Nonlinear Dynamics	Date: *17.04.2021*
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Study of Epidemic Models with Nonlinear Infection Forces and Proposing a Centrality based Intervention Model

1 Project Goal

Throughout history, infectious diseases, which are illnesses caused by a disease agent that can be transmitted from organism to organism, have had a big impact on the human health and social development through their epidemic course of action. Presently the world is under the COVID pandemic which started as an outbreak in China. This causes large scale harm to the society and therefore it is imperative to study such diseases to prevent them from growing to a stage where it is uncontrollable. Thus these diseases are modelled and we study their evolution in a hope to create some intervention by either vaccinating or lockdown or other means. Through this work we aim to **study an epidemic model with the effects of various forces which may intensify/suppress the epidemic**, mostly following the work of Wendi Wang[2] and try to **build an intervention model** on top of the prevention models at local spaces, **using percolation centrality** as the basis[5].

2 Introduction

The initial goal of this work is to understand the evolution of epidemic models over time. A lot of classical epidemic models admit threshold dynamics. One can find example of such in the work of Badshah, Porwal and Tiwari[1], where certain thresholds are suggested which are capable of forecasting whether the disease persists or not. A showcasing of such thresholding goes as, if a basic reproduction number R_0 is below 1, a disease-free equilibrium is globally stable while if it is above 1, an endemic equilibrium is globally stable. However, as models evolved, it became known that a bistable case is more likely to occur which means that a disease-free equilibrium and an endemic equilibrium are stable at the same time.

Epidemic models based on classical analysis failed to capture certain behaviours like periodic oscillations or the presence of bistability. They often consider the contact rates and infection probability per contact as being constant over time. However, the infection forces include the adaptation of individuals to infection risks. Consider the COVID epidemic in India, during second wave, as numbers seem to rise significantly in India, (more than 1 lakh daily rise) various states have implemented various protection measures, close of academic institutions in Telangana, night curfews in the states of Punjab and Maharashtra or limitation on entries of vehicles in Bengal, raising frequent questions on possibility of a forthcoming lockdown. These are some of the intervention forces that are employed to limit the spread. This indicates that the infection force has a nonlinear relation with the infected people which cannot be perhaps modelled using the classical approach. Another factor could be that there is possibility of an infection force being saturated over time as we might develop vaccines or the infected people might increase to an extent that the resulting new infections are very less. These factors probably seem to be missing in the classical models which had been explored by Wendi Weng in 2006.

In this work, our first objective is to understand the classical approach to SIR model (section 3) and then study the epidemic models with intervention strategies and a saturated infection force (section 4). The second objective is to connect the essence of the findings of Nonlinear Dynamic Model to a larger network based intervention model through the notion of percolation (section 5).

Using any such Nonlinear dynamic model we study in section 3 and 4 at a local spectrum (zones in cities/towns) , we hope to build a network centrality based intervention model at the broader level(cities/towns) in section 5.

3 Classical Approach : SIR Model

3.1 SIR model without vital dynamics[4]

The idea is to divide the population into various classes (compartments), where each group represents a specific stage of epidemic. Kermack and McKendrick created a model in which they considered a fixed population with only three compartments: susceptible $S(t)$; infected $I(t)$, and recovered $R(t)$[3].

- S : Number of susceptible individuals, i.e., individuals not yet infected with the disease.
- I : Number of individuals who are infected and are capable of spreading the disease to those in the susceptible category.
- R : Number of removed individuals, i.e., who have been infected and then removed from the disease, either due to immunization or due to death.

(t) denotes the count at time t.

Assumption : **Law of mass action** holds which states that if individuals in a population mix homogeneously, then the encounters between infected and susceptible individuals occur at a rate proportional to their respective numbers in the population

$$S \xrightarrow{\beta S \frac{I}{N}} I \xrightarrow{\gamma I} R$$

- β denotes the rate of transmission of the infection, i.e., the rate at which susceptible person becomes infected.
- γ denotes the recovery rate
- N denotes the total population, here we consider the total population as conserved, so there can be exchange of individuals between compartments, but their sum remains contains, i.e., $N = S + I + R$.

