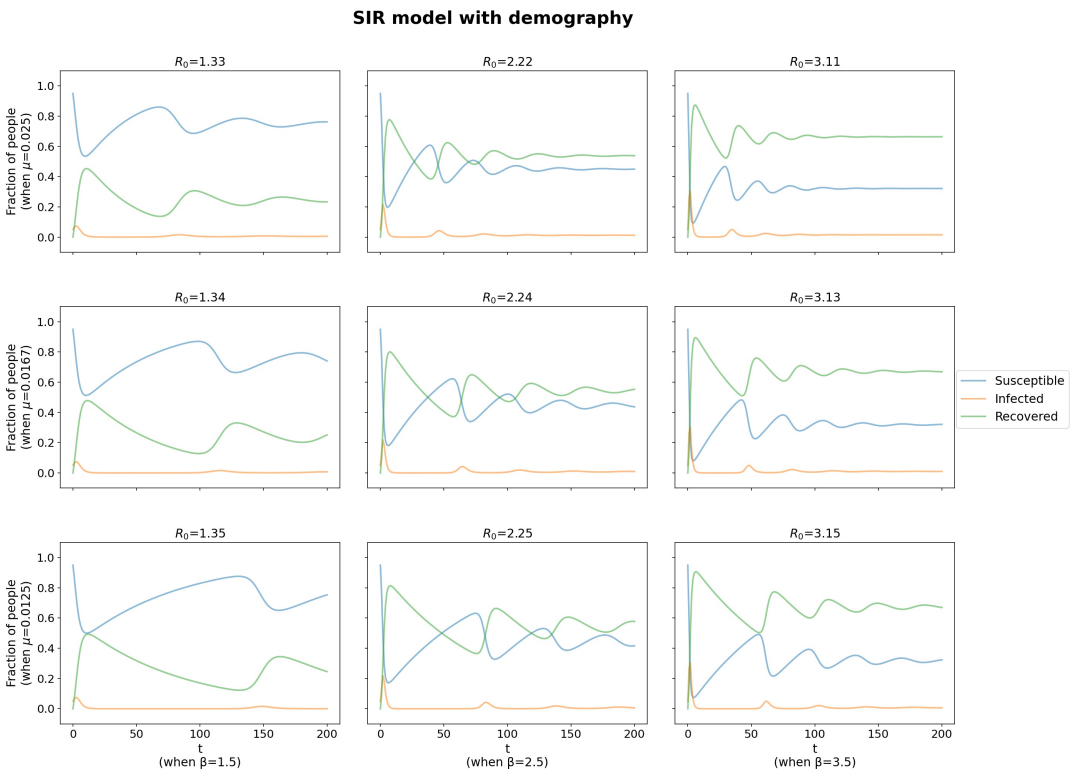

Figure 13: Initialization: S_0 = 0.95, I_0 = 0.05, $Recoverd_0$ = 0, γ = 1.1

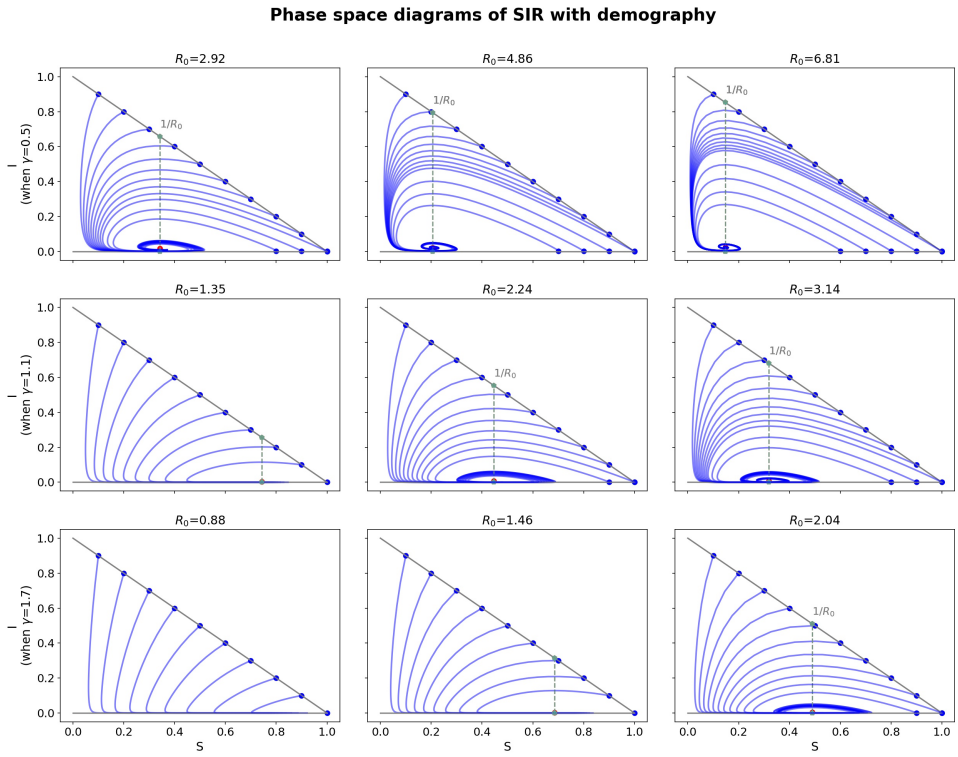

Figure 14: Initialization: S_0 = [from 