On Delayed Differential Equations and Epidemiological Models

by

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STATEMENT BY AUTHOR

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Shwetabh Singh

DECLARATION

I hereby declare that the written work presented here is original and has been carried out by me. All sources used in this investigation have been appropriately referenced. This thesis has not been submitted previously at this University or any other, in part or in its entirety, for the fulfillment of the requirements of a degree or diploma.

Shwetabh Singh

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Contents

Chapter 1

Introduction

In the December of 2019, the first cases of a mysterious new variant of SARS CoV-1, now dubbed SARS CoV-2, were being observed. Since then the virus has spread throughout the world, due to its highly contagious nature and has resulted in a pandemic unseen in last 100 years. The outbreak has been declared a pandemic by the WHO and countries have mobilised all and every resources to counter the pandemic.

The transmission of COVID-19 happens primarily through air, and generally when someone is in close proximity of an infected person. Countries have tried various methods, from social distancing, masking to full fledged total lockdowns, to counter the early spread of the disease. Although initially these were successful in some parts of the world to halt the onslaught of the disease, the economic cost had been enormous, and hence lockdowns had to be eased, which were seen by the governments as the most effective solution to buy time till the capabilities to handle the disease built up.

It was due to this great social and scientific context, that I decided to study the epidemiological models. The initial observation of combining delayed differential equations and SIR epidemiological model to successfully model COVID epidemic in Italy was done by Dell'Anna. The SIR model although in its original form is very simplistic to model an epidemic as big as this., with a lot of more variables and constraints than that could be included. Through my investigation I wished to primarily build further on his work and increase the sophistication of delayed SIR models, in an attempt to bring the model closer to reality.

1.1 Delayed Differential Equations

A Delayed Differential Equation is simply a differential equation in which time derivative of a function at the current time depends on its value or its derivatives at some previous time, mathematically

$$\dot{x} = F(t, x(t), x(t-\tau_1), x(t-\tau_2)...x(t-\tau_n), \dot{x}(t-\sigma_1), \dot{x}(t-\sigma_2)...\dot{x}(t-\sigma_m)); t \geq t_0 \quad (1.1)$$

$$x(t) = \phi(t); t \leq t_0 \quad (1.2)$$

So to solve such equations, instead of a simple initial condition we require a history function $\phi(t)$. The τ_is and σ_js are the time delays in the delay differential equations. There can multiple types of delay, whether constant delays in form of static values of τ_i or even a function $\tau(t, x(t))$. DDEs have a lot of applications especially in life sciences, where events have a memory and don't react immediately. Examples are afforded by population dynamics, epidemiology, immunology, physiology, neural networks etc. We will be using a one such model of epidemiology to further study the implication of DDEs. As of now, we'll be focusing on implementing and working with fixed delay problems and their applications to models.

1.2 SIR Epidemic Model

Considering the global environment, the most logical choice of a model to study was a model of epidemics and understand its implications.

SIR, or (S)usceptible, (I)nfected and (R)ecovered model was chosen as the foundation with the aim to further complicate it incorporating further sophistication and exploring delay in those models. The basic model works by compartmentalising any given population in three separate groups and was first described by Kermack and McKendrick in 1927. Further complications of the model can include various characteristics of the disease, like fatality, short term immunity but no fatality, incubation time etc. Before we move further we must clarify the definitions of the compartments,

Susceptible - People who haven't either been infected with the disease yet, or are simply just susceptible to it.

Infected - People who catch the disease and now act as carriers and further spreaders of the disease to the susceptible individuals.

Recovered - People who have recovered from the disease. In the most basic SIR model, recoveries are assumed to include both living recovered population and, if applicable, any fatalities too.

An immediate addition to the model can be to include death separately, and which is called the SIRD model. This model seems like very elementary upgrade over the SIR model, but it is important if we are to include further upgrades to the model like temporary immunity to the epidemic, making living recovered people re-susceptible to the disease, there the separation of dead and recovered becomes important. The temporary immunity model can be described by SIRS model.

The family of models involves rate equations, and hence are first order, non-linear, ordinary differential equations. The model although pretty simplistic carries some subtle insights. The model is derived under some strong assumptions, having a large and closed population, the outbreak being short lived, homogeneity of population across space and density, equal likelihood of getting infected across various lines like age, sex, previous health conditions, equal probability of any person meeting an infected person, no natural births or natural deaths occur, the infection having a zero latency period. The assumption of Zero latency is the one that would be challenged the most through out this investigation.

Chapter 2

Epidemiological Models

2.1 SIR Model

The basic SIR model is described by three first order, non-linear, ordinary differential equations. The model follows a simple pathway of a susceptible population becoming infected and then recovering from it.

Figure 1 : A block diagram of SIR model

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\frac{\beta}{N}S(t)I(t) \tag{2.1}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\beta}{N}S(t)I(t) - \gamma I(t) \tag{2.2}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I(t) \tag{2.3}$$

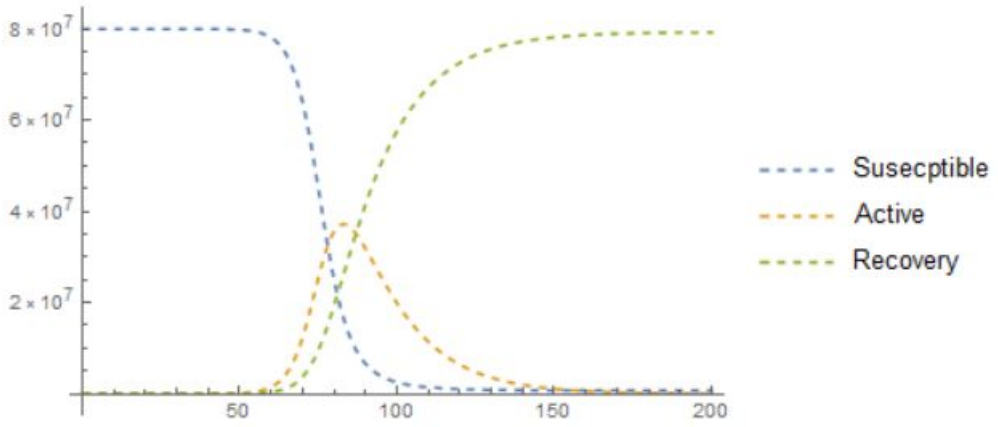

Figure 2 : A plot describing the three compartments of SIR model

The above model is an attempt to fix COVID like parameters in SIR model for a model population of 80 million people, which is the population of Germany. The real world datasets for countries can be sourced online through various research resource centers online, like Covid-19 Data Repository by the Center for Systems Science and Engineering (CSSE) at Johns Hopkins University.

The rate of infection or the average number of contacts of an infected person per unit time or $\beta = 0.3$ here and $\gamma^{-1} = \mathrm{T} = 16$ units of time. The parameters have remained constant throughout the study as our focus is on the delay variable not the parameters themselves[Dell'Anna, 2020]. The initial conditions for the set equations are S(0) = 80 x 10^6, I(0) = 150, and R(0) = 0, I(t) = e^t for t$\leq \tau$. These conditions carry-on for all the models [Dell'Anna, 2020].

The model although simple has some underlying assumptions. The model assumes the population of the model to be static *ie* $S' + I' + R' = 0$. The model due to being described by rate equations assumes the encounters between infected and susceptible compartments occurs at a rate proportional to their current population. Infected people are assumed to recover with a constant probability and each susceptible individual is equally probable to become infected at any time. The model is a deterministic one due to being described by ordinary differential equations and hence can't capture the enormous sophistication of a real epidemic which is, on many aspects, stochastic in nature.

2.2 Introducing Delay

The SIR model requires the presence of a recovery rate related to the number of recoveries without considering new recoveries actually come from the number of infected people previously. So we can actually propose a model where the recovered cases come from infected cases after an average recovery time delay. We also know COVID has an average recovery period of 14 days. This can incorporated in the simple SIR model by introducing a simple retardation of this period in form of a static delay[Dell'Anna, 2020]. We can rewrite the equations of SIR model including this detail as

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\frac{\beta}{N}S(t)I(t) \tag{2.4}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\beta}{N}S(t)I(t) - \frac{\beta}{N}S(t-\tau)I(t-\tau) \tag{2.5}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \frac{\beta}{N}S(t-\tau)I(t-\tau) \tag{2.6}$$

We can solve these equations numerically. **NDSolve** in Mathematica was used to solve these equations numerically with delay. The historical solution for the Infected variable is taken ($I(t) = e^t; t \leq 0$ is the ansatz) to be an exponential function. The solutions are plotted below.

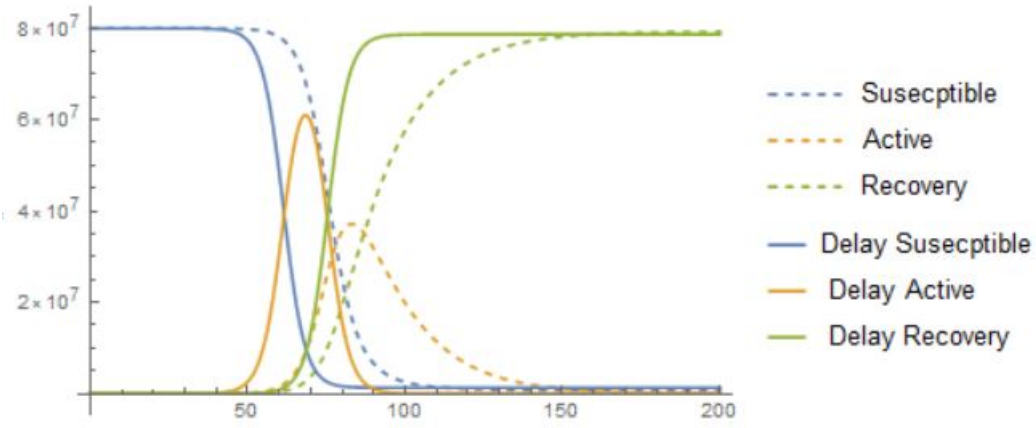

Figure 3 : A plot comparing SIR model with delayed SIR model

A comparison of both models immediately reveals the difference a simple retardation of 14 days causes in all the compartments.

Certain features are highlighted due to the introduction of delay in the model. We observe that since a single individual is now infected with the disease for a longer

period and hence can act as an agent for a longer duration, we see an earlier decline in the susceptible population and a much steeper infected curve. The peak of infected curve is much higher than the without delay model, reaching as high as 3/4 of population at the time. What is also interesting that we can observe a much earlier and steeper recovery for the closed population too. This suggests that the vanilla SIR model would underestimate an epidemic (Fig 3) compared to the delayed variant and the herd immunity threshold (max infections after which infections decline and recoveries increase) occurs faster, although higher, in a model with delay than one without[Dell'Anna, 2020].

Again, although the model is too simplistic to make real world policy, we can indeed think theoretically of different ways to mitigate such high infected population for the short period of time it exists, like lockdowns cutting the spread by a large infected population. We need to flatten the curve.

2.3 SIRD Model

Another logical upgrade to the model would be to introduce fatality (as observed in COVID too), to separate the recovered from the dead. Although since our model has an inherent permanent immunity assumption built in, but this step is necessary, as it will be required when the complexity of the model will be increased by introducing temporary immunity, *ie* Recovered people become susceptible after a certain amount of time. Although in this model, we do include deaths of the population, we do not consider any births and the total number of people in all compartments remains constant (S+I+R+D = N).

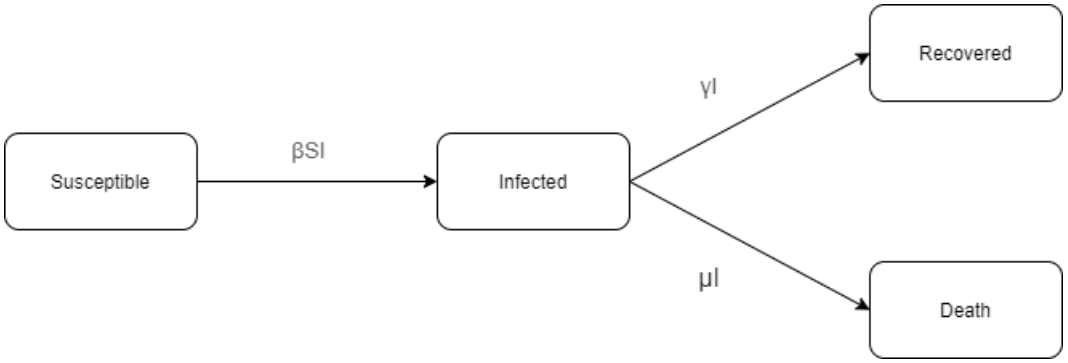

Figure 4 : A block diagram of SIRD model

SIRD model simply introduces a fatality factor and survivability dependent on it. We can write the equations describing the systems as (with delay):

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\frac{\beta}{N}S(t)I(t) \tag{2.7}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\beta}{N}S(t)I(t) - \frac{\beta}{N}S(t-\tau)I(t-\tau)e^{-\mu\tau} - \mu I(t) \tag{2.8}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \frac{\beta}{N}S(t-\tau)I(t-\tau)e^{-\mu\tau} \tag{2.9}$$

$$\frac{\mathrm{d}D}{\mathrm{d}t} = \mu I(t) \tag{2.10}$$

Below is the plot for a SIRD model without delay, whose equations are same as SIR model, just with a fatality factor μ. For these models, μ was assumed to be 0.01, a fatality rate of 1%. Germany's official death rate was just 1.2% around April 2020 [Stafford, 2020], which was the reason for the choice of assumption. The amount of deaths in the model might seem a lot higher than the deaths being observed worldwide, the reasoning behind it is simple, as the model nears the $t = 200$ days mark, entirety of model population gets infected and recovers, while nowhere in the world has any country or state seen entire population getting affected, hence the total deaths would appear a lot more in the model than in reality, but the death rate remains consistent.