Let $\frac{I}{N}$ be the fraction of infected individuals as per total population. Then, as per the Law of Mass Action,

$$\text{Rate of Individuals going from S} \to I \text{ category is } \beta S \frac{I}{N}.$$

Similarly,

$$\text{Recovery rate of infected individuals } = \gamma I.$$

We are hence ready to look at the system through 3 equations stated below. **Note that the model is constructed without vital dynamics which includes the birth and death rates**. However, if we are studying an epidemic model over a long period of time, say close to a decade or two, we necessarily consider the birth and death rates.

$$\frac{dS}{dt} = -\beta S \frac{I}{N}$$
$$\frac{dI}{dt} = \beta S \frac{I}{N} - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

Note that since we considered N, the total population size as constant, $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$. There is no loss term in $\frac{dR}{dt}$ and no gain term in $\frac{dS}{dt}$ since we didn't consider the terms owing to death and birth rates. We have got a nonlinear coupled system of ODEs and no closed form solution exists for it.

Definition 1 (Reproduction Number)**.** *A threshold that helps us decide if the disease is spreading or is gradually diminishing.*

The disease will be an epidemic if disease keeps on increasing, i.e.,

$$\frac{dI}{dt} > 0$$

$$\implies \beta S \frac{I}{N} - \gamma I > 0$$

$$\implies \beta S \frac{I}{N} > \gamma I$$

$$\implies \frac{\beta S}{\gamma N} > I$$

$$\text{since } \frac{S}{N} < 1, \text{ we must have } \frac{\beta}{\gamma} > 1 \text{ for an epidemic}$$

- This ratio of transmission rate and recovery rate is called the *Reproduction Number*, i.e., $R_0 = \frac{\beta}{\gamma}$.
- Clearly, if $R_0 > 1$, the disease will be an epidemic and if $R_0 < 1$, the disease will not be an epidemic and eventually die out.
- It represents the total number of secondary infections produced when one infected individual is introduced into a disease free population. Higher the reproduction number, the faster the disease is going to spread.
- The reproduction number is a significant aspect of mathematical modelling for epidemiological models. In the COVID crisis, people tried figuring out reproduction number of COVID-19, which surely has to be greater than 1, that's why it is spreading so much, however satisfactory quantification of R_0 wasn't possible because the data isn't very consistent and varies across regions.

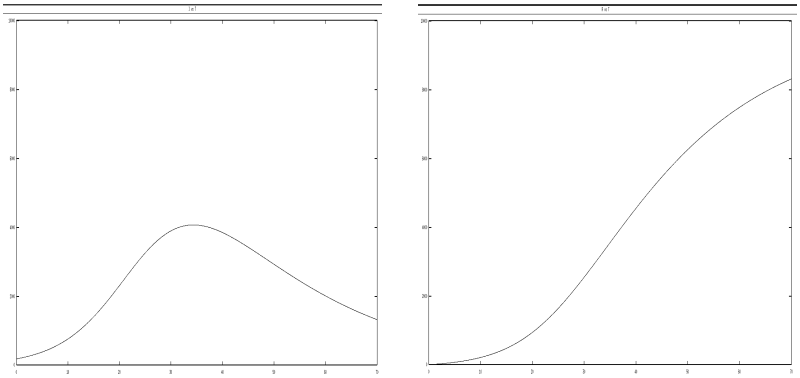


Figure 1: I vs T and R vs T for params: $\beta = 0.2$, N = 10000, $\gamma = 0.05$ ($R_0 > 1$)

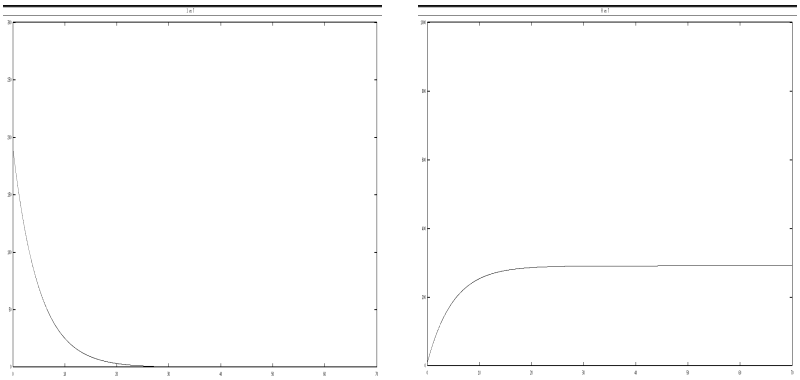


Figure 2: I vs T and R vs T for params: $\beta = 0.1$, N = 10000, $\gamma = 0.3$ ($R_0 < 1$)

3.2 SIR model with vital dynamics : Birth and Death

If an epidemic stays for a long time, it is declared as endemic. For long time scales, the effect of birth of new individuals and death of individuals also need to be counted. The addition of new individuals (birth) contributes to the count of susceptible individuals only, while the removal of individuals (death) takes away from the count of susceptible, infected and removed individuals at the same rate. Hence our modified system of equations look like,

$$\frac{dS}{dt} = bN - \beta S\frac{I}{N} - \mu S$$

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

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

where, b denotes the birth rate and μ denotes the death rate. From continuing sections we will use d for death rate respectively. This surely looks more complicated than previous set of equations and hence we don't look for analytical solutions here as well.

