

Development of a Mathematical Model of the Spread of Chicken Pox in a Contained Population

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12 December 2018

Contents

1	Introduction	3
2	Basic SIS Model	3
3	SIS Model with Periodic Infection Rate	7
4	SIR Model with Periodic Infection Rate	9
5	SIR Model with Periodic Infection Rate and Vaccinations	11
6	SEIR Model	16
7	Conclusions	18
7.1	Summary	18
7.2	Limitations	19
7.3	Future research	19
7.4	Division of labor	20

1 Introduction

Prior to the introduction of the varicella vaccine in 1995, chicken pox was a common disease that affected approximately 4,000,000 people (mostly children) in the United States annually [1]. The widespread adoption of the vaccine has since caused a significant drop in the number of people infected each year. The goal of this paper is to develop a mathematical epidemic model that captures some of the key characteristics of chicken pox.

We begin by analyzing the most basic of the epidemic models: the SIS model. The SIS model divides the total population into a susceptible compartment and an infected/infectious compartment, between which people can move. We then proceed to modify our model to more accurately portray the behaviour of chicken pox. First, we consider an infection rate that varies periodically with time, since the spread of chicken pox has been proved to be seasonal. This is likely due to the increased number of contacts children have while in school as compared to during summer break. Next, we move on to the SIR model. This model has a compartment added in for people who have had the disease and are either recovered and immune or dead. It operates under the assumption that people who have had the disease once cannot catch it again (as is usually the case with chicken pox). We then consider the impact of vaccinations on this model. Vaccinations make it possible for people to either start out in the recovered category, or to move from the susceptible to the recovered category without moving through the infected category first. Finally, our last improvement adds in an exposed compartment, which contains people have been infected with the disease but are not infectious yet. We conclude with a discussion on the implications and the limitations of our models and provide suggestions for future research.

There are a few assumptions that hold throughout the entire paper that we will outline here. The population under consideration is well-mixed and consists of children only, since adults are unlikely to catch the disease and adult chicken pox behaves very differently. We generally introduce the disease at the start of a calendar year and track its progression over the course of 365 days. Finally, we ignore vital dynamics and assume that our given population of children does not change considerably due to births and deaths or immigration and emigration over the course of such a short time span.

2 Basic SIS Model

We begin by examining the simple SIS model (for an unspecified disease). The SIS model assumes that all individuals begin in one of two compartments: susceptible and infected [2]. Susceptible people can become infected and on recovery they are again susceptible. We are ignoring population dynamics, under the assumption that the disease spreads rapidly enough that the population under study is not varying significantly in size or composition.

Let N represent the size of the total population. We will consider proportions of N rather than numbers of people, so let $N = 1$ for simplicity. Let S be the proportion of susceptible individuals and I be the proportion of infected individuals.

Let α and β be rate constants for the model. α is the recovery rate of the disease (with $\frac{1}{\alpha}$ yielding the recovery duration). Then αI is the number of infected individuals who recover and become susceptible again per unit time. β is the infection rate of the disease. So βSI represents the number of newly infected people per unit time. This is because if an infected individual makes contact with βN people per unit time, and there's a S/N chance that person is susceptible, then

each infected individual causes $(\beta N)(S/N) = \beta S$ new cases per unit time, and in total there are βSI newly infected people per unit time.

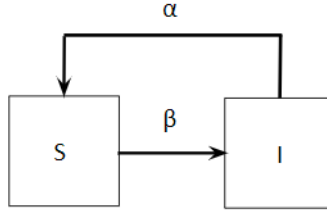


Figure 1: SIS Compartment Model

Thus, the model, as illustrated in Figure 1 above, is:

$$\frac{dS}{dt} = -\beta SI + \alpha I, \quad (1)$$

$$\frac{dI}{dt} = \beta SI - \alpha I. \quad (2)$$

The justification for the equations is as follows: Consider $\frac{dS}{dt}$, the change in the susceptible proportion per unit time. If αI individuals recover per unit time, then this group moves into susceptible. Hence this is a term that should be added into $\frac{dS}{dt}$. Similarly, if βSI individuals are infected per unit time, they move out of the susceptible compartment and into the infected. So this term should be subtracted out. The opposite of above holds for $\frac{dI}{dt}$.

Since

$$\frac{d}{dt}(S + I) = 0,$$

then

$$S + I = \text{constant} = N$$

where N is the total population.

We will now examine an analytical solution for the system of equations describing the SIS model. First, substitute $S = N - I$ into (11). This yields

$$\begin{aligned} \frac{dI}{dt} &= \beta I(N - I) - \alpha I \\ &= (\beta N - \alpha)I - \beta I^2. \end{aligned} \quad (3)$$

Now, to solve (3), let A denote $\beta N - \alpha$. Then:

$$\frac{dI}{(A - \beta I)I} = dt$$

Integrating both sides:

$$\int \frac{dI}{(A - \beta I)I} = \int -\frac{dI}{I^2(\beta - \frac{A}{I})} = \int dt$$

Let $u = \beta - \frac{A}{I}$. Then $du = \frac{A}{I^2}dI$, so:

$$\int -\frac{dI}{I^2(\beta - \frac{A}{I})} = \frac{-1}{A} \int \frac{du}{u} = \frac{-1}{A} \ln(u) = \int dt$$

Thus:

$$-\frac{\ln(\beta - \frac{A}{I})}{A} = t + c$$

Using the properties of logarithms:

$$\begin{aligned} \beta - \frac{A}{I} &= c_1 e^{-At} \\ \beta I - c_1 I e^{-At} &= A \\ I &= \frac{A}{\beta - c_1 e^{-At}} = \frac{A e^{At}}{\beta e^{At} - c_1} \end{aligned}$$

Since α, β, N, I_0 are all constants, choose c_1 such that $I(0) = I_0$, where I_0 denotes the initial infected population:

$$I(0) = I_0 = \frac{A}{\beta - c_1}$$

So $c_1 = -\frac{A - I_0 \beta}{I_0}$. Thus:

$$I(t) = \frac{(\beta N - \alpha) I_0 e^{(\beta N - \alpha)t}}{\beta I_0 e^{(\beta N - \alpha)t} - \beta I_0 + (\beta N + \alpha)} \quad (4)$$

Now, since $S + I = N$, we have

$$S(t) = N - \frac{(\beta N - \alpha) I_0 e^{(\beta N - \alpha)t}}{\beta I_0 e^{(\beta N - \alpha)t} - \beta I_0 + (\beta N + \alpha)} \quad (5)$$

Solving $\frac{dI}{dt} = (\beta S - \alpha)I = 0$ and $\frac{dS}{dt} = (\beta - \alpha S)I = 0$, we find that the system has two equilibrium points, one at which $S = \frac{\alpha}{\beta}$ and one at which $I = 0$. Using $N = S + I$, the system's two equilibrium points are $(\frac{\alpha}{\beta}, N - \frac{\alpha}{\beta})$ and $(N, 0)$. The existence of the former point only makes biological sense if $\frac{\alpha}{\beta} < N$, since the infected population cannot be negative.