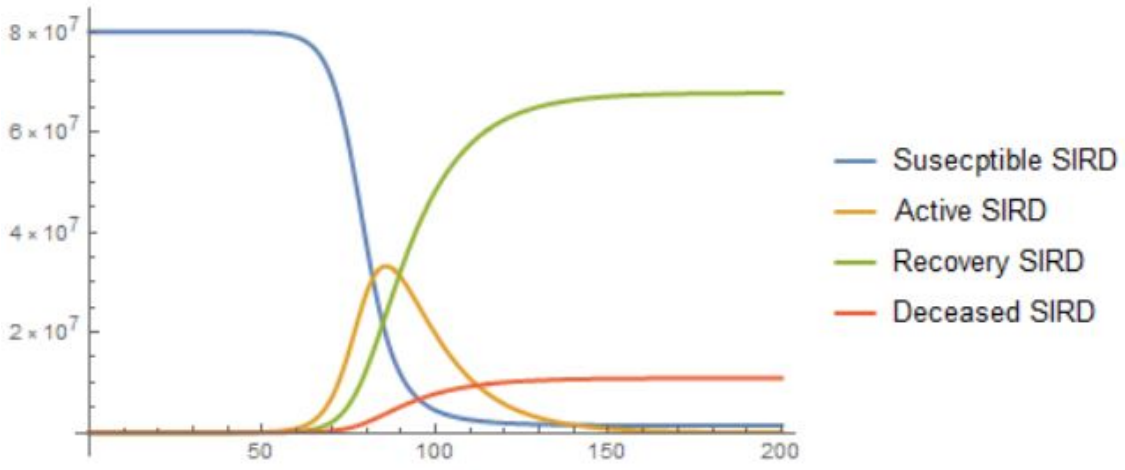

Figure 5 : A plot describing SIRD model

And below is delayed SIRD model superimposed on a SIRD model

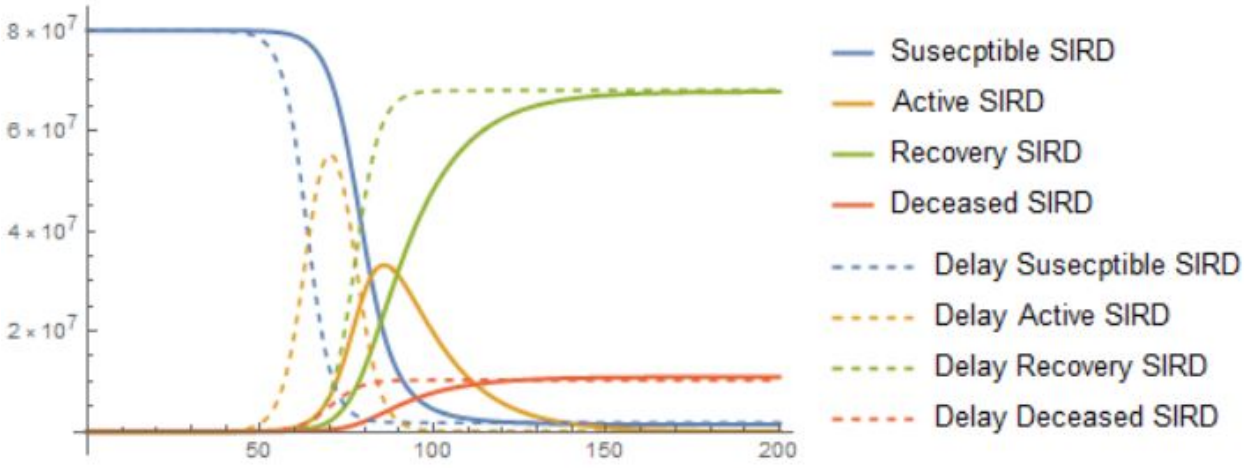

Figure 6 : A plot comparing SIRD model with delayed SIRD model

The plot above gives us similar inferences as the last SIR vs delayed Sir model, after the upgradation in this model was simply to include another compartment that separates deaths from recoveries, guided by a simple fatality factor. Interesting point to note here is that the deaths rise much earlier in the epidemic than a non delay model, but since the fatality factor is same, the total number of deaths remain the same.

2.4 Varying the Delay

Since the focus of our study is to see the effects of delay on these models, one can imagine a COVID like disease, with similar parameters, except the delay, for the same population and see how things change.

Below is the graph for delayed SIR model for the delay of 2 weeks which we had already seen,

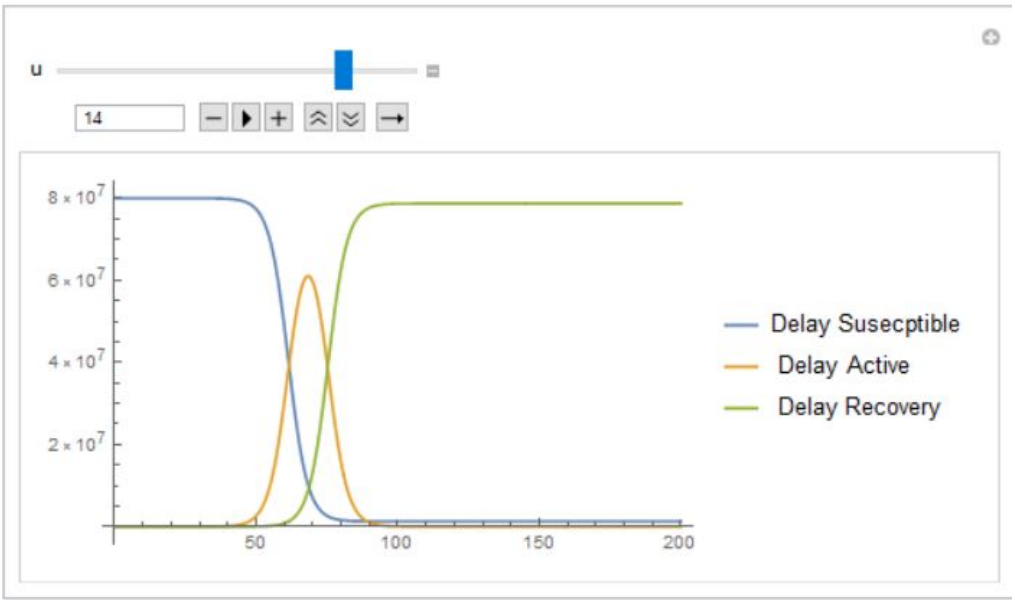

Figure 7 : SIR model with variable delay, delay factor u = 14 days

And below is the graph had the delay been of 6 days only

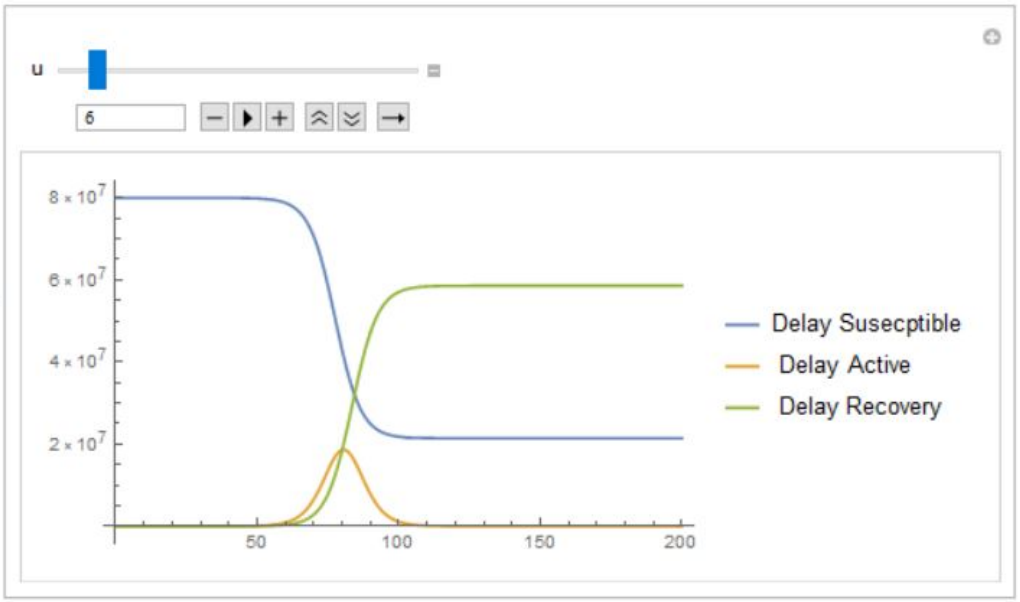

Figure 8 : SIR model with variable delay, delay factor u = 6 days

For a recovery period of 6 days, approximately half of the 14 day recovery period of COVID, we can an astonishing result, the infection curve never passes the susceptibility curve and approximately 1/4 of population remains susceptible even after the

initial outbreak. We also see the curves then continue as they are, suggesting just a singular outbreak which after it dies down and doesn't result in a second wave of the disease. This happens under the assumption that the infected population recovered well before they had the chance to infect a certain threshold population after which the entire population would become infected.

Similarly for SIRD model we observe almost the same results, naturally so. We also observe that the total deaths naturally reduce, as a lot of population still remains susceptible and never gets infected.

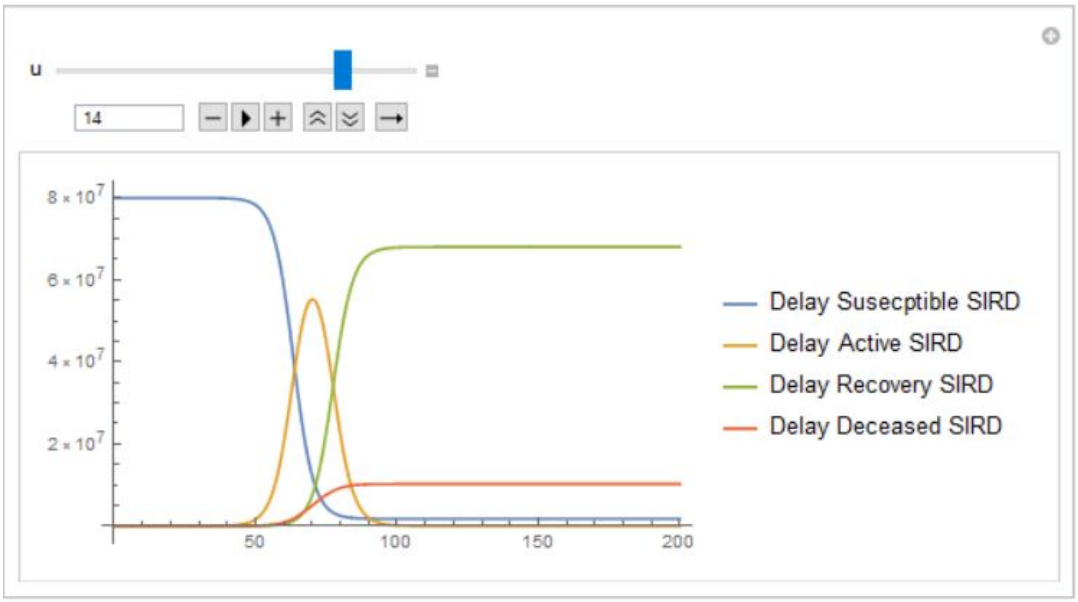

Figure 9 : SIRD model with variable delay, delay factor u = 14 days

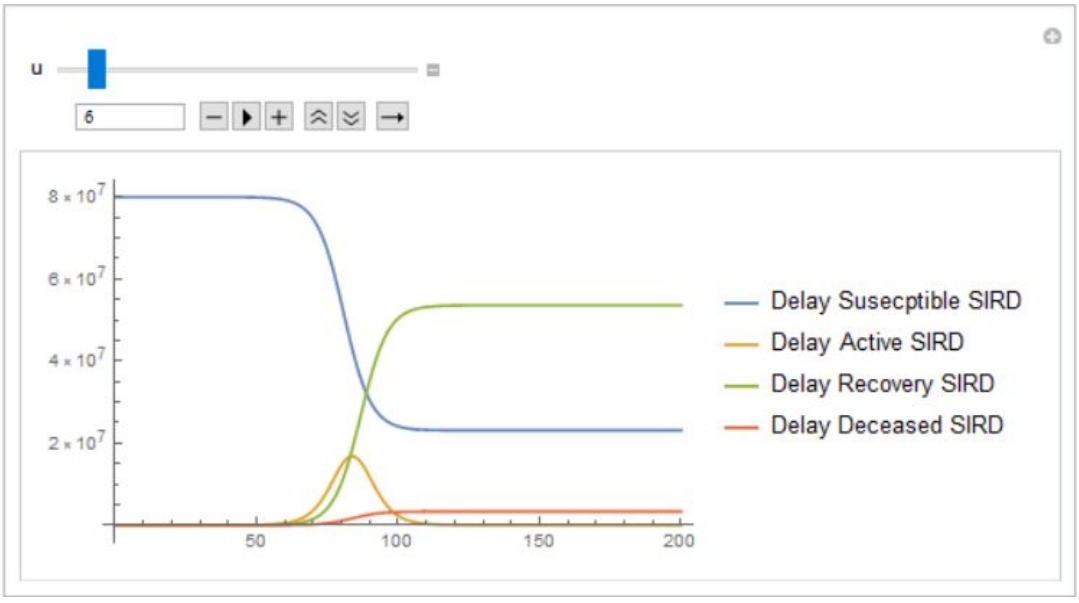

Figure 10 : SIRD model with variable delay, delay factor u = 6 days

2.5 Parametric Phase Diagrams

Of all the solutions that we have gotten above we can plot a parametric phase plot of Susceptible vs Infected compartments

