SIR model(ODE)

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ABSTRACT

The SIR model is an epidemiological model that accounts for the infection efficiency and computes the theoretical number of people in different states through the spread of an infectious disease in a closed population over time. In this report, we first introduce the naive SIR model and numerical integration of this model for different states. Then we add demography and morality to the model and analyze the oscillatory behavior to identify the role R_0 plays in each model. Finally, we explore the SEIR model and analyze the seasonal effects of this model.

1 INTRODUCTION

The SIR model is one of the simplest models to account for infectious diseases. This model was initially studied by Kermack and Mckendrick(1927), categorizes hosts within a population as Susceptible(S), Infected(I), and Recovered(R) [2]. The transitions are from S to I classes and from I to R classes.

We discuss the threshold phenomenon and find out that the basic reproductive rate R_0 is the key point that could determine whether there is an epidemic burnout. Additionally, we analyze the historical data from the case of an influenza outbreak situation and fit a SIR model to this data. Then we propose the mass vaccination strategy [3] to prevent the epidemic.

Sometimes we want to explore the longer-term persistence and endemic dynamics of infectious diseases, thus we introduce demography into the SIR model and analyze the oscillatory dynamics and equilibrium states of this model. Then in some cases disease-induced mortality could cause the decline of population size, thus we add density-dependent transmission and frequency-dependent transmission to the SIR model respectively to clarify the relationship between disease mortality probabilities ρ and reproductive rate R_0.

At last, we refine the SIR model to take into account the latent period-Exposed(E). Then we add seasonal effects to this SEIR model to see the variation.

2 THE BASIC SIR MODEL

2.1 Numerical integration of the naive SIR model

In this section, we introduce the SIR model equations without considering demographics (no births, deaths, or migration) and numerically integrate ODEs for states of an epidemic and no epidemic to find out the parameter that determines whether the infectious disease will become an epidemic burnout.

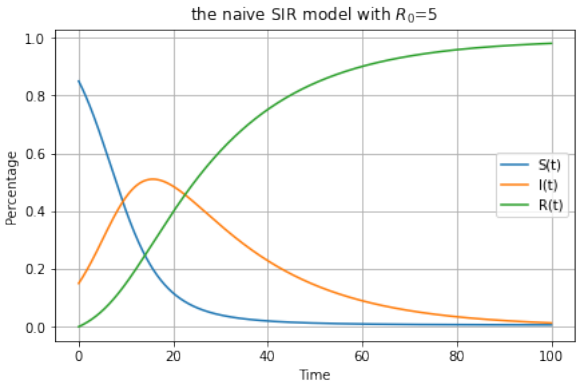

Figure 1: The naive SIR model with $\beta = 1/4$ per day , $\gamma = 1/20$ per day , $S_0 = 0.85$, $I_0 = 0.15$.

2.1.1 Theory. The SIR model shows that the transmissions are from S to I classes and from I to R classes. If we do not take demography into account, we can make this basic model explicit and straightforward.

In the movement from S to I classes, the new infecteds are produced by contact between infecteds and susceptibles. We define the force of infection λ as the per capita rate at which susceptible individuals contract the infection. Thus λX(where X is the number of individuals in class) is the rate of new infecteds and we got $\lambda = \beta Y/N$ and $\lambda = \beta Y$(where Y is the number of infectious individuals, N is the total population size, and β is the product of the contact rates and transmission probability). If the host population size does not vary, we can absorb the $1/N$ term into the parameterization of β in the mass-action term.

In the movement from I to R classes, we define γ as the removal or recovery rate. Additionally, we denote $S(= X/N)$ and $I(= Y/N)$ as the proportion of susceptibles and infecteds respectively. Then we can get the following set of first-order differential equations:

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

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

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

2.1.2 Method and Discussion. We used the python odeint function to integrate these differential equations and drew two different plots by setting different parameters. As shown in Figure 1 and Figure 2, the initial situation is identical except for β and γ so we can analyze how these two parameters determine the occurrence of an epidemic burnout.