Consider the case in which $\frac{\alpha}{\beta} < N$. When $S < \frac{\alpha}{\beta}$, $S' = (\beta - \alpha S)I > 0$ and $I' = (\beta S - \alpha)I < 0$. When $S > \frac{\alpha}{\beta}$, $S' = (\beta - \alpha S)I < 0$ and $I' = (\beta S - \alpha)I > 0$, so $(\frac{\alpha}{\beta}, N - \frac{\alpha}{\beta})$ is stable. It follows that $(N, 0)$ is unstable.

Thus, for the case in which $\frac{\alpha}{\beta} < N$, the phase plot with equilibrium points (illustrating that the sum of S and I is constant) is as follows in Figure 2:

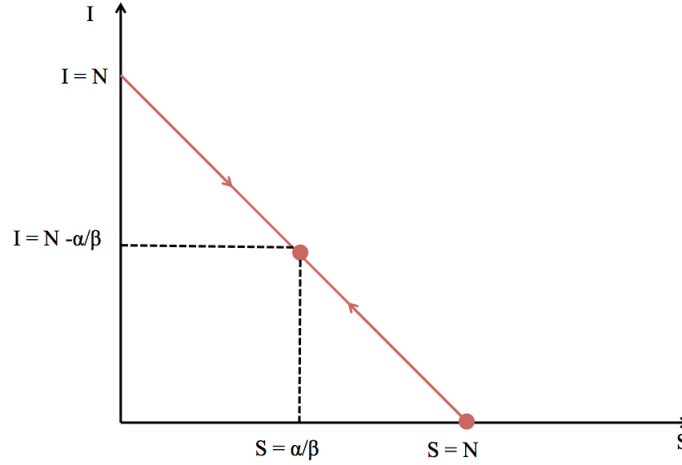


Figure 2: Phase Plot of $S(t)$ against $I(t)$ for $\frac{\alpha}{\beta} < N$

Consider now the case in which $\frac{\alpha}{\beta} > N$. The point $(\frac{\alpha}{\beta}, N - \frac{\alpha}{\beta})$ is not in the first (and only biologically feasible) quadrant. The point S must always be less than $\frac{\alpha}{\beta}$, so $S' > 0$ and $I' < 0$ for all biologically feasible values of S and I , making $(N, 0)$ stable.

Thus, for the case in which $\frac{\alpha}{\beta} < N$, the phase plot with equilibrium points is as follows in Figure 3:

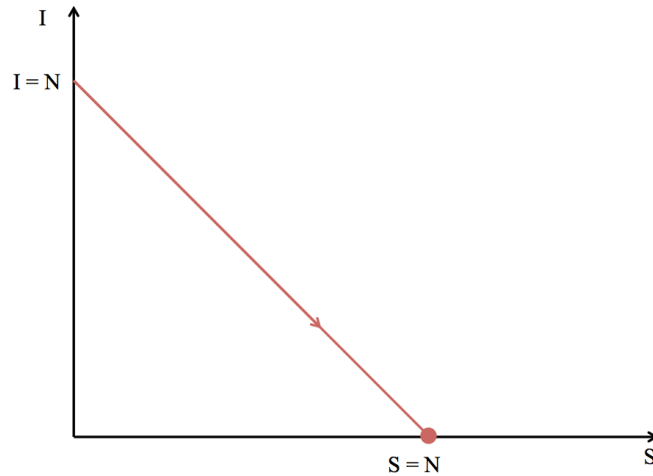


Figure 3: Phase Plot of $S(t)$ against $I(t)$ for $\frac{\alpha}{\beta} > N$

The direction field for this system with randomly chosen parameter values is as shown in Figure 4:

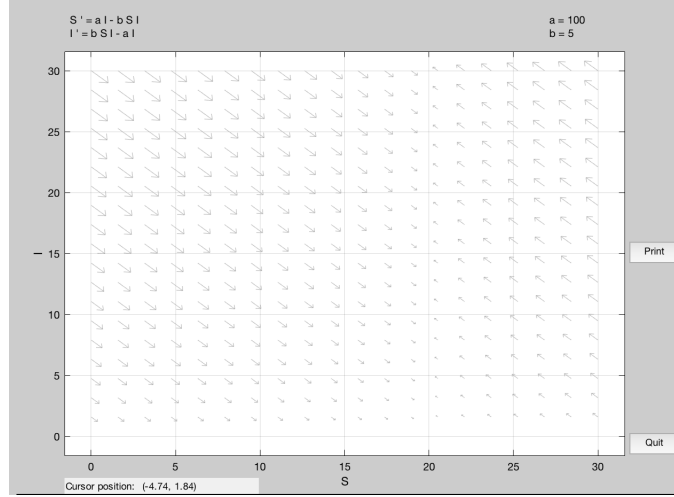


Figure 4: Direction field for SIS model

The implication of this fixed point analysis is that if $\frac{\alpha}{\beta} < N$, the infection rate is high enough relative to the recovery rate so that the disease will spread and approach an endemic steady-state. If $\frac{\alpha}{\beta} > N$, the recovery rate is high enough relative to the infection rate that the population will approach a disease-free steady state.