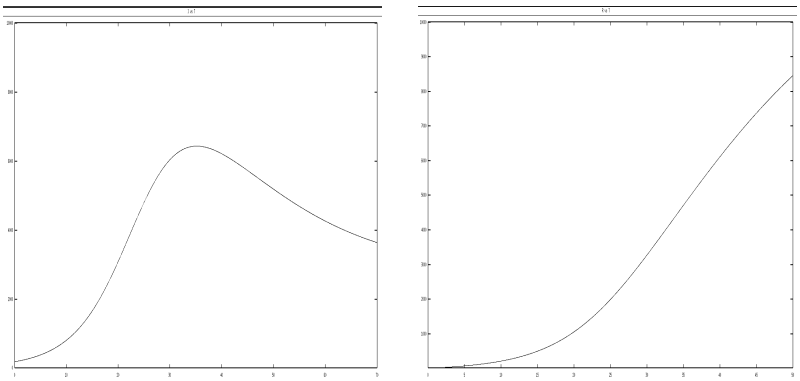


Figure 3: I vs T and R vs T for params: $\beta = 0.2$, N = 10000, $\gamma = 0.05$, b = 0.02, $\mu = 0.007$ ($R_0 > 1$)

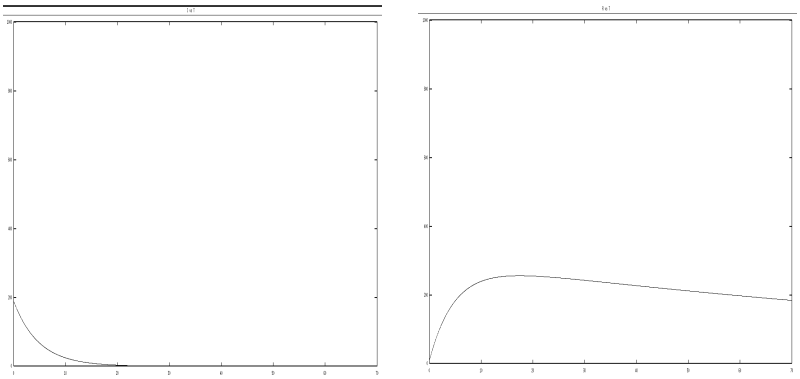


Figure 4: I vs T and R vs T for params: $\beta = 0.1$, N = 10000, $\gamma = 0.3$, b = 0.02, $\mu = 0.007$ ($R_0 < 1$)

3.3 Generalisations of SIR model

- SEIR : E stands for exposed. COVID-19 can be thought of as an example here, where before getting infected, there's a stage where a susceptible might come in contact with an infected individual and hence move from susceptible category to exposed category.
- SEIQR : Q stands for quarantined. Once an infected individual gets stable again, still there might be traces of germ/virus in body of the individual which may spread onto another susceptible individual and hence the need of quarantine for a period.
- SEIRS : the flow goes as $S \to E \to I \to R \to S$, i.e. a recovered individual might again become susceptible. An example can be thought of in terms of the COVID-19 vaccine, after 18 months or so, the effect of vaccine is expected to fade out and the individual will lose his/her immunization and move again into susceptible category.

4 Epidemic Modelling : Study of Ruan and Weng's work

4.1 Spread under Intervention Measures

4.1.1 Equation Modelling

For studying the infection force under *intervention policy*, we are going to consider SIRS model, where removed individuals who lose immunity move back to susceptible class at the rate ν. Also the rate of transmission of infection is modified as a function and hence the rate at which individuals move from susceptible to infected compartment also changes. Instead of considering a constant rate, we treat β as a function of I. Hence, bringing in these changes, our system of equations look like:

$$\frac{dS}{dt} = bN - dS - \beta(I)S + \nu R$$

$$\frac{dI}{dt} = \beta(I)S - (d+\gamma)I$$

$$\frac{dR}{dt} = \gamma I - (d+\nu)R$$

To represent the intervention policies such as lockdowns, we consider infection force $\beta(I)$ as $\lambda I/f(I)$ where $1/f(I)$ represents our intervention policies and λ is a constant. Using this our equations become:

$$\frac{dS}{dt} = bN - dS - \frac{\lambda I}{f(I)}S + \nu R$$

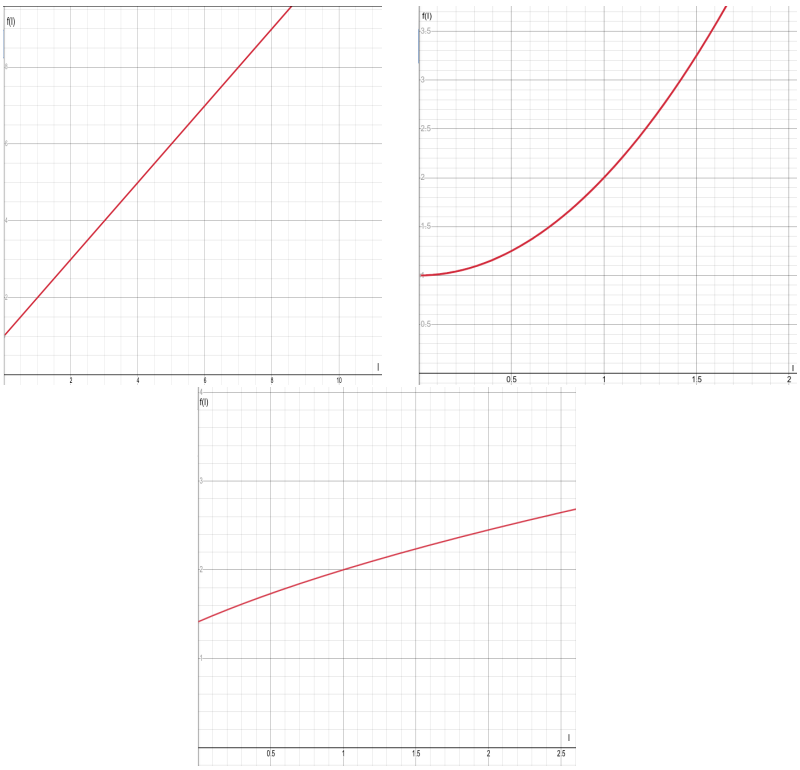
$$\frac{dI}{dt} = \frac{\lambda I}{f(I)}S - (d+\gamma)I$$
$$\frac{dR}{dt} = \gamma I - (d+\nu)R$$

Assuming population to be fixed (C) at a moment, we get:

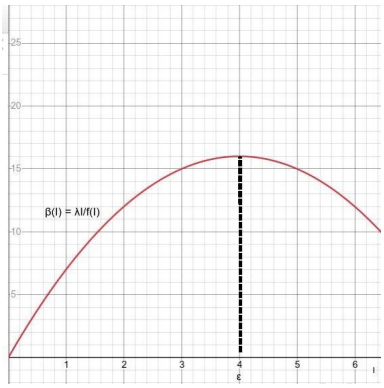
$$\frac{dI}{dt} = \frac{\lambda I}{f(I)}(C - I - R) - (d+\gamma)I$$
$$\frac{dR}{dt} = \gamma I - (d+\nu)R$$

Now to ensure a non monotonic infection force, we make the following assumption:

- (A1) : $f(0) > 0$ and $f'(I) > 0$ for $I > 0$. Following are some visualisations of possible $f()$ according to A1 :



- (A2) : there is a $\xi > 0$ such that the infection force increases, $\beta(I)' > 0$, i.e., $(I/f(I))' > 0$ for $0 < I < \xi$ and then decreases, $\beta(I)' < 0$, i.e.,$(I/f(I))' < 0$ for $I > \xi$. Following is a possible visualisation :



Realisation on f() :

- In case no intervention is adopted for the spread of the disease, $f(I) = 1$.
- But the reverse isn't always the case, i.e., if $f(0) = 1$, may also mean that we perform intervention policies only at a suitable infection level. (maybe when I is too high)
- f is an increasing function, denoting increasing intervention with increase in I which is count of infected individuals. (Note that it reasonable to assume $f(I)$ as an increasing function as stronger intervention policies will be employed if outbreak is serious)

4.1.2 Equilibrium Analysis

Disease free equilibrium : We have $E_0 = (0, 0)$ as our disease free equilibrium, where $I = 0$. We can find the basic reproduction number in general as in previous section, at $I = 0$, $R_0 = \frac{C\lambda}{f(0)(d+\gamma)}$. Corresponding Jacobian matrix at E_0 is:

$$\begin{bmatrix} \frac{\lambda C}{f(0)} - d - \gamma & 0 \\ \gamma & -d - \nu \end{bmatrix}$$

It follows that E_0 is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Endemic equilibrium : For an endemic equilibrium, another solution of the above equations is as follows:

$$\begin{cases} R = C - I - \frac{d+\gamma}{\lambda} f(I), \\ R = \frac{\gamma}{d+\nu} I. \end{cases}$$

From (A1) it is easily observed that there is no endemic equilibrium if $R_0 < 1$ and there is a unique endemic equilibrium if $R_0 > 1$. Let it be $E^* = (I^*, R^*)$

Claim : A unique endemic equilibrium exists for $R_0 > 1$ that is globally stable.