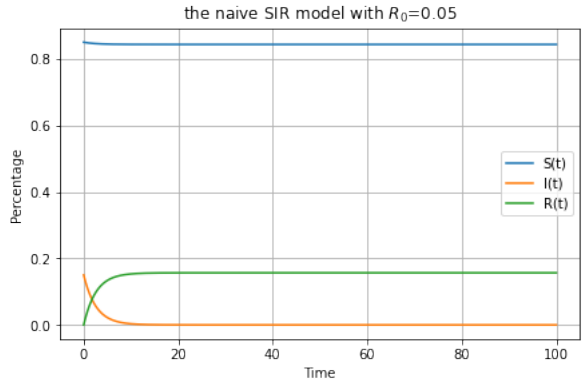

Figure 2: The naive SIR model with $\beta = 1/50$ per day , $\gamma = 1/2.5$ per day , $S_0 = 0.85$, $I_0 = 0.15$.

First, we rewrite equation2 in the form:

$$\frac{dI}{dt} = I(\beta S - \gamma) \tag{4}$$

we can notice that if $S(0)$ is less than γ/β, then $\frac{dI}{dt}$<0 and the infection "dies out" like Figure2. And if $S(0)$ is greater than γ/β, there will be an epidemic burnout like Figure1.Thus we define the inverse of γ/β(the relative removal rate) as the basic reproductive ratio R_0, also known as the maximum reproductive potential for an infectious disease. We can assume everyone is susceptible($S(0) = 1$) at first, then the infectious disease will spread out if R_0>1. If R_0<1, the infectious disease cannot invade and there is no epidemic burnout.

When concerning about the long-term state, we first divide equation1 by equation3 and get following equation:

$$\frac{dS}{dR} = -\frac{\beta S}{\gamma} = -R_0 S \tag{5}$$

Then we integrate with respect to R and obtain

$$S(t) = S(0)e^{-R(t)R_0} \tag{6}$$

Along with the definition $S+I+R = 1$ and the fact that the epidemic ends when $I = 0$, we can obtain the long-term behavior of equations:

$$S(\infty) = 1 - R(\infty) = S(0)e^{-R(\infty)R_0} \tag{7}$$

$$1 - R(\infty) - S(0)e^{-R(\infty)R_0} = 0 \tag{8}$$

Using standard methods, it is possible to find an approximate numerical solution for equation8 and this is shown in Figure3. This figure also indicates that if R_0>1, then the epidemic occurs. When R_0>1, the number of infecteds first increases to then decrease toward 0 and this threshold effect is illustrated in Figure4. We can see in this figure that when S_0>γ/β, the infecteds first increase to then decrease toward 0; when S_0<γ/β, the infecteds decrease toward 0.

2.2 Analysis of historical data

We are given the historical data from a case of an influenza outbreak situation as shown in table1. The outbreak was started by one infected boy in a boy school with a total of 763 boys.

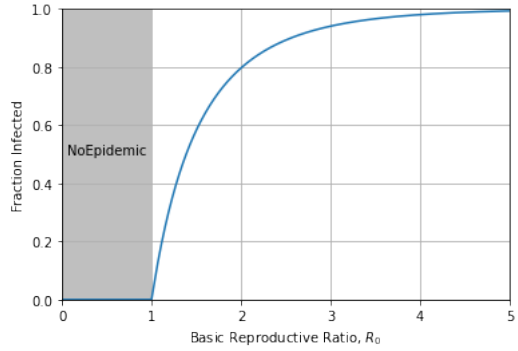

Figure 3: The total fraction of the population infected as a function of disease R_0

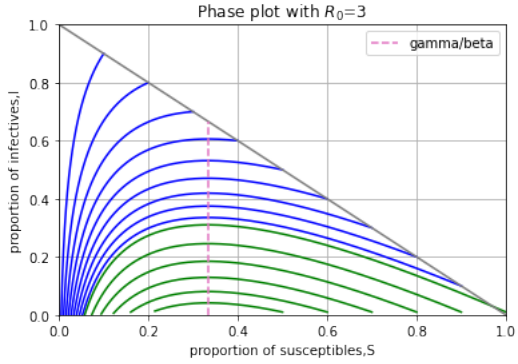

Figure 4: Phase plot of the naive SIR model with R_0=3

2.2.1 Theory. We can see that there is epidemiology without host demography in this case. Thus we can estimate the parameters for the naive SIR model from these data. According to the simple least-squares procedure, the estimated infectious period ($1/\gamma$) is 2.2 days and a mean transmission rate(β) is 1.66 per day[2]. Then we set the initial parameters to this model and try to fit these data to it.