0.1 to 1 increases by 0.1], I_0 = 1 - S_0, $Recoverd_0$ = 0, μ = 1/70

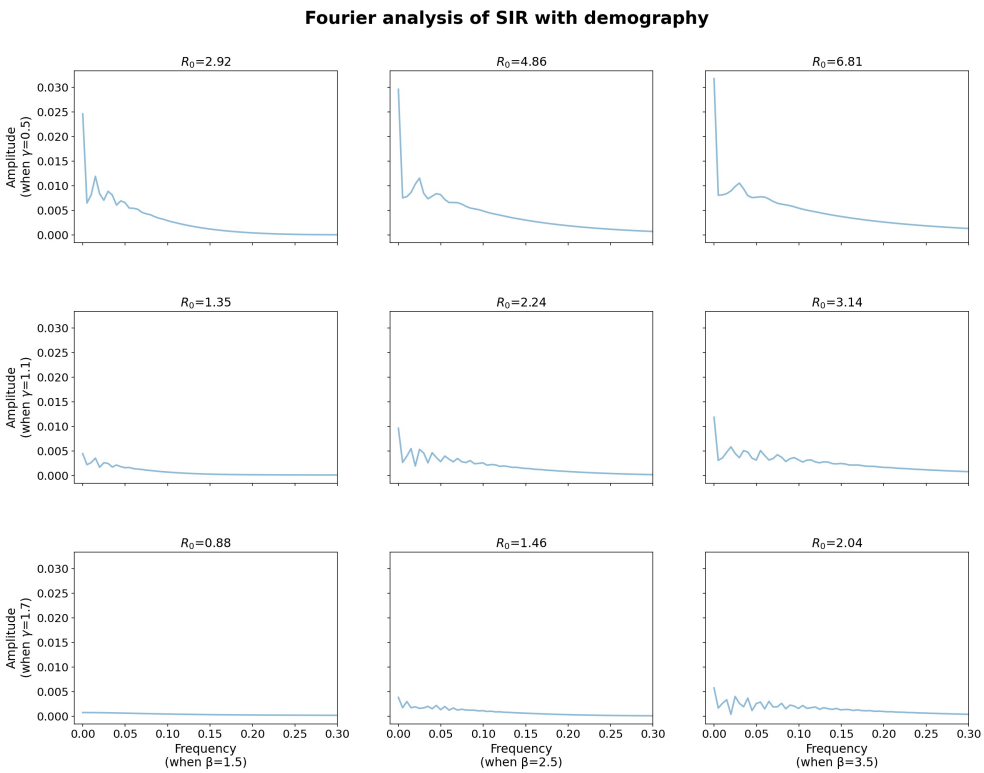

Figure 15: Initialization: S_0 = 0.95, I_0 = 0.05, $Recoverd_0$ = 0, μ = 1/70, t = 200