3 SIS Model with Periodic Infection Rate

As an extension of the SIS model, we consider an SIS model with a periodic infection rate $\beta(t)$ with period p . (We are still considering an unspecified disease, but making the infection rate periodic makes this new model more reasonable for a seasonal disease like the flu or common cold). In this model, all parameters and equations are defined as in the previous section, except that β is a periodic time-dependent function instead of a constant. Given a periodic infection rate, it is reasonable to expect that I and S will vary periodically with time. As an example, we will consider a periodic function

$$\beta(t) = 1 - 0.8\cos(5t) \quad (6)$$

Depending on the values of α and β , two phenomena can be seen: asymptotically periodic behavior in I and S or I asymptotically approaching 0. It turns out there is a threshold value \bar{p} , at which behavior switches between these two phenomena, depending on if N is greater or less than \bar{p} . Hethcote defines this parameter to be $\bar{p} = \frac{\alpha}{\beta}$ where

$$\bar{\beta} = \frac{1}{p} \int_0^p \beta(u) du$$

and determines that if $N > \bar{p}$, the asymptotic behavior is periodic and if $N \leq \bar{p}$, I approaches 0 asymptotically [3]. For equation 6, $\bar{\beta} = 1$.

Below are two plots demonstrating the proportion of susceptible and infected versus for different α values. In Figure 5, $\alpha = 0.5$ so $\bar{p} = 0.5$ and $N > \bar{p}$. In Figure 6, $\alpha = 2$, so $\bar{p} = 2$ and $N < \bar{p}$.

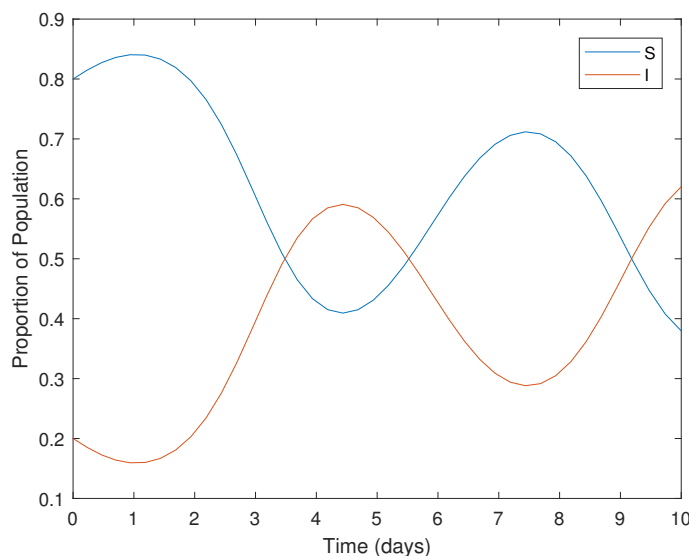


Figure 5: SIS compartment model with $N < \bar{p}$

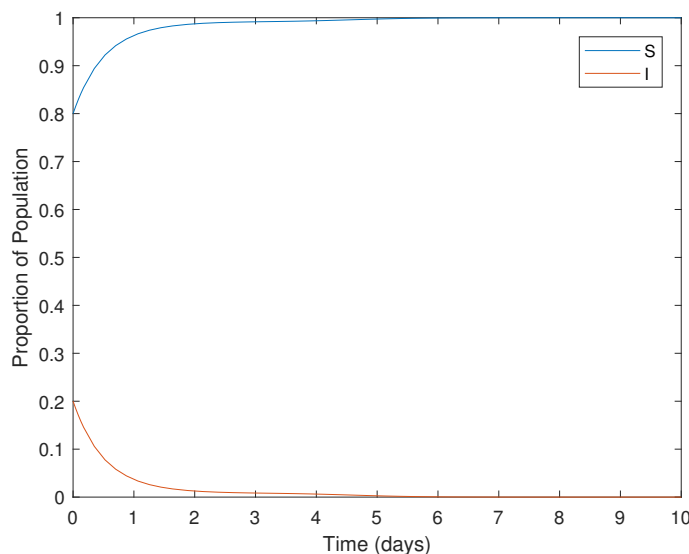


Figure 6: SIS compartment model with $N > \bar{p}$

The implication is that if the recovery rate α is high enough compared to the infection rate $\beta(t)$ over a period, then the population approaches a disease-free steady state (as in Figure 6). If not, the proportions of susceptible and infected people will vary periodically with time as $\beta(t)$ varies (as in Figure 5).

4 SIR Model with Periodic Infection Rate

The overall goal of this paper is to model the epidemiology of chicken pox. While an SIS model with a time-varying infection rate can provide useful information about diseases that can be caught multiple times and that have periods of varying infectiousness (such as the common cold), chicken pox has a fundamental difference. Generally, people do not get chicken pox more than once. So we are going to make our model more realistic: consider an SIR model, where individuals go from being susceptible, to infected, to recovered. Therefore, there are now three compartments for this model, as shown in Figure 7.

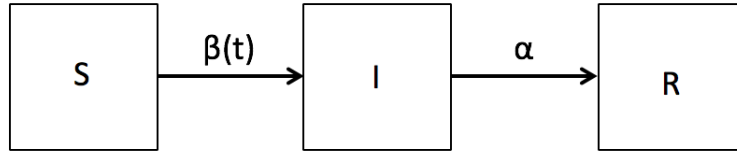


Figure 7: SIR compartment model with time-dependent infection rate

When an individual is recovered, they are no longer susceptible to the chicken pox, so an individual cannot be infected more than once. The governing equations for the SIR model are:

$$\frac{dS}{dt} = -\beta(t)SI, \quad (7)$$

$$\frac{dI}{dt} = \beta(t)SI - \alpha I, \quad (8)$$

$$\frac{dR}{dt} = \alpha I. \quad (9)$$

Note that this model is the same as the Kermack-McKendrick model, but with a time dependent infection rate $\beta(t)$, which as before is of the form $\beta_0 + \beta_1(\cos(\frac{2\pi}{365}t))$, i.e. a symmetric periodic infection rate with a period of 365 days (1 year). Now, β is the product of the number of contacts a susceptible person has in a day and the transmission probability per contact. The transmission probability for chicken pox is approximately 0.09 [4]. This appears very high, but according to the CDC, chicken pox is highly infectious and a susceptible person has a 90 percent chance of catching it if someone in their household gets it [1]. So, a 9 percent transmission probability per contact is not unreasonable. Let's assume that the contact rate peaks at 32 children per day at the beginning and end of a year, and that it goes down to 2 children per day in the middle of the summer (a child might come into contact with a full class and a sports team regularly during the school year, but only with their siblings in the summer). Thus, $\beta(t)$ should vary between 0.18 and 2.88. As such, we set β_0 to 1.53 and β_1 to 1.35, so

$$\beta(t) = 1.53 + 1.35 \cos\left(\frac{2\pi}{365}t\right).$$

The recovery rate α , is the reciprocal of the infectious period, μ . For chicken pox,

$\mu = 15$ days, so $\alpha = \frac{1}{15}$. Using these parameters, we solve the system numerically (Figure 8). The infected proportion of the population spikes quickly, but in the long run everyone moves into the recovered compartment, as expected.