2.2.2 Method and Discussion. We used the python curve-fit function to fit these data to the naive SIR model. As shown in Figure5, the model dynamics with $\beta = 1.66$, $\gamma = 1/2.2$ and $R_0 = \beta/\gamma = 3.652$ is in good correspondence with real data. Thus we can conclude that the influenza data fits entirely into this naive SIR model.

2.3 Vaccination plan

To prevent the epidemic shown in the last section, we can introduce a vaccination plan to this model.

2.3.1 Theory. We define p as the vaccination coverage with vaccine efficacy equal to 100% so the original proportion of susceptibles would decline by p then we get $S^* = 1-p$. We are also familiar with the properties of the endemic equilibrium that R is equal to R_0 times the proportion of susceptibles: $R = R_0 S^*$. If we want the disease to be eradicated, R must be less than 1:$R = R_0 S^* = R_0(1-p) < 1$, or $p > 1 - 1/R_0$. Thus the critical vaccination coverage p_c for disease eradication is $p_c = 1 - 1/R_0$[1]. This means that we do not need

Table 1: Data of an influenza outbreak

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

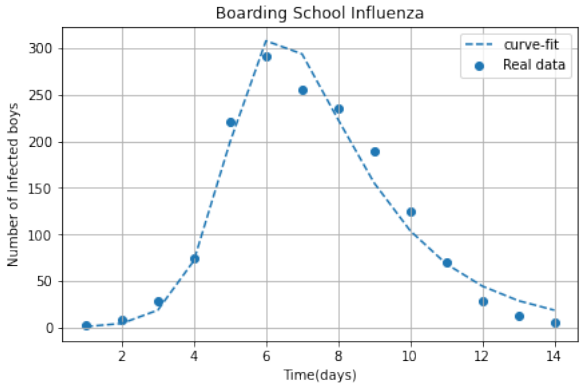

Figure 5: The SIR dynamics. The filled circles represent the real data. The curve represents solutions from SIR model fitted to the data.

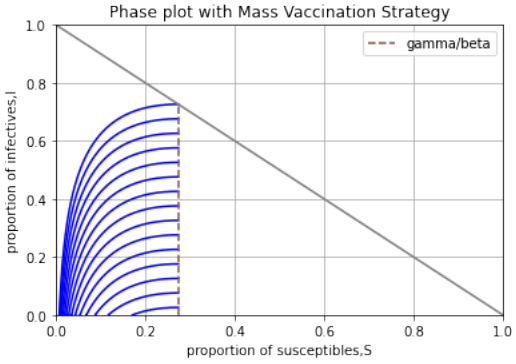

Figure 6: Phase plot of naive SIR model with mass vaccination coverage of $p_c = 1/R_0 = 1/3.652$

to vaccinate every individual to protect the whole population[3]. Note that the higher R_0, the higher p_c should be.

2.3.2 Method and Discussion. According to the mass vaccination strategy above, we can obtain the threshold effect like Figure6. The proportion of susceptibles vaccinated is $p_c = 1-1/R_0$ so the original remaining $S(0) = 1-p_c = 1/R_0$. Thus the proportion of susceptibles is always smaller than γ/β and we can see on this figure that when $S_0<\gamma/\beta$, the infecteds decrease toward 0. There will be no epidemic using this vaccination strategy.

3 DEMOGRAPHY

In this section, we introduce the demographic process into the naive SIR model and present the longer-term persistence and endemic dynamics. Additionally, we explore the consequences of

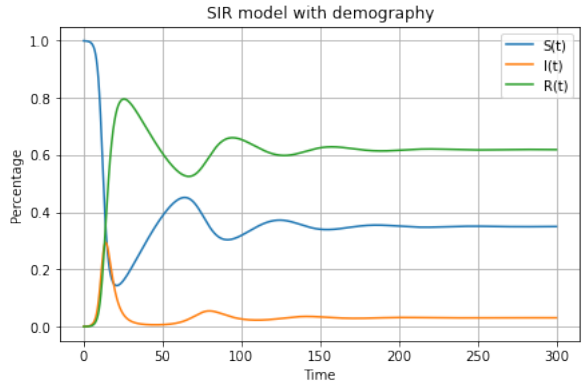

Figure 7: SIR model with demography, β=1 day, $1/\gamma$=3 days,$1/\mu$=60 years, R_0=3

infection-induced morality with density-independent transmission and frequency-independent transmission respectively.