Proof:

The Jacobian at E^* is:

$$J = \begin{bmatrix} \frac{\lambda(C-2I^*-R^*)}{f(I^*)} - d - \gamma - \frac{\lambda I^*(C-I^*-R^*)f'(I^*)}{f^2(I^*)} & -\frac{\lambda I^*}{f(I^*)} \\ \gamma & -d - \nu \end{bmatrix}$$

From above equations, we have:

$$f(I^*) = \frac{\lambda(C - I^* - R^*)}{d + \gamma}$$

On solving, we obtain det(J) and trace(J) as follows:

$$\det(J) = \frac{I^* \left(f'\left(I^*\right)\left(d\gamma + \gamma\nu + \nu d + d^2\right) + \lambda(d + \gamma + \nu)\right)(d + \gamma)}{\lambda\left(C - I^* - R^*\right)} > 0$$

$$\text{trace}(J) = \frac{I^* \left(f'\left(I^*\right) d^2 + \lambda\gamma + 2f'\left(I^*\right) d\gamma + f'\left(I^*\right)\gamma^2 - \lambda\nu\right) + \lambda\left(C - R^*\right)(d + \nu)}{\lambda\left(-C + I^* + R^*\right)}$$

On further solving, we obtain that trace has same sign as:

$$\lambda I^*\nu - \lambda C\nu - I^* f'\left(I^*\right) d^2 - 2I^* f'\left(I^*\right) d\gamma - I^* f'\left(I^*\right)\gamma^2 - \lambda C d$$

Now this is negative as $I^* < C$ and $f'\left(I^*\right) \geq 0$. Hence the eigenvalues of J have negative real parts implying E^* is asymptotically stable.

We introduce 2 functions,

$$\begin{cases} F1 = C - I - \frac{d+\gamma}{\lambda} f(I), \\ F2 = \frac{\gamma}{d+\nu} I. \end{cases}$$

Bendixson–Dulac theorem on dynamical systems states that if there exists a function $\varphi(x, y)$ (called the Dulac function) such that the expression

$$\frac{\partial(\varphi f)}{\partial x} + \frac{\partial(\varphi g)}{\partial y}$$

has the same sign ($\neq 0$) almost everywhere in a simply connected region of the plane, then the plane autonomous system

$$\begin{aligned} \frac{dx}{dt} &= f(x, y) \\ \frac{dy}{dt} &= g(x, y) \end{aligned}$$

has no nonconstant periodic solutions lying entirely within the region.

Choosing a Dulac function as $Q = f(I)/I$, we have

$$\frac{\partial\left(QF_1\right)}{\partial I} + \frac{\partial\left(QF_2\right)}{\partial R} = -\lambda - (d + \gamma) f'\left(I^*\right) - \frac{f(I)}{I}(d + \nu) < 0$$

when $I > 0$. Thus, the system of equations pertaining to endemic equilibrium have no nonconstant periodic solutions and hence no limit cycle in the region $I > 0$. Thus, we show that E^* is globally stable in the region $I > 0$.

4.1.3 Conclusion

- If we adopt to perform intervention only at a suitable infection level, the basic reproduction number, R_0 remains unchanged.

  Reason : In absence of intervention, $R_0 = \frac{C\lambda}{(d+\gamma)}$. If we adopt to perform intervention only at a suitable infection level, $f(0) = 1$, discussed in *Realisation on f()* section, hence the basic reproduction number, $R_0 = \frac{C\lambda}{(d+\gamma)}$, remains unchanged.

  So, consider the equation,

  $$R = C - I^* - \frac{d + \gamma}{\lambda} f(I^*)$$

  consider R as tending to the basic reproduction number,

  $$C - I^* - \frac{d + \gamma}{\lambda} f(I^*) = constant$$

  The variables in above equation is only I^*, $f()$ is an increasing function that increases with I, hence the value of I must be less than that in the absence of the intervention. Thus, the intervention policy for controlling the disease decreases the endemic level.