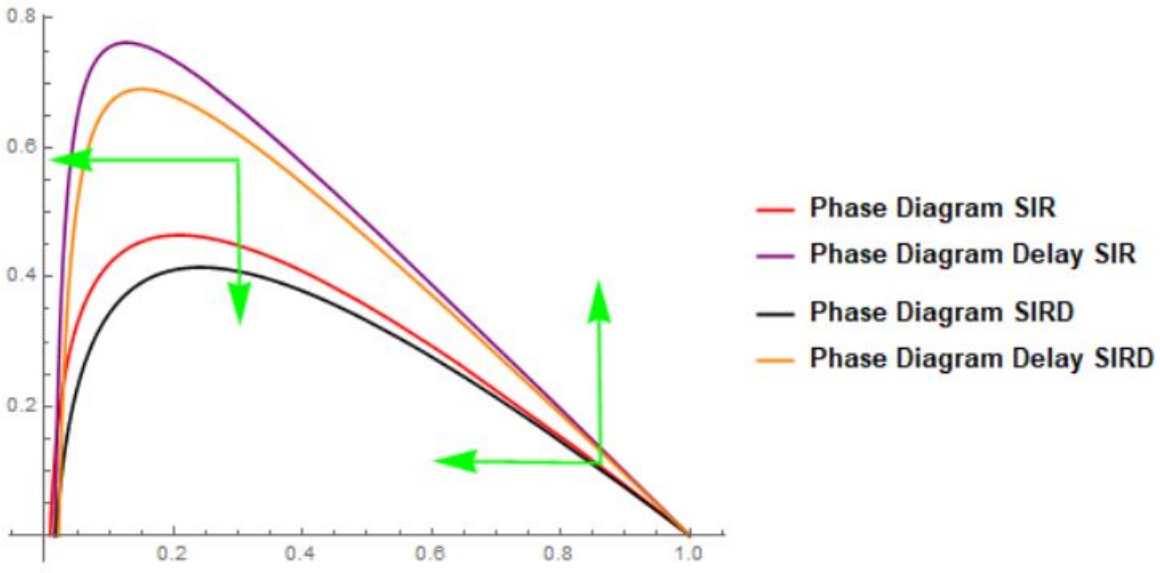

Figure 11 : Phase Diagram of Susceptible vs Infected space for all the models solved

We can observe the movement of infection from this plot and understand the Herd immunity threshold. We can see the number of infected cases rises till the number of susceptible people remain around a certain value and then the number of infection falls. This certain value, or the Herd Immunity Threshold, appears to assume a lower value in the delayed models over vanilla and in SIR models over SIRD models.

One can try introducing a variable delay in the phase space plots themselves to see the implications.

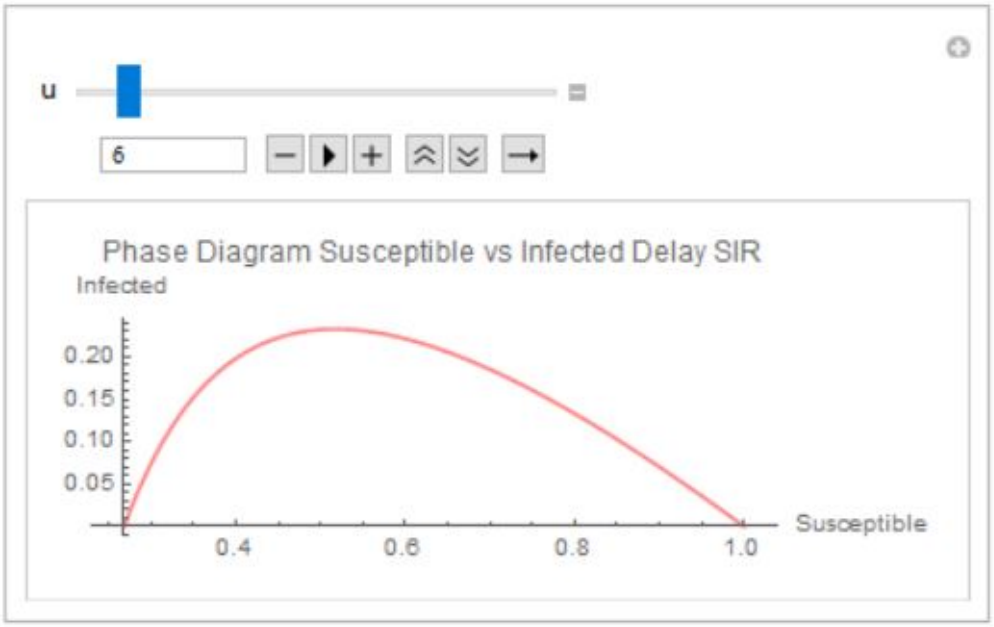

Figure 12 : Phase Diagram of Susceptible vs Infected space for all the models solved

We observe the results that we saw on variable delay section can inferred from this phase plot also. Infection curve remains small, and a certain amount of population (1/4) here remains susceptible even after the epidemic dies down.

One of the important inferences from these models is varying of epidemic overshoot due to herd immunity. Overshoot means the difference between final susceptible population and the point where the population's immunity prevents any further outbreaks, ie when the infection curve begins to fall. In the models with static delay, we can observe the overshoot is much lesser compared to the vanilla models, and when varying the delay we can observe the to decrease as the delay's length increases.

2.6 SIRS Model

One of the upgrades for the SIR model can be done while looking at the context of COVID-19 epidemic, one that of reinfection. The reinfection model or the temporary immunity model assumes that the immunity granted by the recovery from the disease is temporary in nature and around a fixed starts to fade away, making the population

again susceptible. Hence, termed as SIRS model. The model hence naturally results in oscillations of the disease.

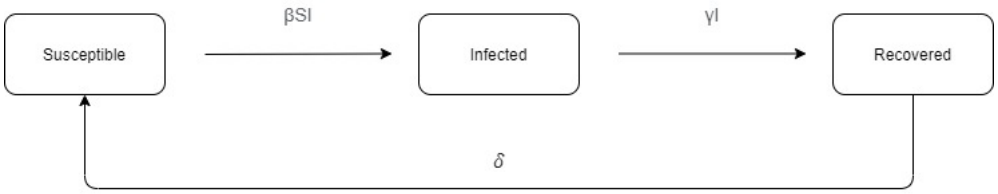

Figure 13 : A block diagram of SIRS model

This type of oscillatory behaviour is also seen in the cases affected with the seasonal flu[Rao et al., 2019][Greenhalgh and Moneim, 2003].

The undelayed SIRS model can be represented by[Greer et al., 2020]

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\frac{\beta}{N}S(t)I(t) + t_i R(t) \tag{2.11}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\beta}{N}S(t)I(t) - \gamma I(t) \tag{2.12}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I(t) - t_i R(t) \tag{2.13}$$

The other factors remain the same as above models, while the temporary immunity factor t_i is defined as $\frac{1}{t_0}$, where t_0 is the time in days by when the immunity from the disease nullifies. In the models under consideration the value of t_0 is taken as 60 days.

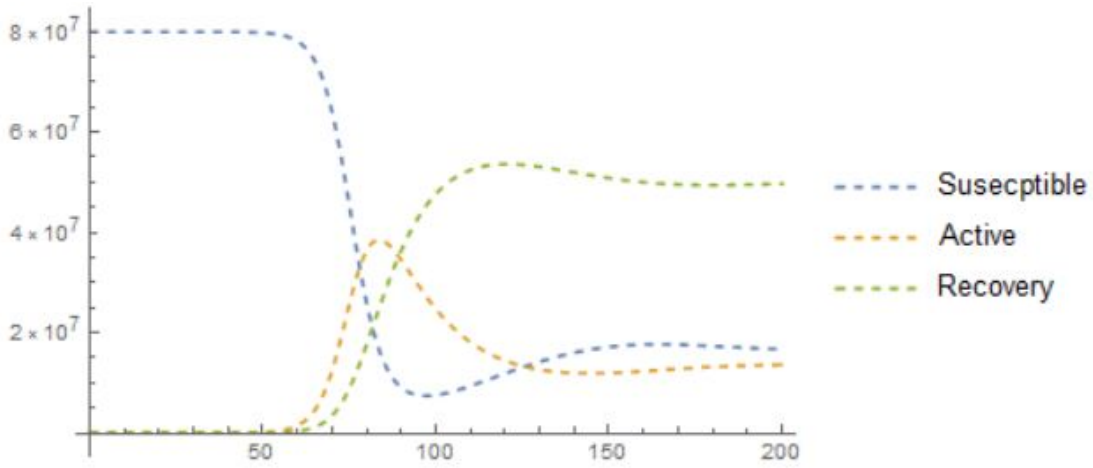

Figure 14 : Vanilla SIRS model

The delayed variant of the model hence becomes

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\frac{\beta}{N}S(t)I(t) + t_i R(t) \tag{2.14}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\beta}{N}S(t)I(t) - \frac{\beta}{N}S(t-\tau)I(t-\tau) \tag{2.15}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \frac{\beta}{N}S(t-\tau)I(t-\tau) - t_i R(t) \tag{2.16}$$

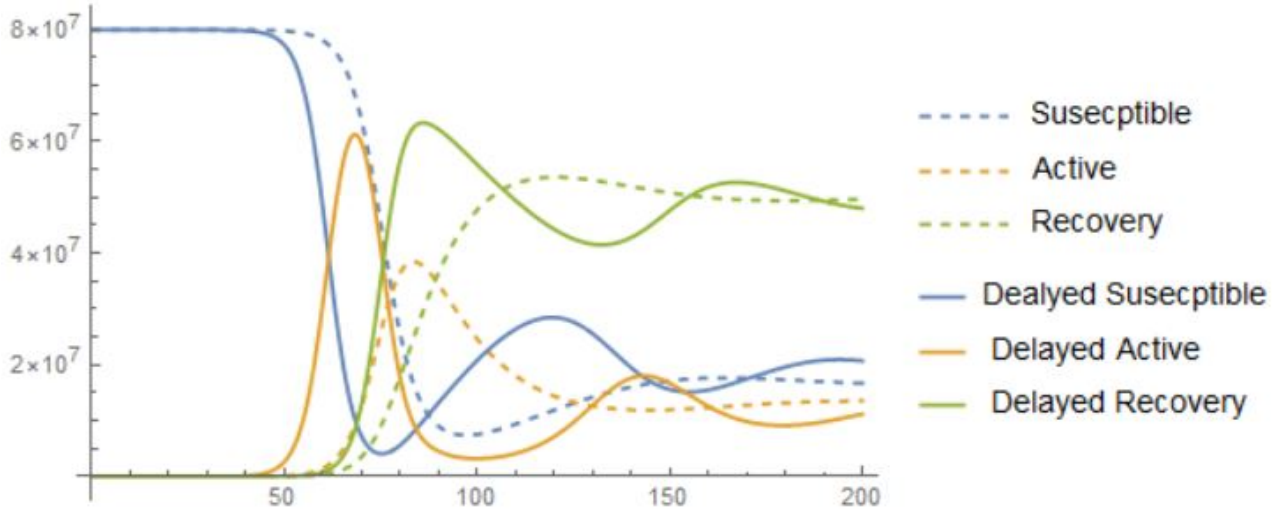

Figure 15 : Delayed SIRS model superimposed to the vanilla SIRS model

Naturally the object of the discussion with this model becomes the oscillatory nature and the impact of delay on the number of oscillations in a given amount of time. Although I couldn't spend more time on this specific problem, but it appears to me the temporal frequency of oscillations might carry something interesting, and it must be investigated as such in future. The interesting nature of the model is also visible in its phase diagram. Till now we have observed closed phase space diagrams for various models, but due to the fact the epidemic doesn't really die down, but rather just oscillates, we get a spiral structure rather than a simple parabolic path.