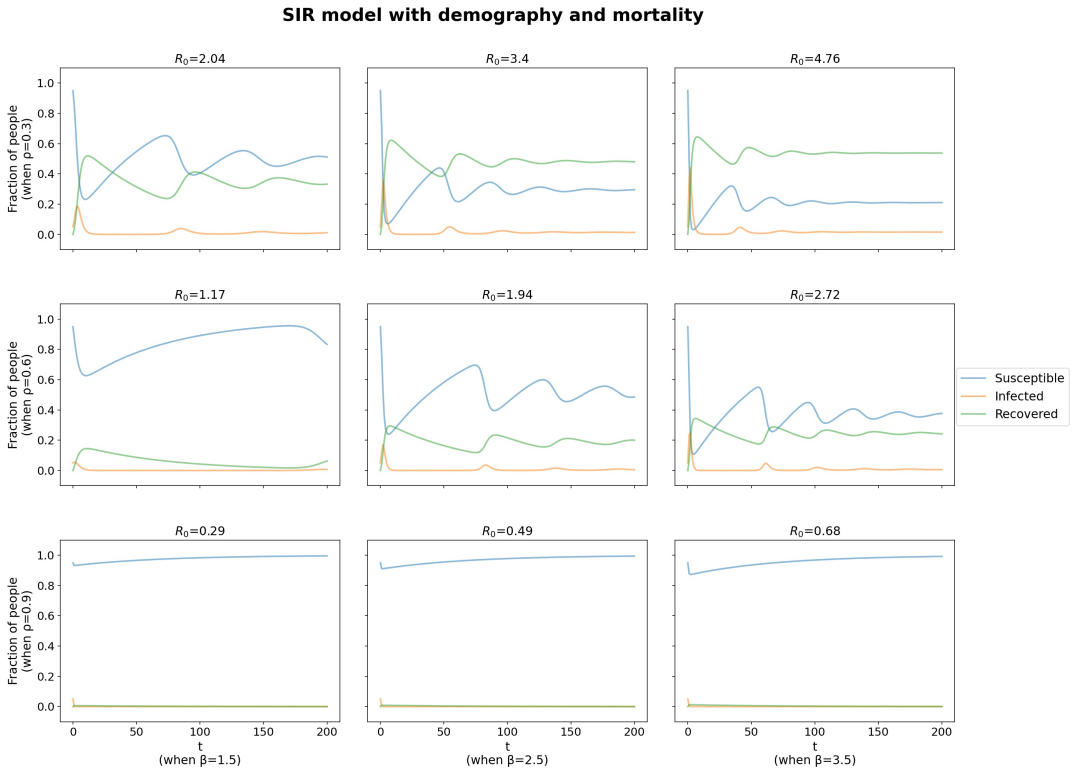

Figure 16: Initialization: $S_0 = 0.95$, $I_0 = 0.05$, $Recoverd_0 = 0$, $\gamma = 0.5$, $\mu = 1/70$