- We also found that E^* is globally stable, hence we expect no complicated behaviors of the model.

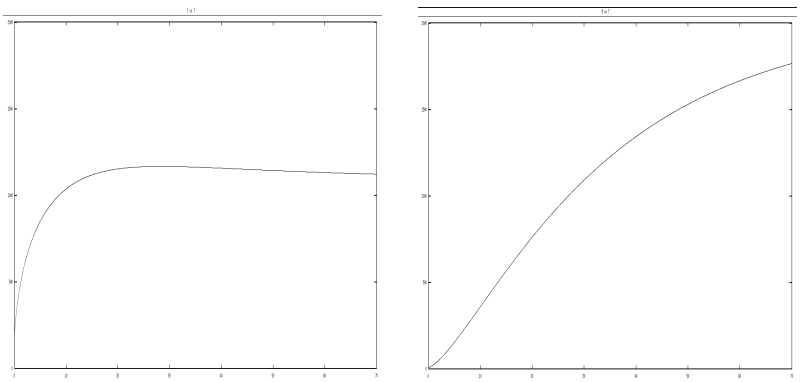


Figure 5: I vs T and R vs T for params: $\lambda = 10$, C = 10000, $\gamma = 0.05$, $\nu = 0.02$, d = 0.007 ($R_0 > 1$)

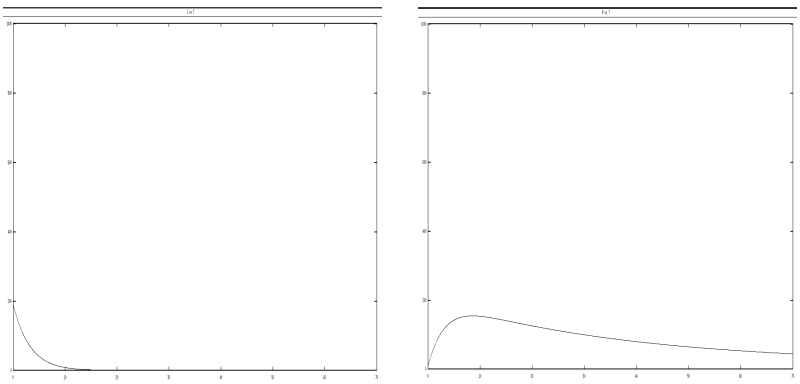


Figure 6: I vs T and R vs T for params: $\lambda = 0.0001$, C = 10000, $\gamma = 0.3$, $\nu = 0.02$, d = 0.007 ($R_0 < 1$)

4.2 Spread under saturated infection force

4.2.1 Equation Modelling

Now we will consider our infection force to be saturated and redo the analysis. We assume that infection force $\beta(I)$ can be now written as $Ig(I)$ where g is continuously differentiable. We get the following equations:

$$\frac{dS}{dt} = bN - dS - Ig(I)S + \nu R$$

$$\frac{dI}{dt} = Ig(I)S - (d+\gamma)I$$

$$\frac{dR}{dt} = \gamma I - (d+\nu)R$$

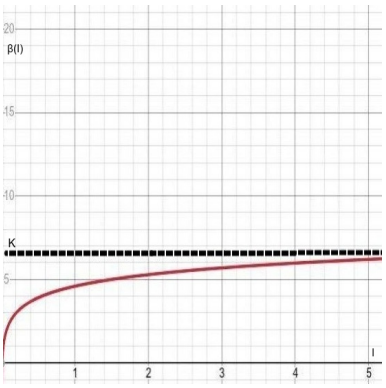
Other two equations change as follows:

$$\frac{dI}{dt} = Ig(I)(C - I - R) - (d+\gamma)I$$

$$\frac{dR}{dt} = \gamma I - (d + \nu)R$$

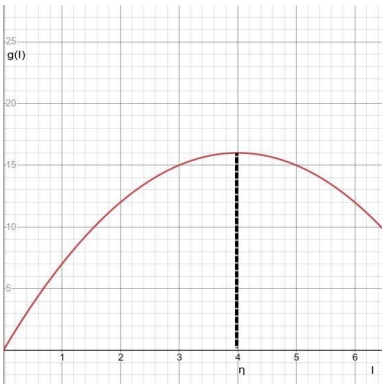
Again we make assumptions regarding our infection force:

- (A3) : $g(I) \geq 0$ is bounded for $I \geq 0; Ig(I) \to k$ as $I \to \infty$ Following is a possible visualisation :