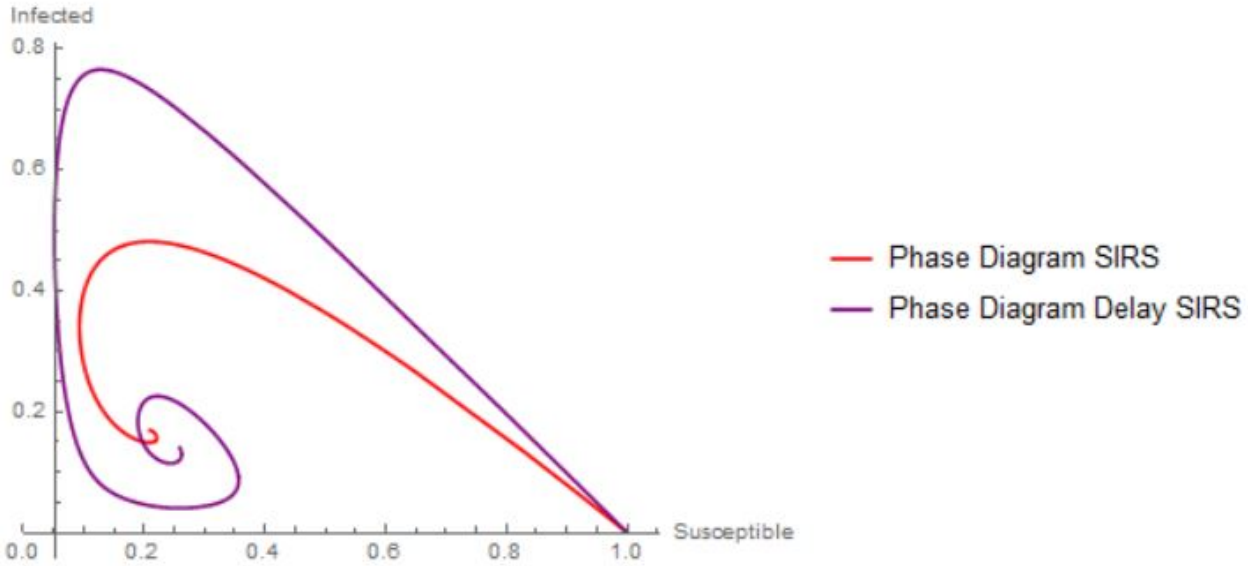

Figure 16 : Phase space diagram of Delayed SIRS model superimposed to the vanilla SIRS model

We can also try to vary the delay in the model, to investigate whether the amount of oscillations, and their amplitudes depend on it.

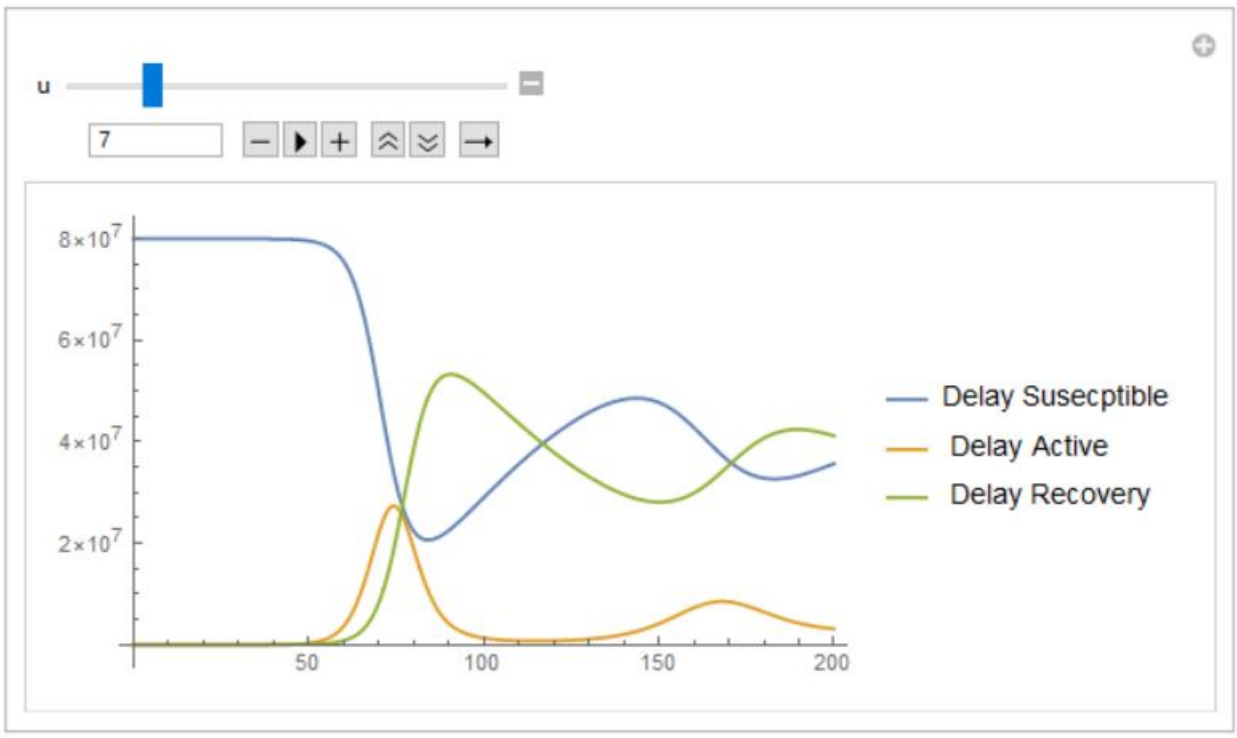

Figure 17 : SIRS model with variable delay, delay factor u = 7 days

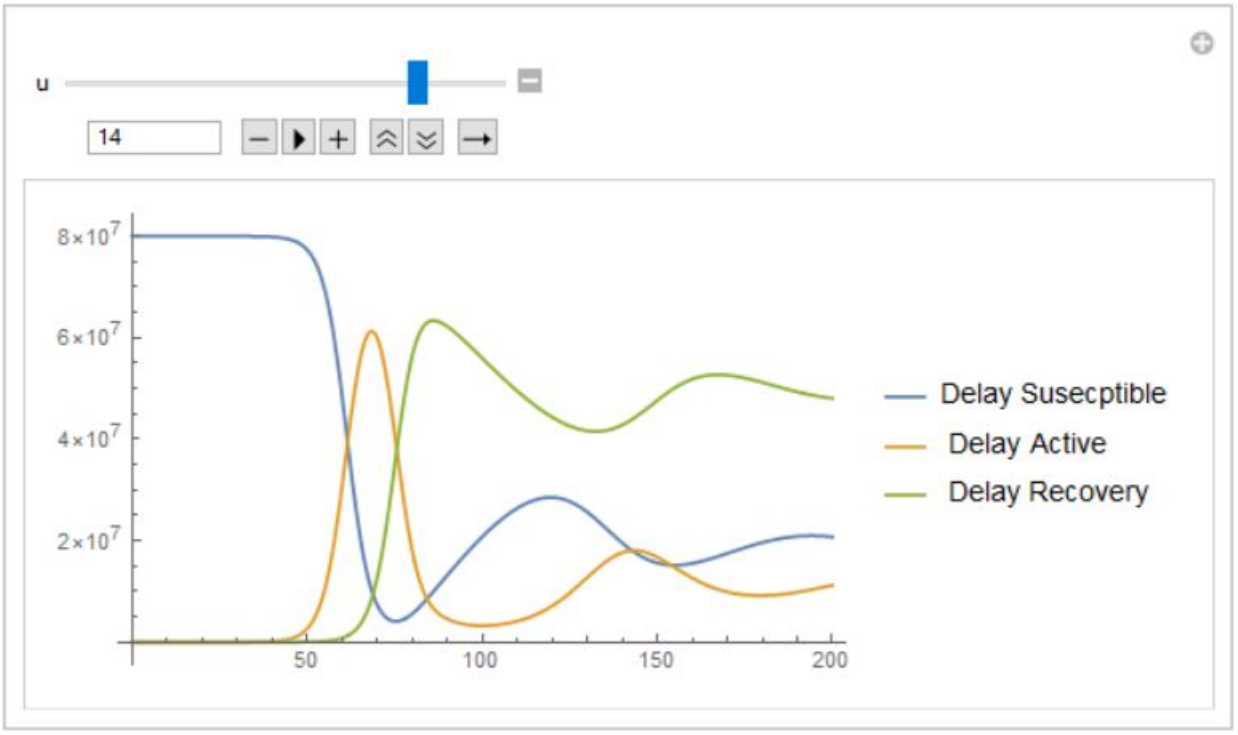

Figure 18 : SIRS model with variable delay, delay factor u = 14 days

From the above plots, its clear that oscillations have moved a bit to the left, thus in essence increasing the frequency of the oscillations. A parametric plot of delayed SIRS model reveals to us that the characteristic spiral of the model, does change with a change in delay.

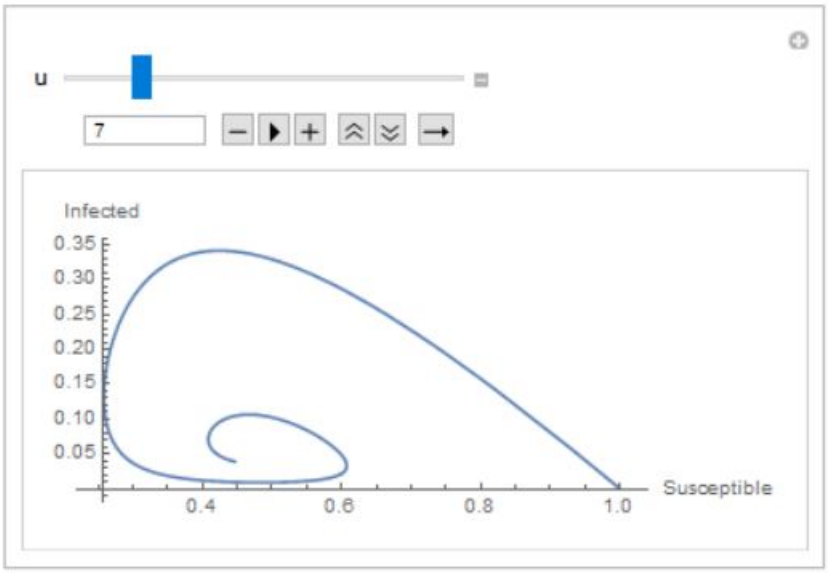

Figure 19 : Parametric Phase plot of SIRS model with variable delay, delay factor u = 7 days

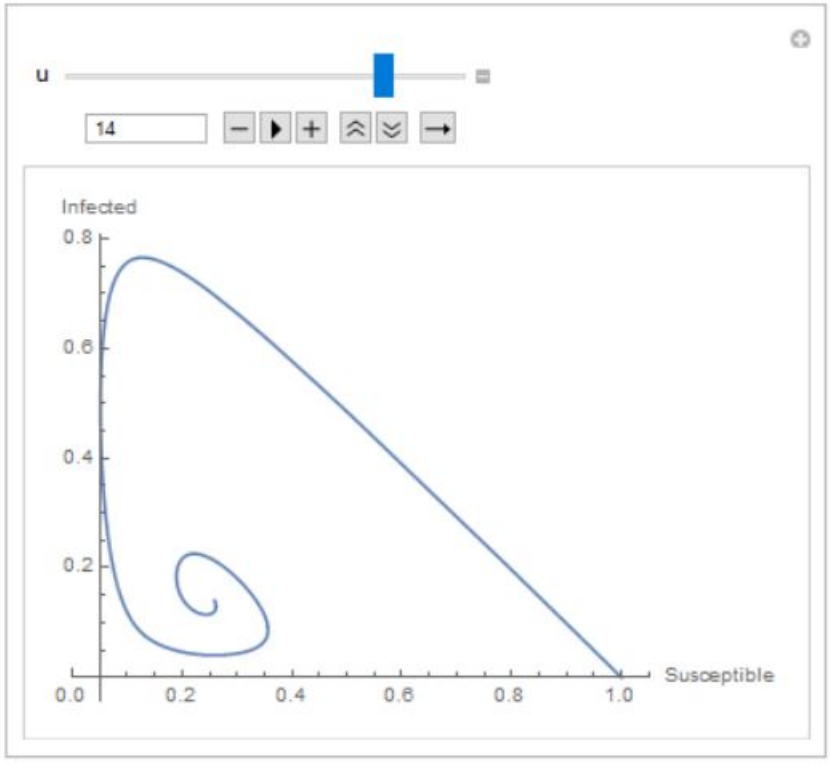

Figure 20 : Parametric Phase Plot of SIRS model with variable delay, delay factor u = 14 days

We can clearly observe from these plots that the spiral length is indeed dependent on the delay length, which also is consistent with the SIR curves moving to the left (refer Fig 16 - 17), meaning an increment in the frequency of oscillations.