3.1 SIR model with demography

In the following part, we introduce the naive SIR model with demography and analyze the equilibrium state and oscillatory dynamics using Fourier analysis.

3.1.1 Theory. We assume that each individual suffers from natural mortality μ, which is independent of the infectious disease. Additionally, μ can represent the birth rate and all newborn individuals are susceptibles, thus the total population size could be constant through time($\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$). According to these assumptions, we can get the SIR model with demography equations:

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

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

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

3.1.2 Method and Discussion. We used the python odeint function to integrate these differential equations and drew two different plots by setting different parameters. As shown in Figure 7, there is an epidemic burnout; in Figure 8, the disease dies out. We can analyze these differences by assuming $\frac{dI}{dt} = 0$, then we obtain $S(0) = \frac{\gamma+\beta}{\mu}$. If we assume the entire population is susceptibles($S(0) = 1$), the infection efficiency is determined by the basic reproductive ratio $R_0 = \frac{\beta}{\gamma+\mu}$. Then if $R_0 > 1$, the infectious disease would invade like Figure7. If $R_0 < 1$, it would die out in the end.

When concerning about the dynamics, we explore the equilibrium state of this model with $\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$. Thus we can find two fixed points and the first one is expressed as(S^*,I^*,R^*)=(1,0,0).

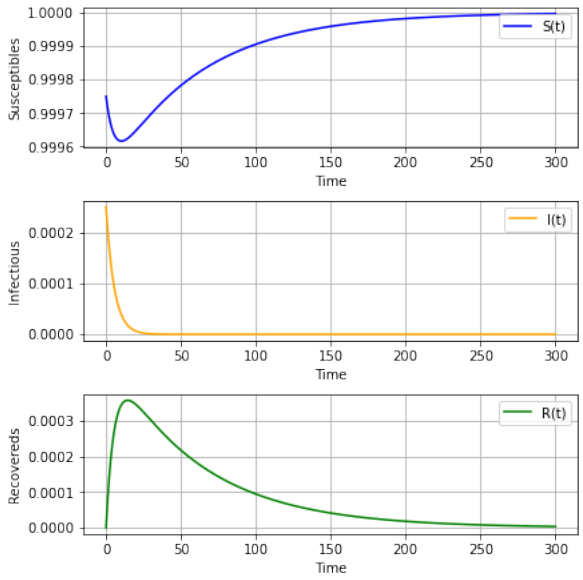

Figure 8: SIR model with demography, β=1/6 day, $1/\gamma$=3 days,$1/\mu$=60 years, R_0=1/2

To find the second one, we start by setting the equation10 to 0 and then factor for I to get:

$$I(\beta S - (\gamma + \mu)) = 0 \tag{12}$$

which is satisfied when I^*=0 or $S^* = \frac{\gamma+\mu}{\beta}$. Thus we ignore the first condition when there is no disease and find out that the second condition $S^* = \frac{1}{R_0}$. So we substitute this into equation9 to obtain:

$$I^* = \frac{\mu}{\gamma}(1 - \frac{1}{R_0}) = \frac{\mu}{\beta}(R_0 - 1) \tag{13}$$

Since $S^* + I^* + R^* = 1$, the second fixed point we get is expressed as:

$$(S^*, I^*, R^*) = \left(\frac{1}{R_0}, \frac{\mu}{\beta}(R_0 - 1), 1 - \frac{1}{R_0} - \frac{\mu}{\beta}(R_0 - 1)\right) \tag{14}$$

We drew two phase plots to further indicate the equilibrium states. As shown in Figure9: when $R_0 > 1$, there is an endemic equilibrium and the lines converge to a fixed point. And Figure10 shows the disease-free equilibrium when $R_0 < 1$.