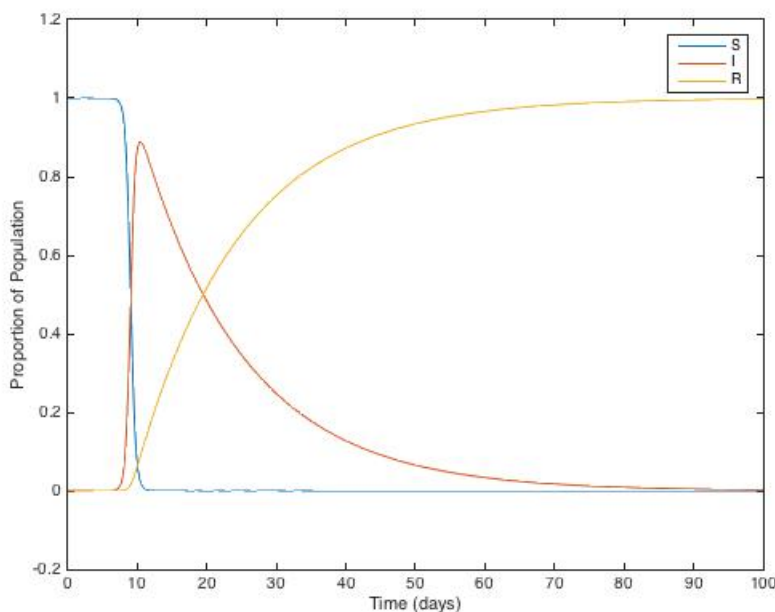


Figure 8: SIR model

We can also solve for $S(t)$ analytically. Observe that

$$\frac{dS}{dt} \frac{dt}{dR} = \frac{dS}{dR} = -\frac{\beta(t)SI}{\alpha I}.$$

Rearranging and integrating both sides, we get

$$\int \frac{1}{S} dS = \int -\frac{\beta(t)}{\alpha} dR,$$

thus

$$\ln(S) = -\frac{\beta(t)}{\alpha} R + c$$

where c is some constant. Solving for S , we get

$$S(t) = ce^{-\frac{\beta(t)}{\alpha} R(t)}.$$

Then, using initial conditions,

$$S(0) = S_0 = ce^0 = c \Rightarrow c = S_0$$

assuming that $R(0) = 0$. Thus,

$$S(t) = S_0 e^{-\frac{\beta(t)}{\alpha} R(t)}.$$

Comparing the ODE45 solution with this analytical solution (using Figure 9 below), we see that the solutions are the same, indicating that our numerical solution is sound.

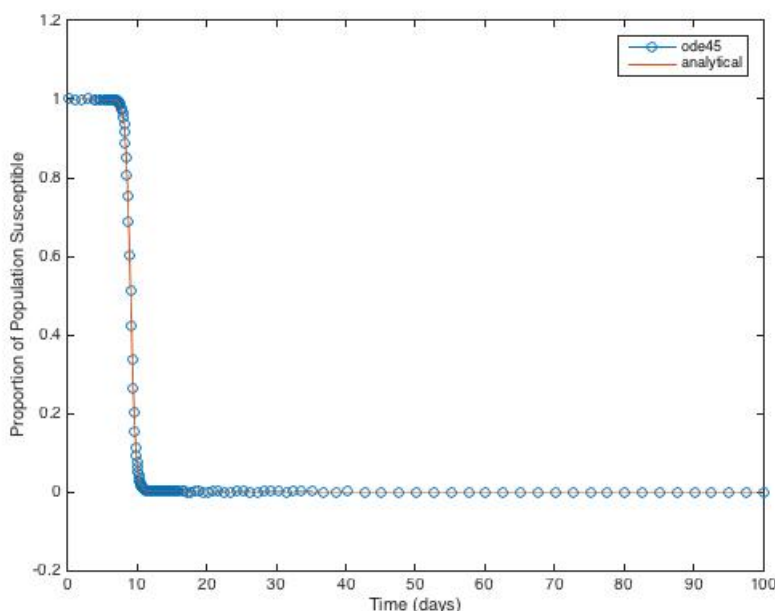


Figure 9: Comparison of ODE45 and analytical solutions for $S(t)$

5 SIR Model with Periodic Infection Rate and Vaccinations

In this section, we will consider the influence of vaccinations on our previous model, since there has been a chicken pox vaccine available since 1995, making the disease much less widespread [1].

The compartment model for this case, shown in Figure 10, is similar to the previous one, except that each day a certain proportion of the susceptible population moves straight to the recovered population (without first becoming part of the infected population) by receiving a vaccine. Here, γ represents the vaccination rate, which we will assume for now is constant. We can recover our previous model by setting $\gamma = 0$.

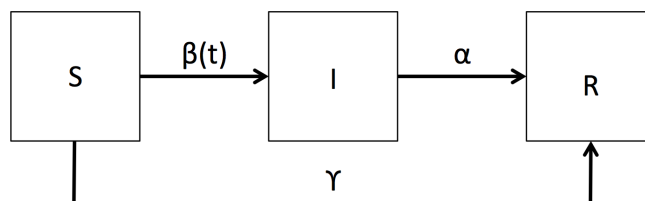


Figure 10: SIR compartment model with time-dependent infection rate and vaccinations

The governing equations for this model are:

$$\frac{dS}{dt} = -\beta SI - \gamma S, \quad (10)$$

$$\frac{dI}{dt} = \beta SI - \alpha I, \quad (11)$$

$$\frac{dR}{dt} = \alpha I + \gamma S. \quad (12)$$

As before, $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$, so the total population, denoted N , is constant. (Again, we are not considering the effects of natural birth, death, immigration, or emigration rates.) We are still considering $\beta(t)$ of the form $\beta_0 + \beta_1(\cos(\frac{2\pi}{365}t))$, i.e. a symmetric periodic infection rate with a period of 1 year, and the recovery rate, α , is still the reciprocal of the infectious period. As discussed in Section 4, we are setting α to $\frac{1}{15} \approx 0.06667$, β_0 to 1.53, and β_1 to 1.35.