2.7 Coupled SIR Model

The next step of increasing the sophistication of the SIR model was to introduce a coupling. The model involved taking two different populations with two different sets of the same disease with their own characteristic coefficients, which depends on external factors like masking, severity of social distancing etc and the intrinsic factors of disease like the delay factor due to mutation etc, modeling as two different geographical locations[Magal et al., 2016][Chen et al., 2014]. After this, a pathway for exchange of population was introduced, thus making both populations susceptible to both sets of diseases.

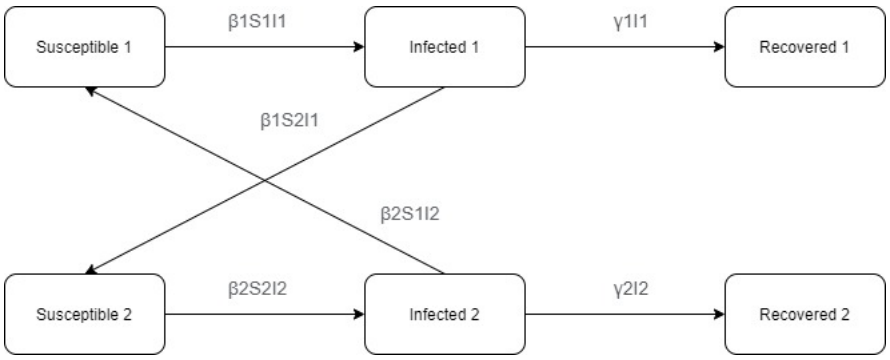

Figure 21 : A block diagram of coupled SIR model

The vanilla coupled SIR can be written as [Ambrosio and Aziz-Alaoui, 2020]

$$\frac{\mathrm{d}S_1}{\mathrm{d}t} = -S_1(t)(\frac{\beta_1}{N_1}I_1(t) + \frac{\beta_2}{N_1}I_2(t)) \tag{2.17}$$

$$\frac{\mathrm{d}S_2}{\mathrm{d}t} = -S_2(t)(\frac{\beta_1}{N_2}I_1(t) + \frac{\beta_2}{N_2}I_2(t)) \tag{2.18}$$

$$\frac{\mathrm{d}I_1}{\mathrm{d}t} = S_1(t)(\frac{\beta_1}{N_1}I_1(t) + \frac{\beta_2}{N_1}I_2(t)) - \gamma I_1(t) \tag{2.19}$$

$$\frac{\mathrm{d}I_2}{\mathrm{d}t} = S_2(t)(\frac{\beta_1}{N_2}I_1(t) + \frac{\beta_2}{N_2}I_2(t)) - \gamma I_2(t) \tag{2.20}$$

$$\frac{\mathrm{d}R_1}{\mathrm{d}t} = \gamma_1 I_1(t) \tag{2.21}$$

$$\frac{\mathrm{d}R_2}{\mathrm{d}t} = \gamma_2 I_1(t) \tag{2.22}$$

Setting the values of $\beta_1 = 0.3$, $N_1 = 80$ x 10^6 $\gamma^{-1} = \mathrm{T} = 16$ days, $\beta_2 = 0.5$, $N_2 = 50$ x 10^6, $\gamma_2^{-1} = 10$ days, we can finally solve the coupled model, which gives us

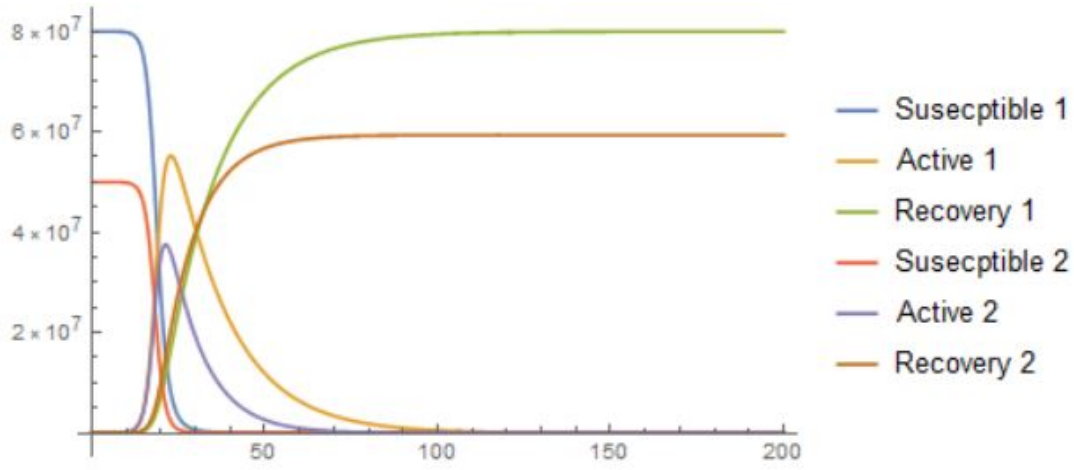

Figure 22 : Coupled SIR model

Now, we introduce a delay into the model, the equations then turn into

$$\frac{\mathrm{d}S_1}{\mathrm{d}t} = -S_1(t)(\frac{\beta_1}{N_1} I_1(t) + \frac{\beta_2}{N_1} I_2(t)) \tag{2.23}$$

$$\frac{\mathrm{d}S_2}{\mathrm{d}t} = -S_2(t)(\frac{\beta_1}{N_2} I_1(t) + \frac{\beta_2}{N_2} I_2(t)) \tag{2.24}$$

$$\frac{\mathrm{d}I_1}{\mathrm{d}t} = S_1(t)(\frac{\beta_1}{N_1} I_1(t) + \frac{\beta_2}{N_1} I_2(t)) - S_1(t-\tau_1)\frac{\beta_1}{N_1} I_1(t-\tau_1) - S_1(t-\tau_2)\frac{\beta_2}{N_1} I_2(t-\tau_2) \tag{2.25}$$

$$\frac{\mathrm{d}I_2}{\mathrm{d}t} = S_2(t)(\frac{\beta_1}{N_2} I_1(t) + \frac{\beta_2}{N_2} I_2(t)) - S_2(t-\tau_1)\frac{\beta_1}{N_2} I_1(t-\tau_1) - S_2(t-\tau_2)\frac{\beta_2}{N_2} I_2(t-\tau_2) \tag{2.26}$$

$$\frac{\mathrm{d}R_1}{\mathrm{d}t} = S_1(t-\tau_1)\frac{\beta_1}{N_1} I_1(t-\tau_1) + S_1(t-\tau_2)\frac{\beta_2}{N_1} I_2(t-\tau_2) \tag{2.27}$$

$$\frac{\mathrm{d}R_2}{\mathrm{d}t} = S_2(t-\tau_1)\frac{\beta_1}{N_2} I_1(t-\tau_1) + S_2(t-\tau_2)\frac{\beta_2}{N_2} I_2(t-\tau_2) \tag{2.28}$$

Keeping other coefficients values' constant, the delays, τ_1 and τ_2, can assume a similar value, assuming the two locations are suffering from the same strain of disease vector or different depending on the assumption that new variants with different

severity have arisen. For the sake of the investigation, both cases were considered. When $\tau_1 = \tau_2 = 14$ days, we get

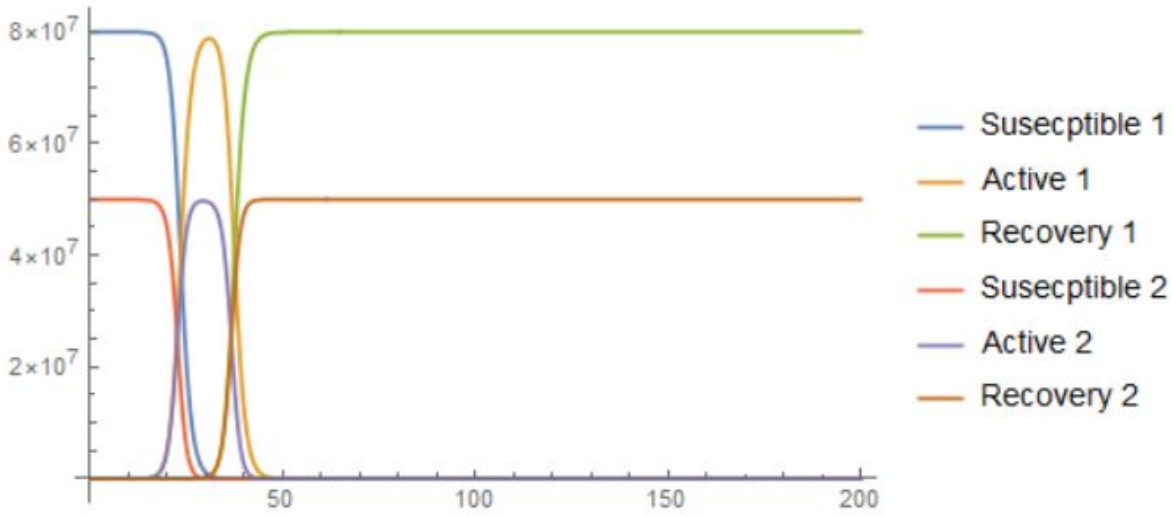

Figure 23 : Coupled Delayed SIR model with same delay, $\tau_1 = \tau_2 = 14$ days

and when $\tau_1 = 14$ days and $\tau_2 = 10$ days, we get

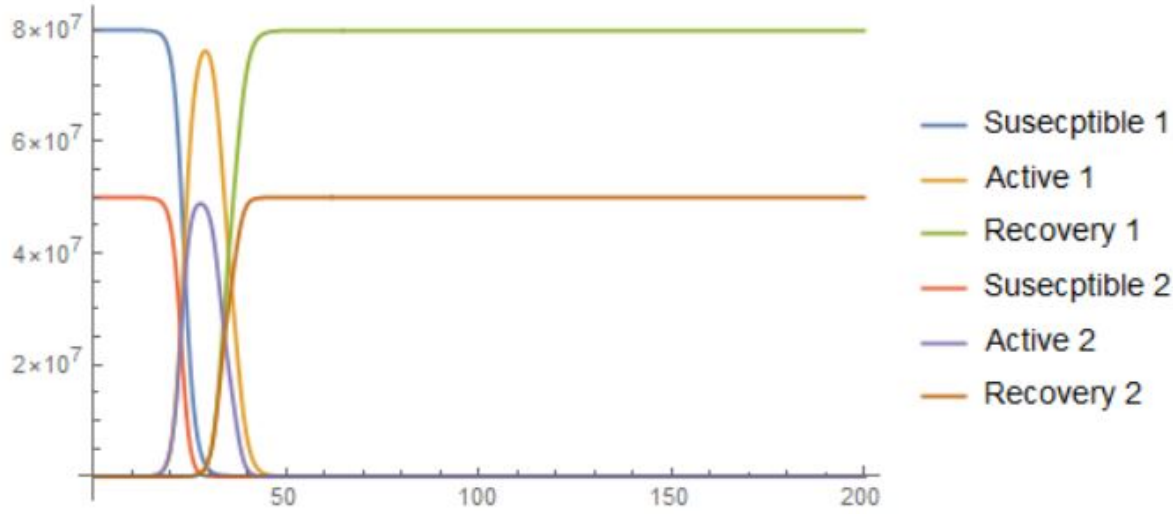

Figure 24 : Coupled Delayed SIR model with different delays, $\tau_1 = 14$ days and $\tau_2 = 10$ days

The phase plot of the coupled model is also very interesting, parting from the more parabolic plots that appeared in the investigation before, what now appears resembles a triangle more. This feature is dependent on the value of the spread factors, and their relative differences. If both the spread factors assume a lower end value and are hence closer to each other, the feature starts becoming smoother on its vertex, while even if one spread assumes a little higher value, for eg $\beta_1 = 0.3$ and $\beta_2 = 0.5$, the feature retains its sharp vertex. Higher values of spread factor, like 0.7, only make it sharper. Thus, there is a influence of spread factors on the characteristic shape of the parametric phase plot of the coupled model.