The SIR model with demography shows a damped oscillator with the amplitude of these fluctuations declining through time. To explore the equilibrium dynamics, we take linear stability analysis based on eigenvalues. As mentioned in the equation14, we now introduce the deviations from the equilibrium into it and obtain:

$$S(t) = S^* + \xi(t) \tag{15}$$

$$I(t) = I^* + \zeta(t) \tag{16}$$

We then focus on the dynamics of the deviations $\xi(t)$ and $\zeta(t)$ and rewrite them into:

$$\frac{d\xi}{dt} = -(\beta I^* + \mu)\xi - \beta S^* \zeta + NL(\xi, \zeta) \tag{17}$$

$$\frac{d\zeta}{dt} = \beta I^* \zeta + NL(\xi, \zeta) \tag{18}$$

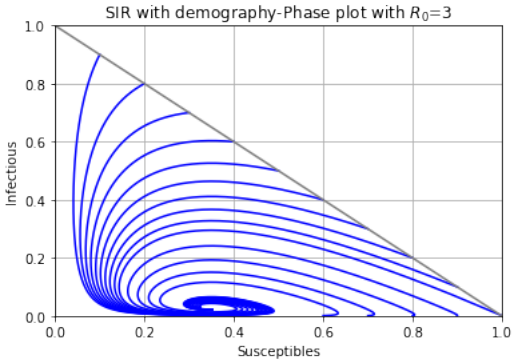

Figure 9: Phase plot of SIR model with demography, β=1 day, $1/\gamma$=3 days,$1/\mu$=60 years, R_0=3

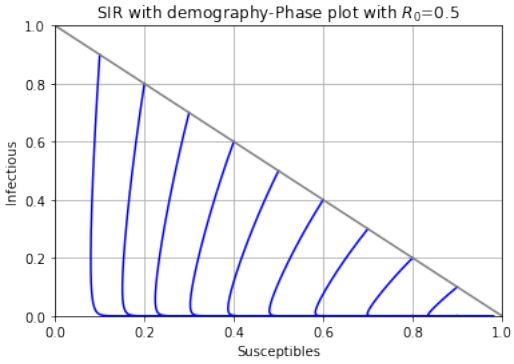

Figure 10: Phase plot of SIR model with demography, β=1/6 day, $1/\gamma$=3 days,$1/\mu$=60 years, R_0=1/2

where $NL(\xi, \zeta)$ contains all nonlinear terms. Substitute S^* and I^* to get:

$$\frac{d\xi}{dt} = -[\mu(R_0 - 1) + \mu]\xi - \frac{\beta}{R_0}\zeta + NL(\xi, \zeta) \tag{19}$$

$$\frac{d\zeta}{dt} = \mu(R_0 - 1)\xi + NL(\xi, \zeta) \tag{20}$$

We change part of them into matrix form, then they become

$$\begin{bmatrix} -\mu(R_0 - 1) - \mu & -\beta/R_0 \\ \mu(R_0 - 1) & 0 \end{bmatrix} \tag{21}$$

And this is called the Jacobian matrix $\vec{J}$ and its eigenvalues can indicate the dynamics of the system. The eigenvalues are the solutions of the following equation

$$\Lambda^2 - Tr(\vec{J})\Lambda + det(\vec{J}) = 0 \tag{22}$$

We then substitute the trace and the determinant of $\vec{J}$ into this equation and get

$$\Lambda^2 - \mu R_0 \Lambda + \mu(\mu + \gamma)(R_0 - 1) = 0 \tag{23}$$

$$\Lambda_{1,2} = \frac{-\mu R_0 \pm \sqrt{(\mu R_0)^2 - 4\mu(\mu + \gamma)(R_0 - 1)}}{2} \tag{24}$$

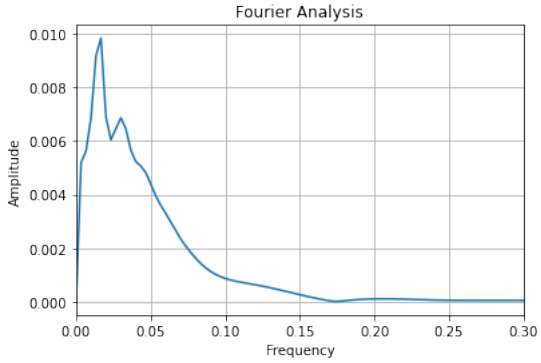

Figure 11: Fourier Analysis, β=1 day, $1/\gamma$=3 days,$1/\mu$=60 years, R_0=3

Note that μ is a quite small number(the inverse of lifespan), thus $(\mu R_0)^2$ can be ignored and we end with

$$\Lambda_{1,2} = \frac{-\mu R_0 \pm \sqrt{4\mu(\mu+\gamma)(R_0-1)}}{2} = -\frac{\mu R_0}{2} \pm \frac{i}{\sqrt{AG}} \tag{25}$$