In the cases considered below, we used initial conditions $I_0 = 10^{-10}$, $R_0 = 0$, and $S_0 = 1 - I_0 - R_0$. These initial conditions indicate that to begin with, no one is vaccinated and a very small portion of the population is infected.

Figure 11 shows the resulting behaviour of $S(t)$, $I(t)$ and $R(t)$ when γ is set to 0.01. In this case, the dynamics of the disease are very similar to those in the model in the previous section, since γ is small compared to α and β .

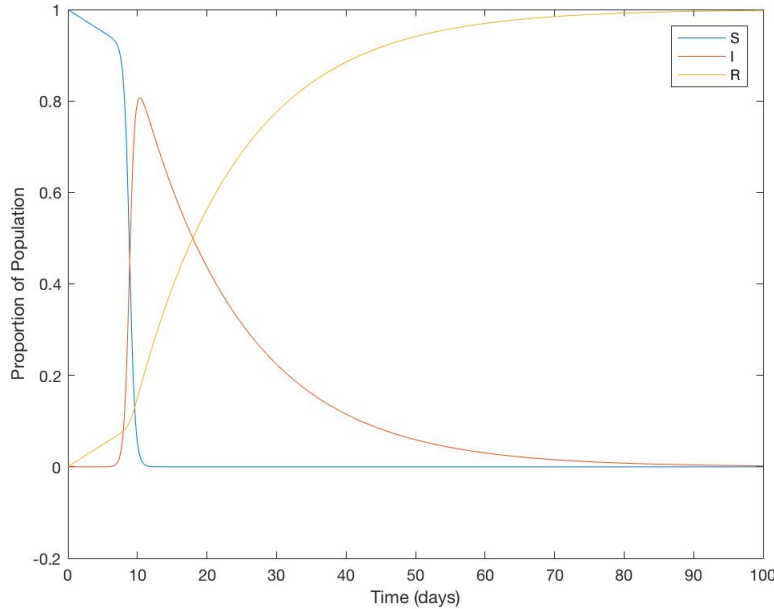


Figure 11: $S(t)$, $I(t)$, and $R(t)$ for $\gamma = 0.01$

Figure 12 shows the resulting behaviour of $S(t)$, $I(t)$ and $R(t)$ when γ is set to 0.1:

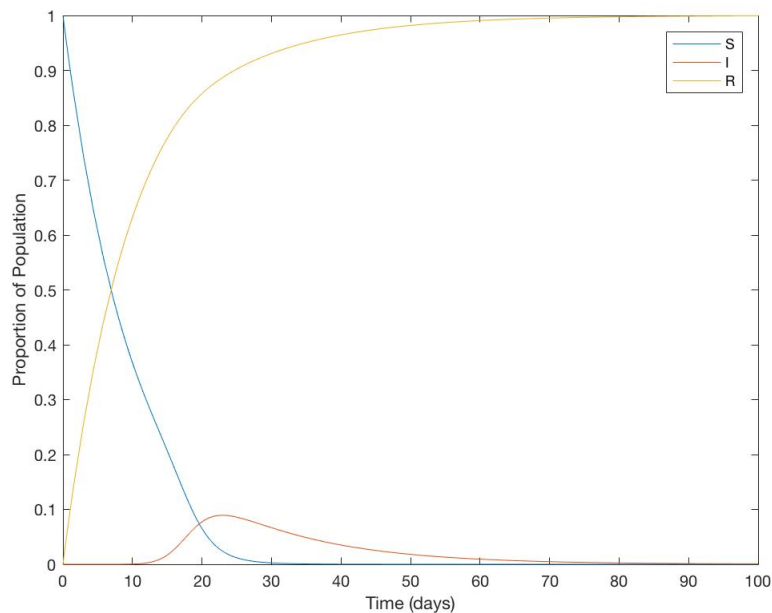


Figure 12: $S(t)$, $I(t)$, and $R(t)$ for $\gamma = 0.1$

Figure 13 shows the resulting behaviour of $S(t)$, $I(t)$ and $R(t)$ when γ is set to 0.3:

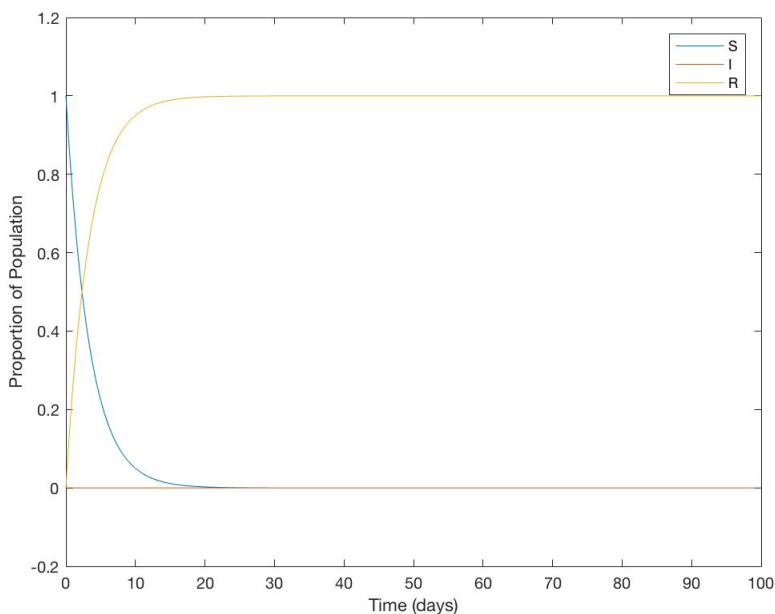


Figure 13: $S(t)$, $I(t)$, and $R(t)$ for $\gamma = 0.3$

As these figures illustrate, if γ is above a certain critical value γ_c , the disease does not spread beyond a small proportion of the population according to this model. Figure 14 shows the maximum

value of $I(t)$ plotted against γ , with γ going from 0 to 1 in intervals of 0.01.