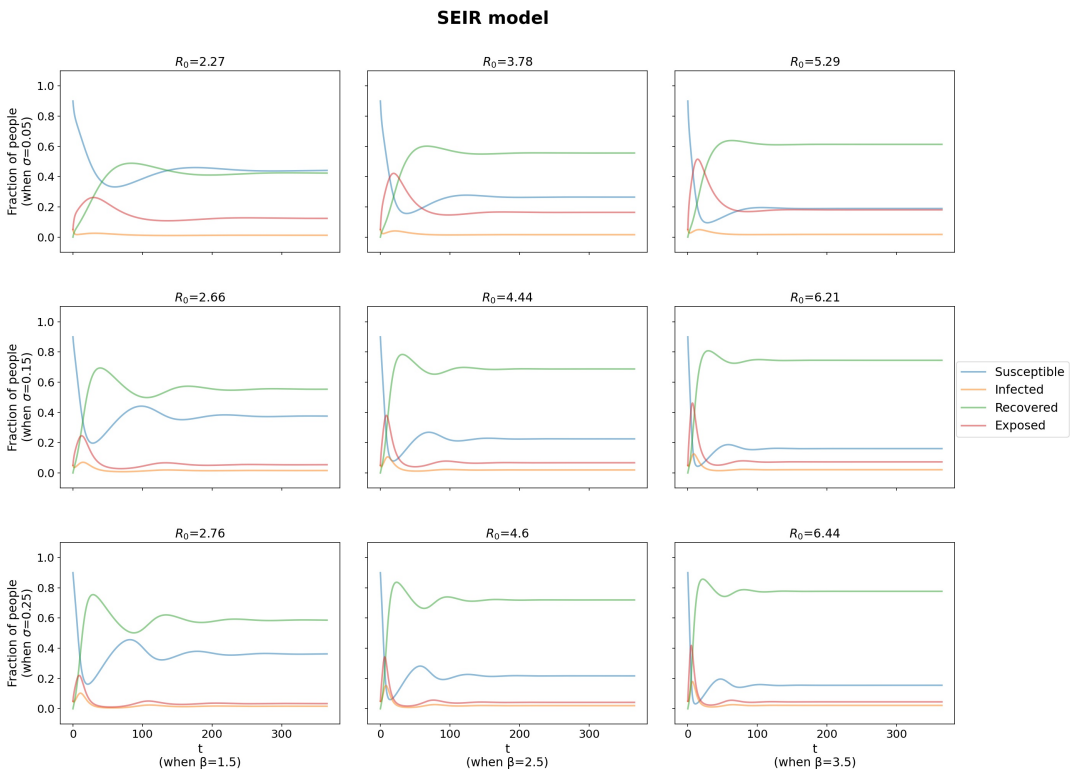

Figure 17: Initialization: $S_0 = 0.9$, $E_0 = 0.05$, $I_0 = 0.05$, $Recoverd_0 = 0$, $\gamma = 0.5$, $\mu = 1/70$

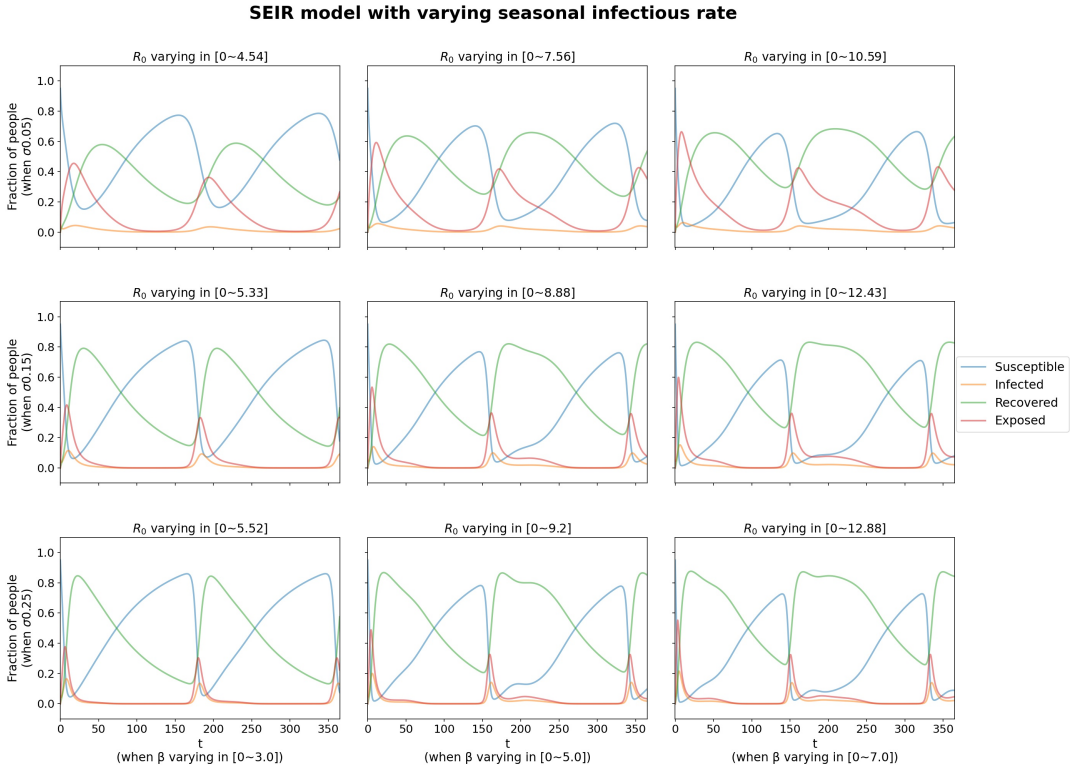

Figure 18: Initialization: S_0 = [from 0.1 to 1 increases by 0.02], $E_0 = 0$, $I_0 = 1 - S_0$, $Recoverd_0 = 0$, $\beta = 2.5$, $\gamma = 0.5$, $\mu = 1/70$, $\sigma = 0.15$