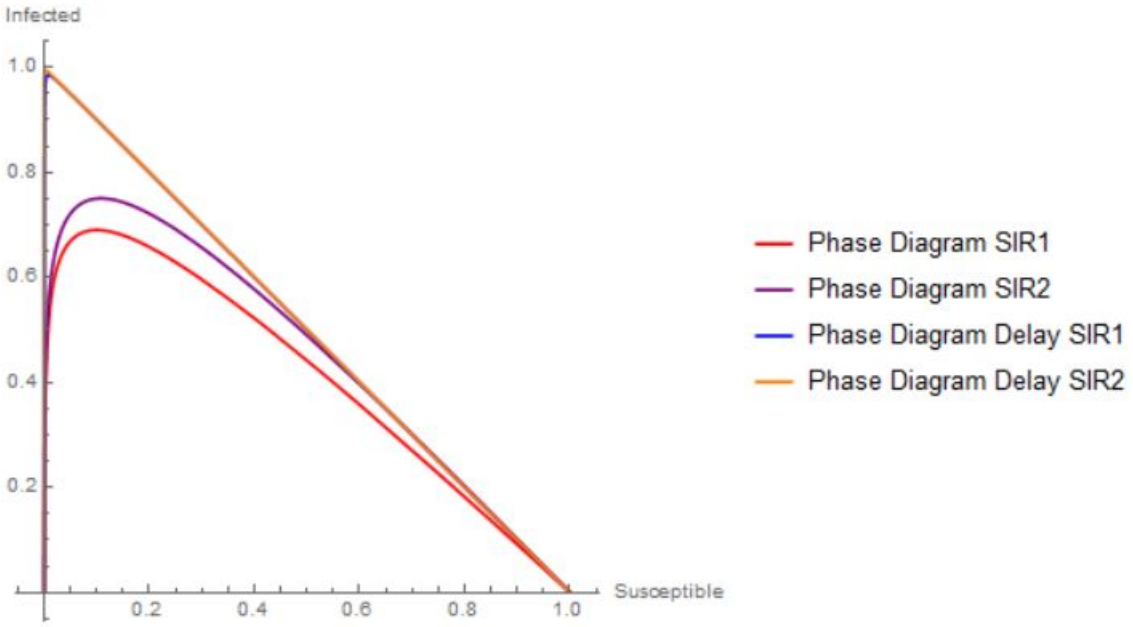

Figure 25 : Coupled Delayed SIR model with different delays, τ_1= 14 days and τ_2 = 10 days

2.8 Coupled SIRS Model

Now a natural progression of this investigation would be to merge these models to create a more complex scenario, bringing the model one step closer to mimicking real world conditions, which although due to the simplicity of the model still isn't possible. This model assumes like the above model assumes two different set of geographical locations with different coefficients and an allowed exchange, and then

introduces a temporary immunity. The temporary immunity associated with both strains is also assumed to be different.

The undelayed vanilla model can obtained by merging the SIRS model with the Coupled model, *ie* Eq. 2.11-2.13 and Eq. 2.17-2.22, and carrying forward the values of coefficients, we get

$$\frac{\mathrm{d}S_1}{\mathrm{d}t} = -S_1(t)(\frac{\beta_1}{N_1}I_1(t) + \frac{\beta_2}{N_1}I_2(t)) + t_{i1}R_1(t) \tag{2.29}$$

$$\frac{\mathrm{d}S_2}{\mathrm{d}t} = -S_2(t)(\frac{\beta_1}{N_2}I_1(t) + \frac{\beta_2}{N_2}I_2(t)) + t_{i2}R_2(t) \tag{2.30}$$

$$\frac{\mathrm{d}I_1}{\mathrm{d}t} = S_1(t)(\frac{\beta_1}{N_1}I_1(t) + \frac{\beta_2}{N_1}I_2(t)) - \gamma I_1(t) \tag{2.31}$$

$$\frac{\mathrm{d}I_2}{\mathrm{d}t} = S_2(t)(\frac{\beta_1}{N_2}I_1(t) + \frac{\beta_2}{N_2}I_2(t)) - \gamma I_2(t) \tag{2.32}$$

$$\frac{\mathrm{d}R_1}{\mathrm{d}t} = \gamma_1 I_1(t) - t_{i1}R_1(t) \tag{2.33}$$

$$\frac{\mathrm{d}R_2}{\mathrm{d}t} = \gamma_2 I_1(t) - t_{i2}R_2(t) \tag{2.34}$$

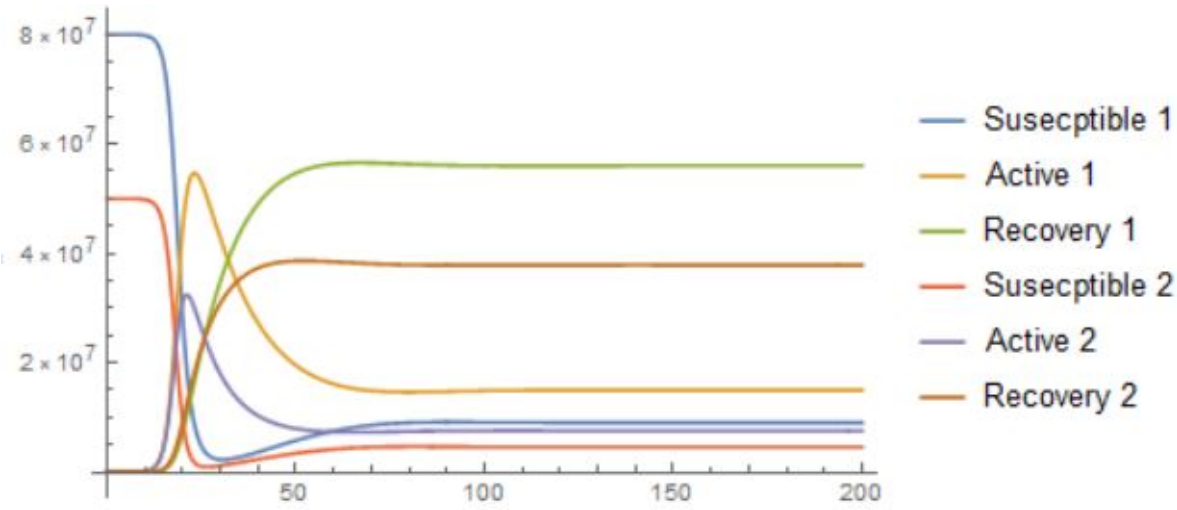

Figure 26 : Coupled SIRS model with $t_{01} = 60$ days and $t_{02} = 50$ days

The phase plot of the same comes out to be

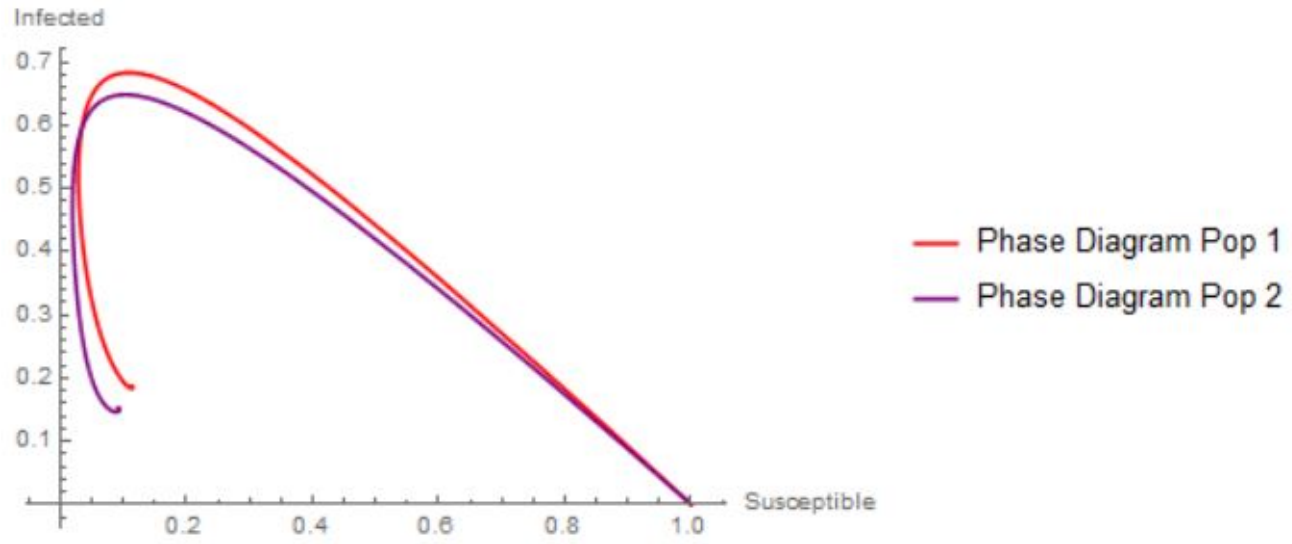

Figure 27 : Coupled SIRS model with $t_{01} = 60$ days and $t_{02} = 50$ days

The phase plot shows us the characteristic of vanilla SIRS and and coupled model, having a parabolic trajectory, but not a closed figure and a small curve at the end suggesting a spiral nature. This plot isn't as important in itself, but rather is required to provide a contrast to the delayed version. The delayed version of the model can be obtained by merging Eqs 2.14-2.16 with Eqs 2.23-2.28, combining them we get

$$\frac{\mathrm{d}S_1}{\mathrm{d}t} = -S_1(t)(\frac{\beta_1}{N_1}I_1(t) + \frac{\beta_2}{N_1}I_2(t)) + t_{i1}R_1(t) \tag{2.35}$$

$$\frac{\mathrm{d}S_2}{\mathrm{d}t} = -S_2(t)(\frac{\beta_1}{N_2}I_1(t) + \frac{\beta_2}{N_2}I_2(t)) + t_{i2}R_2(t) \tag{2.36}$$

$$\frac{\mathrm{d}I_1}{\mathrm{d}t} = S_1(t)(\frac{\beta_1}{N_1}I_1(t)+\frac{\beta_2}{N_1}I_2(t))-S_1(t-\tau_1)\frac{\beta_1}{N_1}I_1(t-\tau_1)-S_1(t-\tau_2)\frac{\beta_2}{N_1}I_2(t-\tau_2) \tag{2.37}$$

$$\frac{\mathrm{d}I_2}{\mathrm{d}t} = S_2(t)(\frac{\beta_1}{N_2}I_1(t)+\frac{\beta_2}{N_2}I_2(t))-S_2(t-\tau_1)\frac{\beta_1}{N_2}I_1(t-\tau_1)-S_2(t-\tau_2)\frac{\beta_2}{N_2}I_2(t-\tau_2) \tag{2.38}$$

$$\frac{\mathrm{d}R_1}{\mathrm{d}t} = S_1(t-\tau_1)\frac{\beta_1}{N_1}I_1(t-\tau_1) + S_1(t-\tau_2)\frac{\beta_2}{N_1}I_2(t-\tau_2) - t_{i1}R_1(t) \tag{2.39}$$

$$\frac{\mathrm{d}R_2}{\mathrm{d}t} = S_2(t-\tau_1)\frac{\beta_1}{N_2}I_1(t-\tau_1) + S_2(t-\tau_2)\frac{\beta_2}{N_2}I_2(t-\tau_2) - t_{i2}R_2(t) \tag{2.40}$$

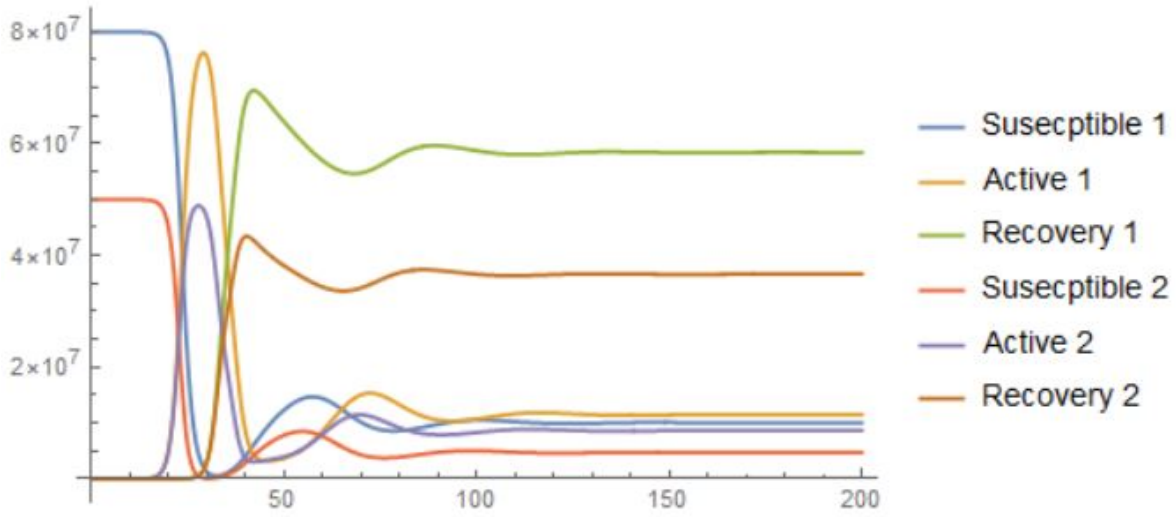

Figure 28 : Coupled Delayed SIRS model with different delays, τ_1= 14 days and τ_2 = 10 days and t_{01} = 60 days and t_{02} = 50 days

The phase space plot of both the populations gives us an interesting plot. Seeing how this model was made by conjunction of SIRS and Coupled SIR models, we can see the characteristic qualities of both models, the triangular plot of coupled and the spiral plot of SIRS, in the phase diagram.