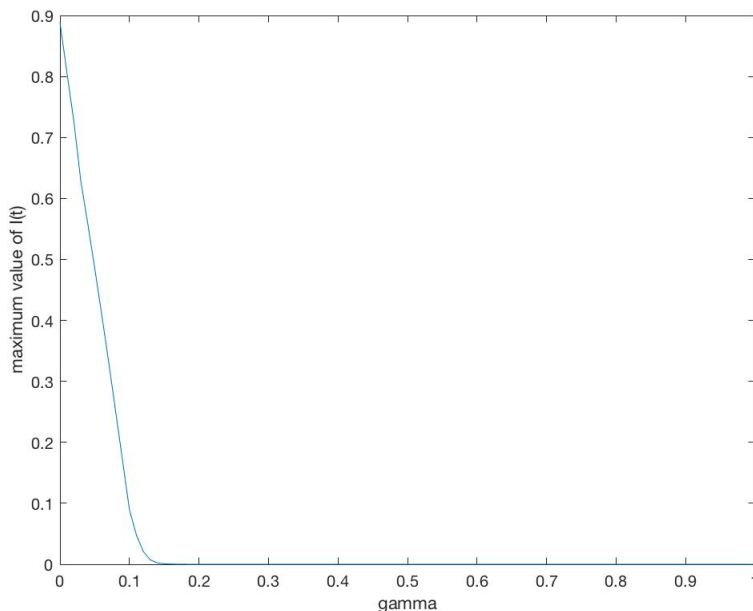
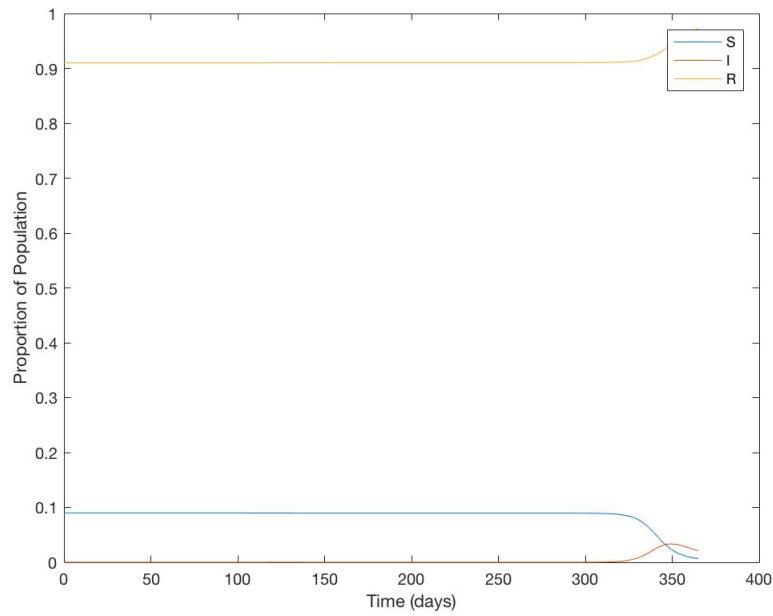
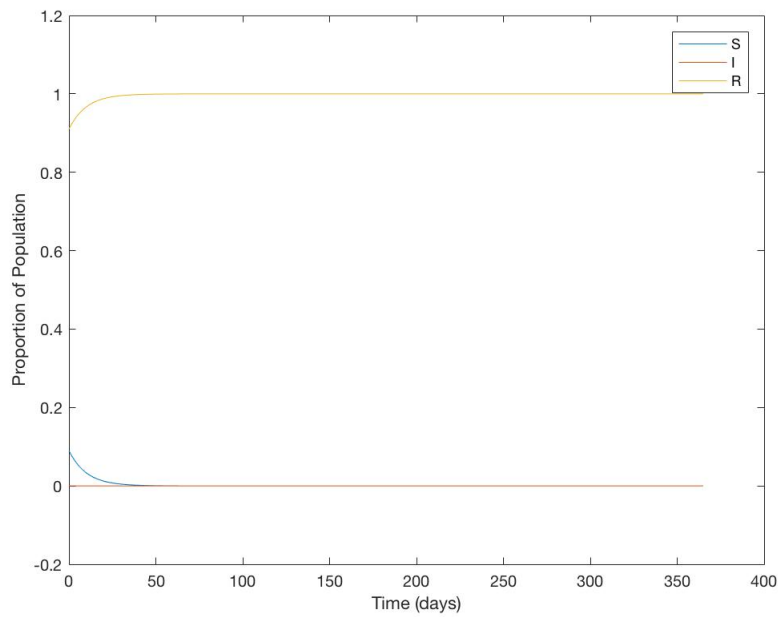


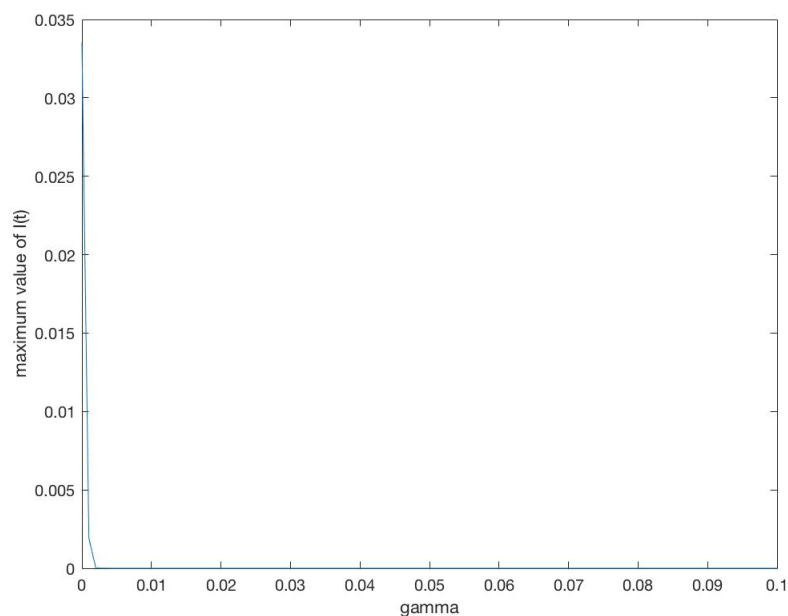
Figure 14: the maximum value of $I(t)$ against γ

Thus, if we define “small” as 10^{-5} , we can numerically determine that $\gamma_c = 0.22$ for these given parameters. That is, for the given parameters and for this model, if γ is at least 0.22, the proportion of the population in the Infected compartment is never above 10^{-5} .

The above analysis, with the initial value of $R(t)$ set to 0, may provide some interesting insight into the behavior of chicken pox back when the vaccine was first introduced, but it does not accurately model conditions today, since most children in the US are now vaccinated very early in life. According to the CDC, approximately 91 percent of American toddlers are vaccinated against chicken pox [1]. Thus, we now set $R_0 = 0.91$, to model the case in which chicken pox is introduced into a population of children of which 91 percent are already in the Recovered category.