- (A4) : $(Ig(I))' > 0$ for $I > 0$; there is an $\eta > 0$ such that $g'(I) > 0, I \in (0, \eta)$ and $g'(I) < 0, I \in (\eta, \infty)$, and such that $g''(I) \leq 0$ for $I \in (0, \eta)$

  Note when $\eta = 0$, since g is decreasing after $I > \eta$, it is similar to the reciprocal of $f(I)$ function studied in the previous section, hence we need not to do any further analysis for $\eta = 0$. Following is a possible visualisation :



4.2.2 Equilibrium Analysis

Disease Free Equilibrium : Clearly $E_0 = (0, 0)$ is a disease free equilibrium and Jacobian at E_0 is:

$$\begin{bmatrix} g(0)C - d - \gamma & 0 \\ \gamma & -d - \nu \end{bmatrix}$$

It follows likewise as in previous section that E_0 is asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.
We can get the basic reproduction number by trying to equate $\frac{dI}{dt} = 0$ at $I, R = 0$, i.e., the disease free equilibrium, hence, $R_0 = g(0)C/(d + \gamma)$.

Endemic Equilibrium : The solutions of the following equations would give us the endemic equilibrium :

$$\begin{cases} R = C - I - \frac{d+\gamma}{g(I)}, \\ R = \frac{\gamma}{d+\nu} I \end{cases}$$

Let us try to make it a $1-$ variable system, so we substitute R from the bottom equation into the top equation to get :

$$C - (\frac{(d+\nu+\gamma)}{d+\nu} I + \frac{d+\gamma}{g(I)}) = 0$$

$$\implies \frac{(d+\nu+\gamma)}{d+\nu} I + \frac{d+\gamma}{g(I)} = C$$

Let us denote the LHS of the above equation in terms of variable I, i.e., we define a function F() as follows :

$$F(I) = \frac{(d+\nu+\gamma)}{d+\nu} I + \frac{d+\gamma}{g(I)}$$

We are required to find solutions for $F(I) = C$ for finding the solutions pertaining to endemic equilibrium.

Let us look at the first and second derivatives of F,

$$F'(I) = 1 + \frac{\gamma}{d+\nu} + \left(-\frac{d}{g^2(I)} - \frac{\gamma}{g^2(I)} \right) g'(I),$$

$$F''(I) = 2\frac{(d+\gamma)}{g^3(I)} \left(g'(I)\right)^2 - \frac{(d+\gamma)}{g^2(I)} g''(I),$$

We know that $g(I)$ is increasing and $g(I)' > 0$ for $I \in (0, \eta) \implies F'' > 0$ for $I \in (0, \eta)$. This further leads to $F(I)$ either is an increasing function or has an minimum at $\bar{I} \in (0, \eta]$ owing to (A3) and (A4). Few cases arise henceforth :

- If $R_0 > 1$, $F(I) = C$ has a unique positive solution I^*, and therefore the system has a unique endemic equilibrium $E^* = (I^*, R^*)$ with $R^* = \gamma I^*/(d+\nu)$.
- if $R_0 < 1$ and $F(\bar{I}) < C$ $F(I) = C$ has two positive roots $0 < I_1 < I_2$, which implies that we have two endemic equilibria: $E_1 = (I_1, R_1), E_2 = (I_2, R_2)$ with $R_i = \gamma I_i/(d+\nu)$.
- If $R_0 < 1$ and $F(\bar{I}) = C$, the system has a unique endemic equilibrium $\bar{E}$.
- If $R_0 < 1$ and $F(\bar{I}) > C$, the sysyem has no endemic equilibrium.

Stability of E^* : As observed in case 1 above, if $R_0 > 1$, we have an endemic equilibrium E^*. We make the following claim regarding its stability.

Claim : E^* is stable when:

$$\textit{stable bound} : g'(I^*) < \frac{C\nu - I^*\nu + Cd}{I^* \left(-C + I^* + R^*\right)^2}$$

and unstable when:

$$\textit{unstable bound} : g'(I^*) > \frac{C\nu - I^*\nu + Cd}{I^* \left(-C + I^* + R^*\right)^2}$$

Proof:

The Jacobian at E^* is:

$$J_1 = \begin{bmatrix} (C - I^* - R^*)\left(g\left(I^*\right) + I^* g'\left(I^*\right)\right) - I^* g - d - \gamma & -I^* g \\ \gamma & -d - \nu \end{bmatrix}$$

Then,

$$\det\left(J_1\right) = g'\left(I^*\right) I^*(d+\nu)\left(-C + I^* + R^*\right) + D$$

where

$$D = (d+\nu)(d+\gamma) + g\left(I^*\right)\left(-Cd - C\nu + 2dI^* + 2I^*\nu + dR^* + R^*\nu + \gamma I^*\right)$$