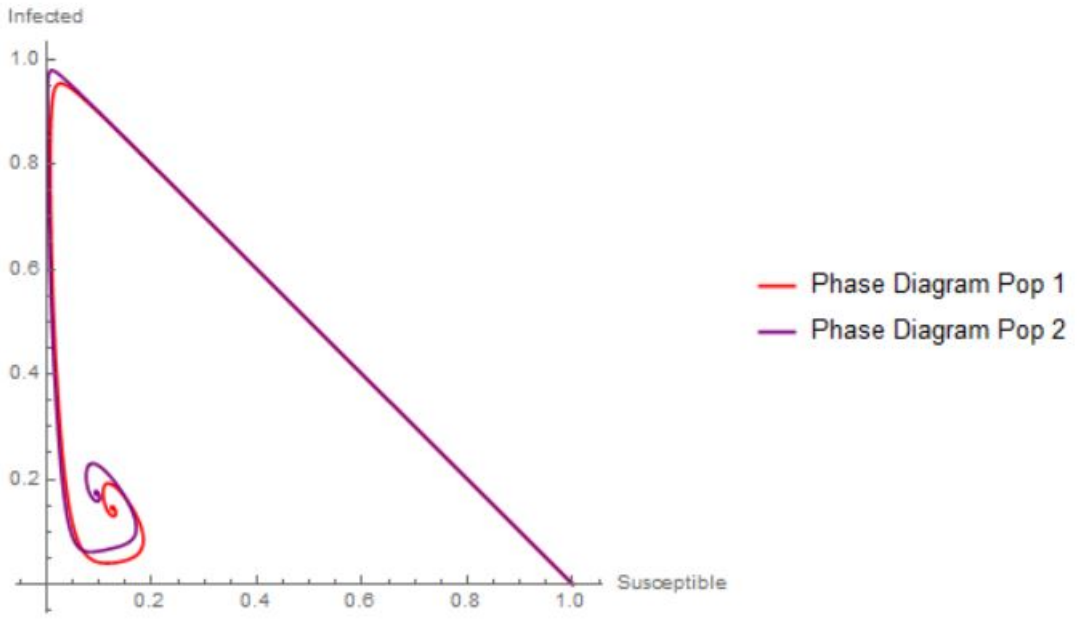

Figure 29 : Phase space plot of Coupled Delayed SIRS model with different delays, τ_1= 14 days and τ_2 = 10 days and t_{01} = 60 days and t_{02} = 50 days

2.9 Lockdowns and SIR

In the light of pandemic, with vaccines far away, one of the primary steps taken by countries worldwide was to introduce lockdown, of varying degrees, in an attempt to gain the upper hand and bide some time. Lockdowns essentially work on the concept of reducing the spread factor. Since the diseases of epidemics are communicable, it is a logical step to reduce the chance of contracting it by reducing the chances of communicating. Mathematically, we can model it by assuming a variable spread factor, that depends on the severity of lockdowns. A variable lockdown model can be written as

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\frac{\tilde{\beta}}{N} S(t)I(t) \tag{2.41}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\tilde{\beta}}{N} S(t)I(t) - \frac{\tilde{\beta}}{N} S(t-\tau)I(t-\tau) \tag{2.42}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \frac{\tilde{\beta}}{N} S(t-\tau)I(t-\tau) \tag{2.43}$$

where $\tilde{\beta}$ = (1-l_t)β, is the new spread factor which depends on l_t, which represents lockdown severity. l_t's value ranges from 0 to 1, with 1 being the extreme lockdown of no contact whatsoever, and hence effectively a zero spread factor.

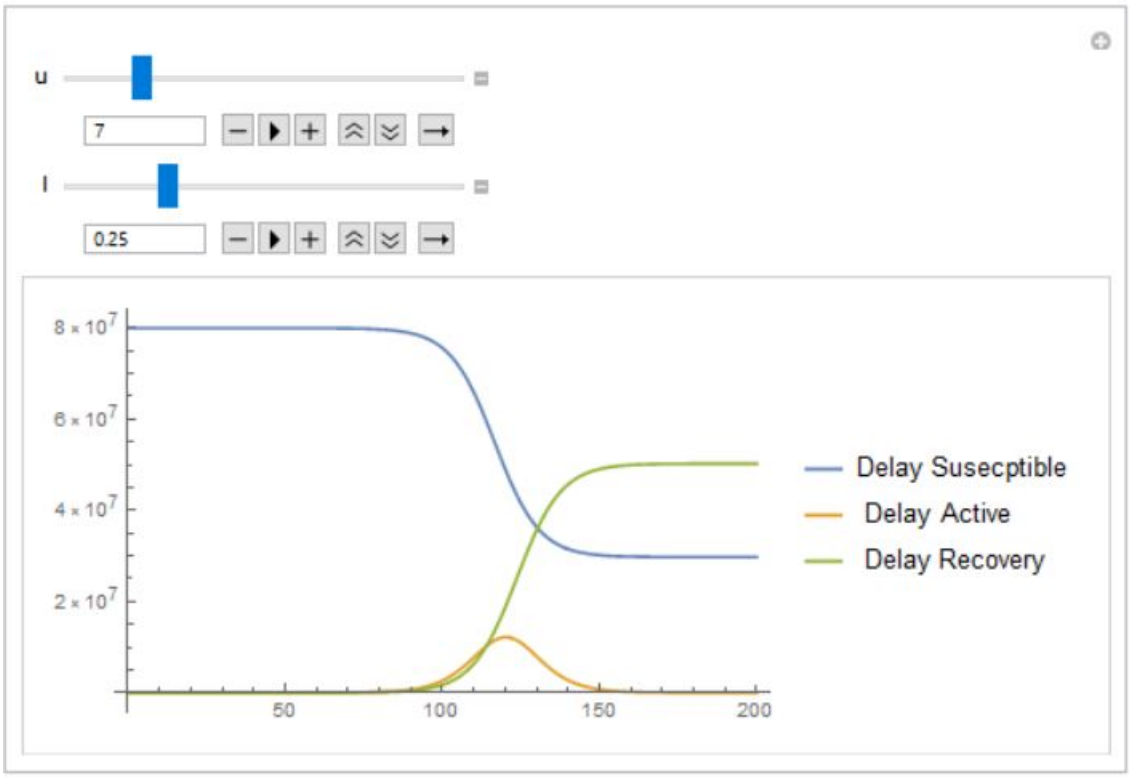

Figure 30 : Lockdown delayed SIR model, with u = 7 days and a lockdown of severity $l_t = 0.25$

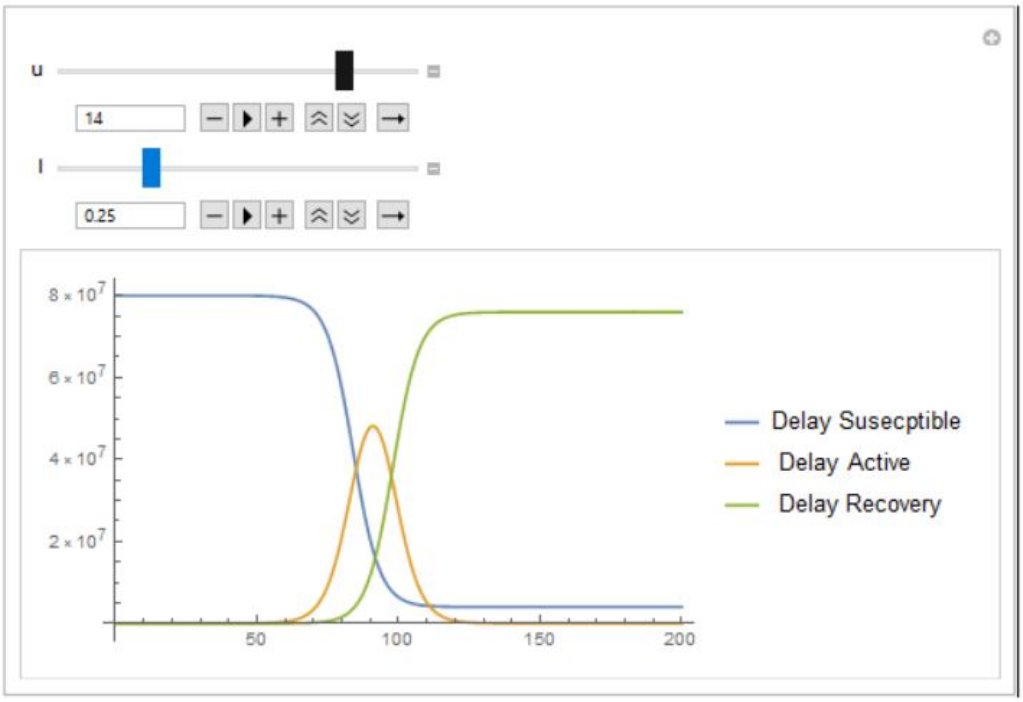

Figure 31 : Lockdown delayed SIR model, with u = 14 days and a lockdown of severity $l_t = 0.25$

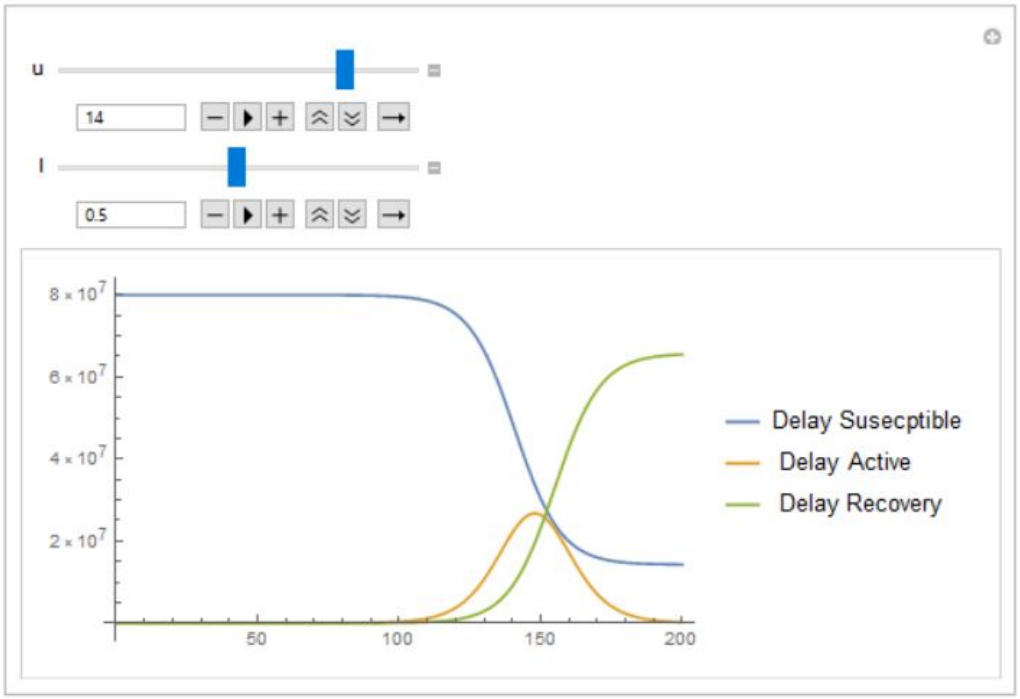

Figure 32 : Lockdown delayed SIR model, with u = 7 days and a lockdown of severity $l_t = 0.5$

A similar variable lockdown model with varying temporary immunity can be modelled on similar lines.