On the next page, Figure 15 shows the dynamics over the course of 1 year for this new initial condition, with $\gamma = 0$ (i.e. 91% of the population is vaccinated to begin with but no one gets vaccinated after the disease is introduced). Figure 16 shows the dynamics over the course of 1 year for this new initial condition with $\gamma = 0.1$. Finally, Figure 17 shows how the maximum value of $I(t)$ changes as γ is varied from 0 to 0.1 (still with $R_0 = 0.91$). These Figures illustrate that according to this model, if 91 percent of the population is vaccinated to begin with, a small vaccination rate (approximately 0.25 percent of the population per day) is needed to prevent an outbreak altogether.

Figure 15: $S(t)$, $I(t)$, and $R(t)$ for $\gamma = 0$ and $R_0 = 0.91$ Figure 16: $S(t)$, $I(t)$, and $R(t)$ for $\gamma = 0.1$ and $R_0 = 0.91$

Figure 17: the maximum value of $I(t)$ against γ with $R_0 = .91$

6 SEIR Model

Many diseases, including chicken pox, have an incubation period, during which people have caught the disease but are not yet infectious. In order to include this aspect of chicken pox in our model, we now add a new compartment E for exposed members of the population, who are assumed to be infected but not yet infectious [5]. Now we introduce a new variable, σ , which is the rate at which exposed individuals become infectious. Thus, the model is as shown in Figure 18 below:

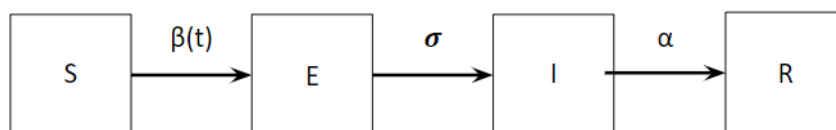


Figure 18: SEIR compartment model with periodic infection rate (without vaccination rate)

The governing equations for the model are:

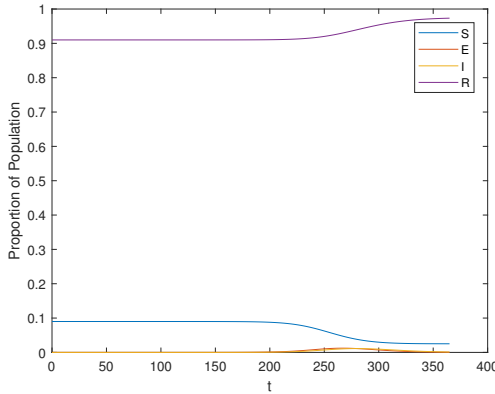
$$\frac{dS}{dt} = -\beta SI, \quad (13)$$

$$\frac{dE}{dt} = \beta SI - \sigma E, \quad (14)$$

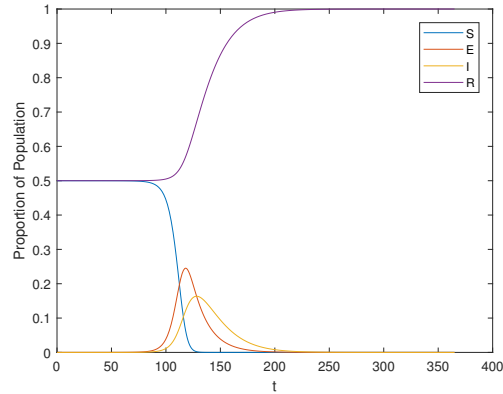
$$\frac{dI}{dt} = \sigma E - \alpha I, \quad (15)$$

$$\frac{dR}{dt} = \alpha I. \quad (16)$$

We assume that the contact rate and recovery rate are the same as in the previous models. The average incubation period for chicken pox is approximately 15 days [1] so we set $\sigma = \frac{1}{15} \approx 0.06667$. We consider vaccines by varying by varying R_0 (i.e. the initial number of vaccinated individuals). For simplicity's sake, we are not including the γ parameter from our previous model, under the simplifying assumption that people do not continue to get vaccinated over the course of the year in which the disease is introduced (i.e. no one moves directly from S to R). In the two cases considered below, we used two different initial conditions for R ($R_0 = 0.5$ and $R_0 = 0.91$). In both cases, we used initial conditions $S_0 = 1 - E_0 - I_0 - R_0$, $E_0 = 0$, and $I_0 = 10^{-5}$, indicating that to begin with, the disease is introduced to a very small proportion of the population, while no one is exposed and most people are susceptible. The lower R_0 corresponds to a lower proportion of the population being vaccinated and vice versa for the higher R_0 . Figure 19a below shows dynamics for a chicken pox outbreak with an exposed compartment with $R_0 = 0.91$. Because such a high percentage of the population is vaccinated, no one really gets sick with the disease and it eventually disappears. Figure 19b shows the dynamics when $R_0 = 0.5$. For this much lower initial vaccinated proportion, there are enough susceptible people to create an epidemic before the disease dies out over time.



(a) $S(t)$, $I(t)$, $E(t)$, and $R(t)$ for $R_0 = 0.91$



(b) $S(t)$, $I(t)$, $E(t)$, and $R(t)$ for $R_0 = 0.5$

Figure 19: Population dynamics for two different vaccination proportions

As Figure 19b illustrates, the main activity of the outbreak does not occur until a third of the way into the year, because the incubation period causes delays the spread of the disease. (Under these particular conditions, the disease is first introduced to the population at the beginning of the year, when $\beta(t)$ is at its maximum, and since $\beta(t)$ is continually decreasing throughout the first half of the year, the spike in infected people is less prominent when it does occur than in our previous models).

Clearly, as shown by the above Figures, there is a threshold value for the proportion of vaccinated individuals, past which the disease does not spread beyond a small proportion of the population. Figure 20 shows how $\max I(t)$ varies with R_0 , illustrating that according to this model, approximately 91 percent of the population needs to be vaccinated to keep the disease from spreading. This

is reasonable, since it has empirically been determined that the herd immunity value for chicken pox is between 86 and 91 percent [6].