where A represents the mean age at infection, $A = \frac{1}{\mu(R_0-1)}$ and G represents the mean infection period, $G = \frac{1}{\gamma+\mu}$. So we will have damped oscillations with frequency $\omega = \frac{i}{\sqrt{AG}}$, the period of oscillations will be $T = \frac{2\pi}{\omega} = 2\pi\sqrt{AG}$. Thus we can measure this in simulations using the python Fourier transform function to analyze the frequency and amplitude. As shown in Figure11, we analyze the data from Figure7 and find two peaks. The more important one is the first peak, so we obtain the value of this peak frequency which is 0.0167. Then we substitute same parameters into equations to calculate frequency $1/T = \frac{1}{2\pi\sqrt{AG}} = 0.0172$. Thus our result of Fourier analysis is similar to the calculation result, which further verifies all the assumptions and deductions above.

3.2 SIR model with disease induced mortality

In this section, we will introduce a mortality probability into the SIR model and consider density-dependent transmission and frequency-dependent transmission respectively. Additionally, we explore the impact of the basic reproduction ratio R_0 on the population.

3.2.1 Theory. We first introduce ρ(the per capita probability of dying from the infection before either recovering or dying from natural causes) to the infectious classes and get the following equation:

$$\frac{dI}{dt} = \beta SI - \frac{(\gamma+\mu)}{1-\rho} I \tag{26}$$

Then we incorporate a fixed birth rate(υ) into the susceptible equation9 and get:

$$\frac{dS}{dt} = \upsilon - \beta SI - \mu S \tag{27}$$

Now we need to consider the population size N which varies and figure out the density and frequency-dependent transmission respectively.

For density-dependent(pseudo mass action)transmission, the total population size N decreases because of disease-induced morality.

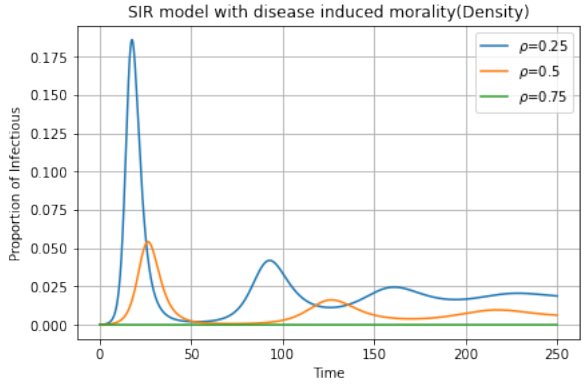

Figure 12: SIR model with disease-induced morality (Density-dependent), β=1 day, $1/\gamma$=3 days,$1/\mu$=60 years, R_0=3

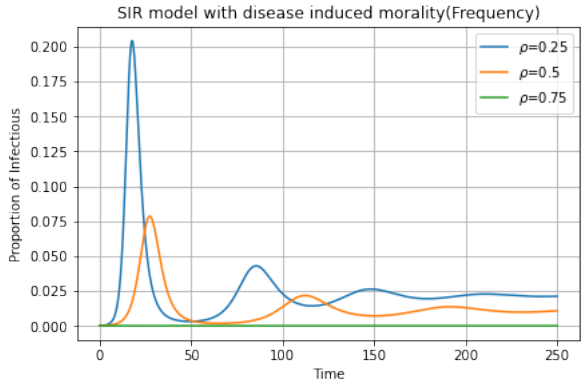

Figure 13: SIR model with disease-induced morality (Frequency-dependent), β=1 day, $1/\gamma$=3 days,$1/\mu$=60 years, R_0=3

Using the deductions mentioned above, we can find two fixed points: one disease free($\frac{\upsilon}{\mu}$,0,0) and one endemic(X^*,Y^*,Z^*)=($\frac{\upsilon}{\mu R_0}$,$\frac{\mu}{\beta}(R_0-1)$,$\frac{\gamma}{\beta}(R_0-1)$) where $R_0 = \frac{\beta(1-\rho)\upsilon}{(\mu+\gamma)\mu}$. Then we obtain

$$N^* = \frac{\upsilon}{\mu R_0}[1+(1-\rho)(R_0-1)] \tag{28}$$

As for frequency-dependent(mass action)transmission, the endemic equilibrium can again be found and obtain

$$N^* = \frac{\upsilon}{\mu}\left(\frac{R_0(1-\rho)}{(R_0-\rho)}\right) \tag{29}$$

3.2.2 Method and Discussion. Based on both density and frequency models, we drew Figure12 and Figure13 with three different mortality probabilities ρ. From these figures, we can see that the higher ρ, the smaller amplitude of oscillators, the smaller proportion of infectious. When ρ approaches 1, the proportion of infectious would go down to almost zero as can be seen from both figures and the equation of R_0. This can be interpreted by the infection process when infectious individuals are more likely to die before they pass this disease to susceptibles.