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\frac{\tilde{\beta}}{N}S(t)I(t) + t_i R(t) \tag{2.44}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\tilde{\beta}}{N}S(t)I(t) - \frac{\tilde{\beta}}{N}S(t-\tau)I(t-\tau) \tag{2.45}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \frac{\tilde{\beta}}{N}S(t-\tau)I(t-\tau) - t_i R(t) \tag{2.46}$$

Figure 33 : Lockdown delayed SIRS model, with u = 7 days, r = 0.05 and a lockdown of severity $l_t = 0.25$

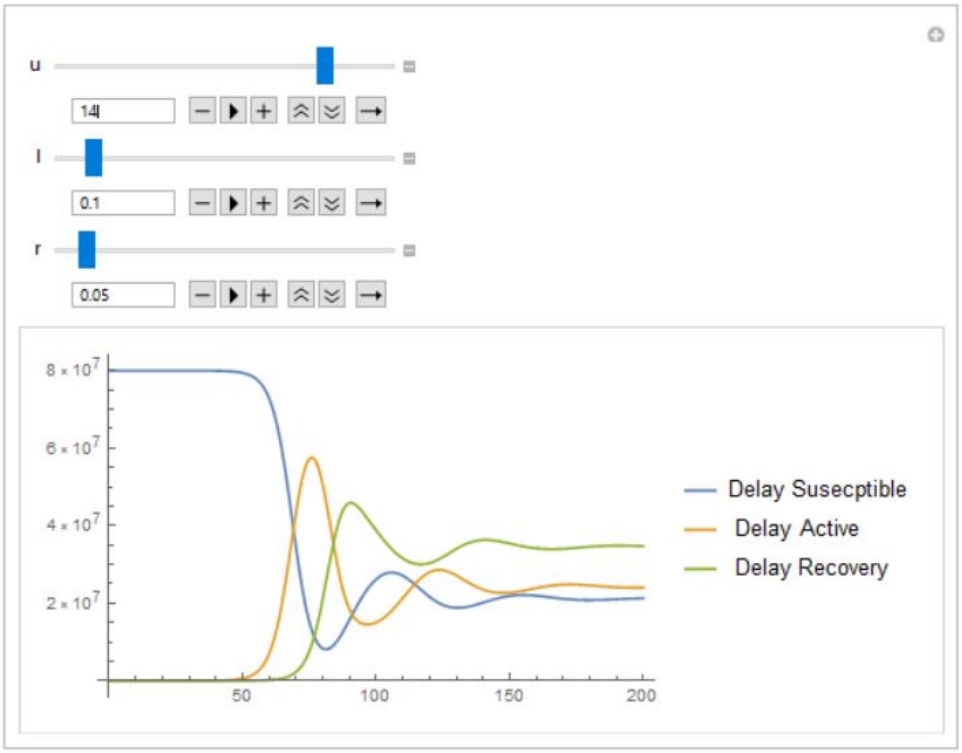

Figure 34 : Lockdown delayed SIRS model, with u = 14 days, r = 0.05 and a lockdown of severity $l_t = 0.25$

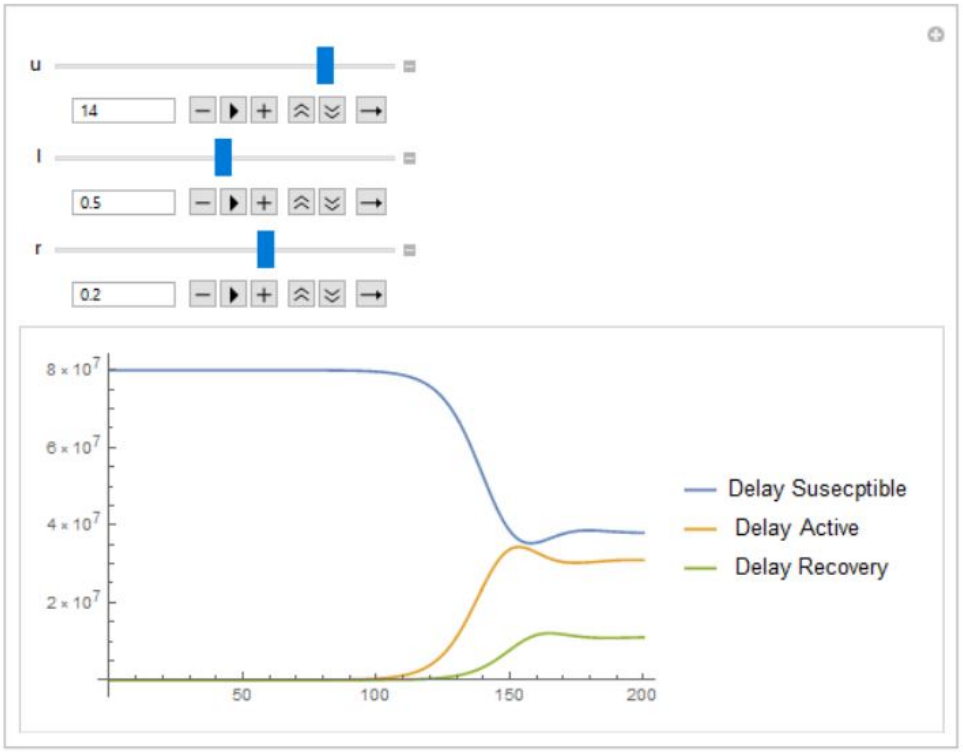

Figure 35 : Lockdown delayed SIRS model, with u = 7 days, r = 0.2 and a lockdown of severity $l_t = 0.5$

The values of variables have been chose similarly throughout the experiments and are meant to depict the behaviour of the model to the change in them, as throughout the range of values behaviour remained consistent and nothing abrupt or of particular interest was observed.

The severity of the lockdowns has a straightforward effect, the graph moves to right as a whole. This can be interpreted as the SIR sections all remaining constant at their initial values, due to the lockdown no one meets and disease doesn't spread. The other phenomenon to be noticed in the latter sub-model is the variation of temporary immunity factor r. The increment in the value of r results in more people remaining in the infected category, due to the lapse of immunity after a while, and the recovery category doesn't come out on top of infected curve later, something we hadn't observed in other models till now.

Chapter 3

Discussion and Further Space for Investigation

3.1 Discussion

As we can see from the plots above that initial SIR models are quite simplistic due to a lot of inherent and strong assumptions like homogeneity of population, equal spreading probability over age, sex and location etc.

These are the assumptions that make the model unsuitable for any real life planning, but nevertheless seen from a larger scale, the model does posses a certain amount of predicting power, which can be improved by inclusion of various parameters in the model. One of assumption the standard SIR model is that the time in which individuals remain infectious is described by an exponential distribution, which is a bit unrealistic.

A more realistic assumption would be a constant infection period. One of the important point to be noted is that vanilla models seem to severely underpredict the outbreak if the infection has a recovery period. The inclusion of a delay period corrects for these underestimations. In fact, Dell'Anna showed in his paper exactly this result. Comparing with real world data from Italy, the paper shows how standard SIR model underpredicts the epidemic and how of inclusion of delay improves the model's predictive capacity[Dell'Anna, 2020].

As the models' sophistication grew, coupled with the conclusion that delayed model's

predict the outbreak better than the vanilla models, we get to see some serious deviations between the two. The behaviour more or less still remains the same globally, but we can see locally delayed models react much more to stimulus than the vanilla models. With the inclusion of lockdowns and spatial coupling, we can further create much more complicated models, but there are two immediate upgrades to the model that I wished to work upon but alas wasn't able to, these will be discussed in the next section.

Aside from these, the various models offer us certain observations, that might help in general decision making. We can use the SIR model to calculate a basic 'Effective Reproductive Number', ie how many susceptible individuals does a singular infected person infect per unit time. Even such a basic idea can help guide policy decisions. The SIRS model, coupled with lockdowns, can give us an idea on how to find a combination of coefficients that keeps the infected and recovered curve that would find decisions for maximum effectiveness in a pandemic that might just turn a seasonal disease.

3.2 Further Investigation

As seen throughout this study that SIR is a highly modular model with a lot of scope for sophistication. In this section, I wish to discuss further additions to the model that one can pursue to further create a more complex model and challenge a few of the other inherent assumptions of the basic model.

3.2.1 Multi Strain Model and Vaccinations

By the end of 2020, multiple strains of SARS CoV-2 were emerging out of various parts of worlds, by the virus' adaptation to their local conditions. Major countries of the world had finished vaccine development by then, and were just starting to roll them out. The emergence of a new strain of the virus can come about from mutation of the virus, antigenic drift/shift or even through introduction of a newer strain from an external source [Fudolig and Howard, 2020] [Ahn et al., 2014].

Hence, one of the developments of the Coupled model, which assumes two sets of coefficients for two geographical locations, can be assumption of two different strains

of virus itself. This model can be applied either by introduction of two strains in a singular population, with either both of them attacking simultaneously or one leading the other by a certain amount of time. The other application could be to introduce this multi-strain in the coupled model itself, modelling the August 2020 - March 2021 era, when lockdowns had been lifted in major parts of the world, but the newer strains were making a comeback and through the exchange of population (through resumption of travel), two locations became coupled with multi strains as well.

The development of vaccines was a much awaited point in this pandemic. By the end of 2020, a lot of major countries had either developed vaccines of their own or had concluded deals to source them from the countries that had. The emergence of newer strains has also presented a challenge to this fixation on the vaccine, and whether they would be effective against the newer variants[Elazzouzi et al., 2019].

The other upgrade could be to introduce vaccination in the SIR model, and in the multi strain model. Both the cases of blanket immunity from all strains by the vaccines and selective immunity from original strain but not from newer strain can be considered.

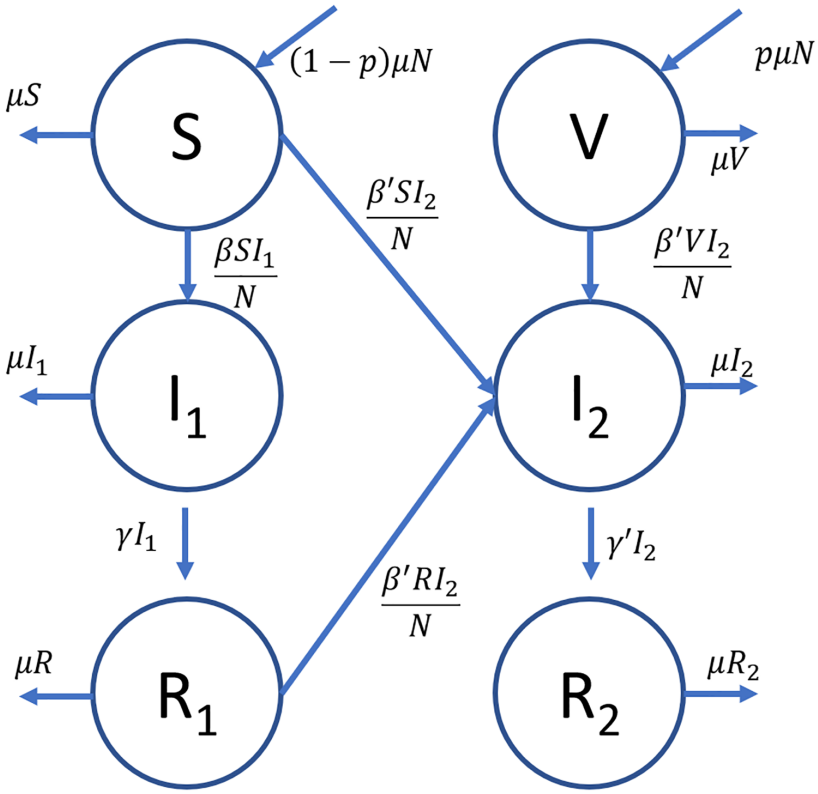

Figure 35 : Compartment diagram for the emerging disease model. The transitions between compartments, together with the corresponding rates, are described by the arrows directed in and out of each compartment.[Fudolig and Howard, 2020]

3.2.2 Lockdowns

In early 2022, when the spread of COVID was first being taken seriously, countries were imposing social distancing rules and masking mandates. By the middle of 2020, when it became clear that those methods were proving ineffective against the virus, the first of lockdowns were imposed. Lockdowns were the first major steps taken by government's worldwide to control the spread of pandemic. The investigation did cover lockdowns as well, yet only one type of lockdown was covered. Any further investigation should look at changing the the lockdowns' nature and severity.

The upgradation of the lockdown model can start with the changing of the function representing the lockdown in the model. These can further be applied in the coupled model, mimicking the situations when one country or state had lifted lockdown but the other hadn't and based on the severity of lockdown, a small transfer of population still took place. One could also focus on finding a lockdown function that minimises the total deaths. One could also working on finding the lockdown function which effectively minimises the total deaths.

3.2.3 Changing the Nature of Delay

For the entirety of the investigation, we have assumed the delay to be constant in nature, *ie* τ assumes a constant value, but that isn't necessarily true. One could consider a case of disease such as malaria, where not only the susceptible individual gets the disease from an infected vector(mosquito), but also an uninfected vector getting the infection from an infected individual. This situation, when coupled with the assumption that the disease might show retardation like COVID, we arrive at a situation where the delay isn't constant, but rather can be modeled as a distribution [Beretta and Takeuchi, 1995].

3.2.4 Different Vectors

Aside from studying different strains, different spatial and other couplings, one could even try to study impact of different vectors of the same disease on SIR model. As COVID was earlier thought to spread not only from close contact with the infected person, and transmitting through air, but also small droplets suspended on various surfaces, and thus bringing haptic aspect to the transmission. The delayed model there could be a further development of the continuous type delay, as discussed in last subsection [Miller, 2017].

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