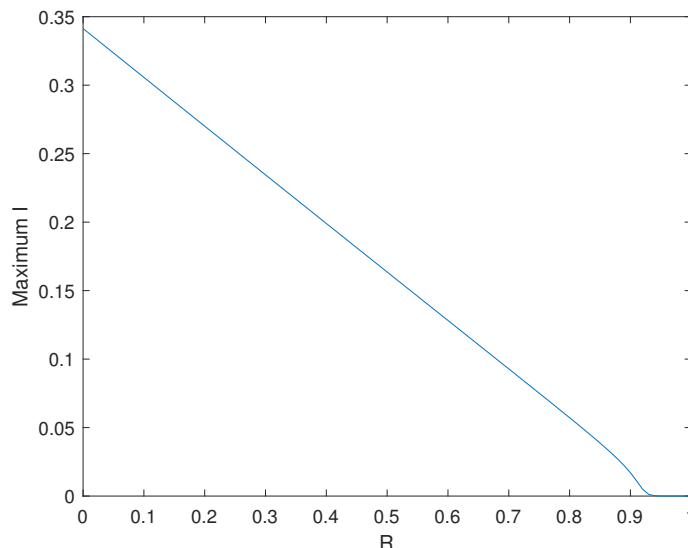


Figure 20: Maximum $I(t)$ for varying R_o

7 Conclusions

7.1 Summary

We started with the simplest model for disease modeling for a general disease and gradually made our model more sophisticated and picked a specific disease, chicken pox, to model. Throughout the project, we ignored the effects of immigration, births, and natural deaths (i.e. our total population stayed constant), and we assumed our population was well mixed.

We began with the SIS model for a general disease, which has two compartments, S and I . Individuals begin as susceptible, move to the infected compartment, and on recovery return to the susceptible compartment. This very simple SIS model assumes that the infection rate is constant; however, this is not the case for chicken pox, whose infection rate depends on time. Since chicken pox is most common in school children, the chicken pox rate of transmission is higher during the school year than during the summer. Therefore, our next model was SIS with a time dependent infection rate $\beta(t)$.

Note that chicken pox is still not well described by an SIS model, since most individuals will only get chicken pox once in their lives. We then considered an SIR model which has an additional third compartment, R for recovered. Individuals move from the susceptible compartment, to infected, and finally to recovered. Recovered individuals are immune and do not return to susceptible. As before, we had a time dependent $\beta(t)$.

Since there exists a vaccine for chicken pox, our next step was to construct a SIR model with periodic infection rate and with vaccinations. With vaccinations, individuals may start in the recovered compartment or go directly from the susceptible compartment to recovered, since vaccinated individuals will not contract chicken pox. We modeled this in two different ways: one where

initially no one is vaccinated (perhaps the vaccine has only just been invented), and one where initially some percentage of the population is already vaccinated (more accurate to today).

Finally, since chicken pox has an incubation period, we created a SEIR model with periodic infection rate. This added a fourth compartment to the model, E , for exposed. Individuals move from the susceptible compartment, to exposed, to infected, and then to recovered. The exposed compartment represents the fifteen day incubation period, the time during which an individual has contracted chicken pox, is not contagious, and shows no symptoms. We set different initial conditions for the proportion of the population that had been administered the vaccine and demonstrated the existence of a threshold value for herd immunity. Any vaccination proportion below this threshold value leads to an epidemic.

The main takeaway from this project is that vaccines are an excellent way to prevent many people from contracting an infectious disease such as chicken pox. According to our final model, with a population that is at least initially 91% vaccinated (typical for the U.S.), herd immunity will protect a population from an outbreak of chicken pox. However, allowing the percentage of vaccinated individuals to decrease from this value would enable chicken pox to spread widely once it is introduced into a population.

7.2 Limitations

Throughout the project, we assumed that the population is well mixed. Depending on the population under consideration, this may not be true. For example, assuming a well mixed population would work for studying one school's population but would not work well for studying a population of children that is geographically very separated.

We also assumed that any individual's chance of infection was equal, i.e. that no susceptible person was more likely to get chicken pox than any other susceptible person. In reality, this is not true, since some people's immune systems are stronger than others. Similarly, we assumed that people get better at the same rate, which is also untrue, since this rate may vary across socioeconomic groups due to differing degrees of access to medical care. We also did not consider the impact of awareness on our model: in reality, people may consciously decrease the number of contacts they have per day if they know an infectious disease has been introduced into their population.

Finally, we ignored population dynamics by assuming that total population stayed constant. In reality, the population should vary somewhat over time with the one year time scale we considered.

7.3 Future research

Future analysis should address the limitations outlined above. In particular, social network dynamics could be introduced to fix the problem with the assumption of a well mixed population. The introduction of some kind of random variable could help fix the assumption that all individuals get infected and recover at the same rate, since then the number of individuals moving from susceptible to infected (or exposed) and from infected to recovered would better describe the complexities of a real human population. Varying the contact rate over the course of the epidemic could account for the influence of awareness, and adding terms for immigration and emigration rates to each of the equations would better account for population dynamics.

To further improve our model, more data and research would be needed to help understand where our assumptions might be wrong. We know chicken pox is highly contagious, and one disease

modeling paper gave the chance of transmission at one contact to be 9% [4], which is the number we used in our analysis. This does seem high, however, so perhaps further research could verify this or give a more accurate rate. In addition, it would be good to compare the results of our model with recorded data to see how well we described the epidemiology of chicken pox. We also only considered chicken pox in the United States. It's possible that in other countries, the transmission rate might be different due to different health/sanitary practices. The initial condition for percent of population already vaccinated may be different. The social dynamics, like number of people an individual may come into contact with, may be different. Research would be needed to understand what the new initial conditions or rates should be in other countries.

In order to test our model, we would need data about how chicken pox spreads when it is introduced into a population (for example, a school). We would want to know who got sick to begin with and when, how many people caught the disease, who they caught it from, and how many people got sick/recovered per day. We would need a wide variety of data across socio-economic and geographic groups, since parameters like the recovery rate can depend on these factors. We would use this data both to improve the accuracy of our parameters, and to test the validity of our model and its assumptions.

7.4 Division of labor

Katherine

- SIS (analytical solution)
- SIR with periodic transmission rate
- Conclusion

Nina

- Introduction
- SIS (phase plot and fixed point analysis)
- SIR with periodic transmission rate and vaccinations

Dan

- SIS (numerical solution)
- SIS with periodic transmission rate
- SEIR with periodic transmission rate and vaccinations

All

- Overall proofreading
- Construction of model and deciding which improvements to make

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