We can also see this trend in Figure14. Infectious diseases with the highest ρ and the largest R_0 have the greatest impact on the

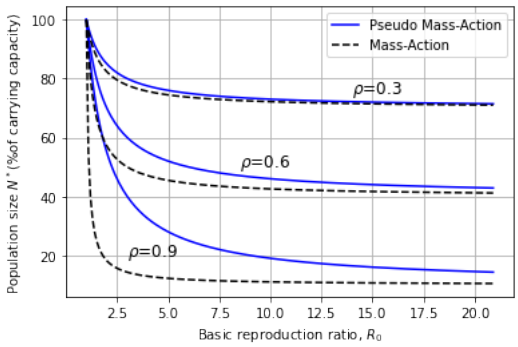

Figure 14: The population size,N^*,for diseases that are associated with mortality.Three different mortality probabilities are considered, ρ=0.3/0.6/0.9, and two mixing assumptions of density dependence(solid line) and frequency dependence(dashed line) are shown.

population. When ρ is high, the total population size declines fast in both models. Additionally, the population size of the frequency-dependent transmission decreases more quickly than density-dependent transmission. This may due to the property of frequency-dependent transmission that the contact rates between individuals decline when the population size decreases and thus limiting disease spread and reducing disease-induced mortality.

4 VARIATION

In this section, we introduce variation to the SIR model and provide details and analysis about this model using the same methods mentioned above.

4.1 The SEIR model

We introduce a refinement to the SIR model and take the latent period- the Exposed(E) classes into account. The Exposed classes mean that they are infected by the pathogen but cannot infect other susceptibles due to a low level of pathogen abundance. Thus the process of transmission turns into $S \longrightarrow E \longrightarrow I \longrightarrow R$.

4.1.1 Theory. We assume the average duration of the exposure period is $1/\sigma$, then get the SEIR equations:

$$\frac{dS}{dt} = \mu - (\beta I + \mu)S \tag{30}$$

$$\frac{dE}{dt} = \beta SI - (\mu + \sigma)E \tag{31}$$

$$\frac{dI}{dt} = \sigma E - (\mu + \gamma)I \tag{32}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{33}$$

4.1.2 Method and Discussion. The SEIR model also has two equilibrium solutions like the SIR model, a disease-free(1,0,0,0) and an endemic $(S^*,E^*,I^*,R^*)=(\frac{1}{R_0},\frac{\mu(\mu+\gamma)}{\beta\sigma}(R_0-1),\frac{\mu}{\beta}(R_0-1),1-S^*-E^*-I^*)$.If the exposed period is very small that can be ignored, then we get the estimated R_0 the same as the SIR model($R_0 = \beta/(\gamma+\mu)$). So if $R_0 > 1$, we could expect an epidemic as shown in Figure15. It is obvious that the dynamic properties of the SEIR model are similar

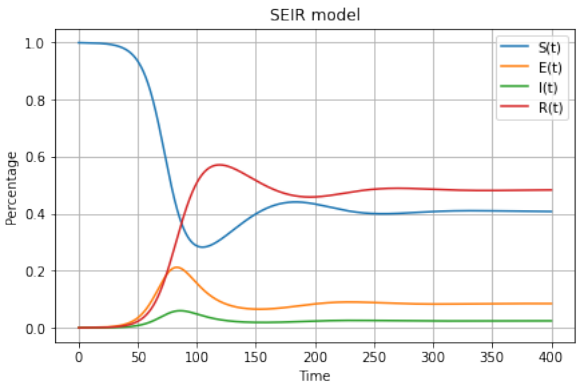

Figure 15: The SEIR model, β=1 day, $1/\gamma$=3 days,$1/\mu$=60 years, $1/\sigma$=10 days

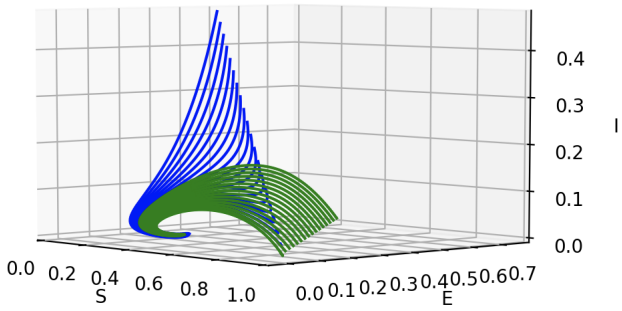

Figure 16: Phase plot of the SEIR model, β=1 day, $1/\gamma$=3 days,$1/\mu$=60 years, $1/\sigma$=10 days

to the SIR model because the exposed period could be regarded as a slight time delay which is introduced into the SIR model.

We also drew a phase plot to further indicate the dynamics. As shown in Figure16, the curves seem to converge to a fixed point, which further verifies our assumptions and deductions above.

4.2 The SEIR model with seasonal effect

Additionally, we add seasonally varying parameters as a temporary forcing mechanism to the SEIR model and explore the consequences.

4.2.1 Theory. We assume the transmission rate is a function of time, thus β is transformed into $\beta(t) = \beta_0(1 + \beta_1 \cos(\omega t))$ where β_0 represents the average transmission rate, ω is the period of the forcing, and β_1 is the amplitude of seasonality. Then we obtain the equations:

$$\frac{dS}{dt} = \mu - (\beta(t)I + \mu)S \tag{34}$$

$$\frac{dE}{dt} = \beta(t)SI - (\mu + \sigma)E \tag{35}$$

4.2.2 Method and Discussion. Due to the seasonal effect, the basic reproductive ratio R_0 is now $\frac{\beta_0}{\gamma+\mu}$(if the latent period is infinitesimally small, $\sigma \longrightarrow \infty$). This value β_0 is the average of the whole year and at a certain point in time, the rate may be higher than this average value. We use python odeint function to draw the

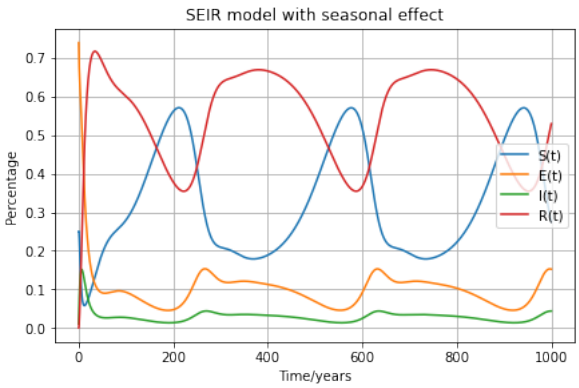

Figure 17: The SEIR model with seasonal effect, β_0=1.5 days,β_1=0.5 days $1/\gamma$=3 days,$1/\mu$=60 years, $1/\sigma$=10 days

dynamics in Figure17. We can also see the periodic variations in these oscillatory behaviors. Thus the absence of seasonal forcing generates variation in the SEIR model and we could also observe this situation in daily life.

5 CONCLUSION

We first introduce the basic building of most epidemiological models: the SIR model and discuss the dynamics of this model. We make it clear that the basic reproductive ratio R_0 is one of the most important quantities in epidemiology and a pathogen can invade only if $R_0 > 1$. We also confirm that the historical data of an influenza outbreak can be fitted into the SIR model and the epidemic could be prevented by the mass vaccination strategy.

Then we explore the SIR model with the demographic process to see the equilibrium state and oscillatory dynamics. According to our assumptions and deductions, we can calculate the frequency and amplitude of the damped oscillations when $R_0 > 1$ which could also be verified by Fourier analysis. Additionally, we take disease-induced mortality into account and find out that the high mortality leads to a large drop in the population size in both density- and frequency-dependent transmissions.

At last, we add an exposed period into the SIR model to see the variation of dynamics. The SEIR model shows similar behavior with the SIR model at equilibrium and has a slower growth rate after invasion due to the process of the exposed period before contributing to the transmission. We also add seasonality to the SEIR model to consider the consequences of seasonal changes in the transmission rate.

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