From above equations, it follows:

$$D = \frac{(d+\gamma)I^*(d+\nu+\gamma)}{C - I^* - R^*}$$

By the analysis for the existence of E^*, we see that $F'\left(I^*\right) > 0$. Thus,

$$g'\left(I^*\right) < \frac{g^2\left(I^*\right)(d+\nu+\gamma)}{(d+\nu)(d+\gamma)} = \frac{(d+\gamma)(d+\nu+\gamma)}{\left(-C + I^* + R^*\right)^2 (d+\nu)}$$

It follows that $det(J_1 > 0)$, implying that stability is now determined by the trace. Note that,

$$\operatorname{trace}\left(J_1\right) = g'\left(I^*\right)\left(I^*\left(C - I^* - R^*\right)\right) + \left(C - 2I^* - R^*\right) g\left(I^*\right) - 2d - \gamma - \nu$$

Substituting values we get,

$$\operatorname{trace}\left(J_1\right) = g'\left(I^*\right) I^*\left(C - I^* - R^*\right) - \frac{\nu\left(C - I^*\right) + Cd}{C - I^* - R^*}.$$

It follows that $\operatorname{trace}\left(J_1\right) < 0$ if *stable bound* holds and $\operatorname{trace}\left(J_1\right) > 0$ if *unstable bound* holds. Therefore, E^* is stable if *stable bound* holds and unstable if *unstable bound* holds.

Further Analysis:

- *How do the other equilibrium's behave?*
  Let us discuss a bit further about the stability of the 2 endemic equilibria, though we will not go into further details of it. The analysis for the existence of E_1 and E_2 implies $F'\left(I_1\right) < 0$ and $F'\left(I_2\right) > 0$. If J_{21} and J_{22} are the Jacobian matrices corresponding to the system at E_1 and E_2, then by similar discussions as those in proving the stability of E^*, we see that $\operatorname{trace}\left(J_{21}\right) < 0$, implying E_1 is a saddle and trace $\left(J_{22}\right) > 0$, implying E_2 is a node or focus.
- *Possibility of Saddle Node Bifurcation at* $R_0 < 1$:
  Thus, for the case $R_0 < 1$, as I moves, actually $F(\bar{I})$ varies, we initially find 2 endemic equilibria, one saddle, another node or focus at $F(\bar{I}) < C$, changing to an unique endemic equilibrium at $F(\bar{I}) = C$, finally leading to disappearance of the equilibrium as $F(\bar{I}) > C$. Thus, this means that the model may have a saddle node bifurcation.

4.2.3 Conclusion

The saturated infection force in reality describes "crowding effect" or "protection measures." Indeed, effective contacts between infectious individuals and susceptible individuals cannot grow quickly when there are many infectious individuals because of the crowding of infective individuals or because of protection measures by susceptible individuals.

- We witness the disease free equilibrium and an unique endemic equilibrium for the case $R_0 > 1$, just as we had found in the model with intervention forces.
- However, the major difference that we witness here is in the case $R_0 < 1$, where there may exist saddle node bifurcation. Hence we find the possibility of the rich dynamical behavior of a disease evolution that can be induced by a saturated infection. force.

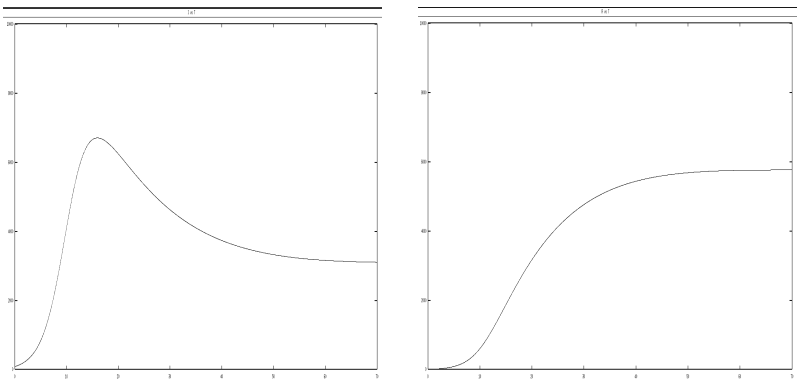


Figure 7: I vs T and R vs T for params: g(0) = 0.1, C = 10000, $\gamma = 0.05$, $\nu = 0.02$, d = 0.007 ($R_0 > 1$)

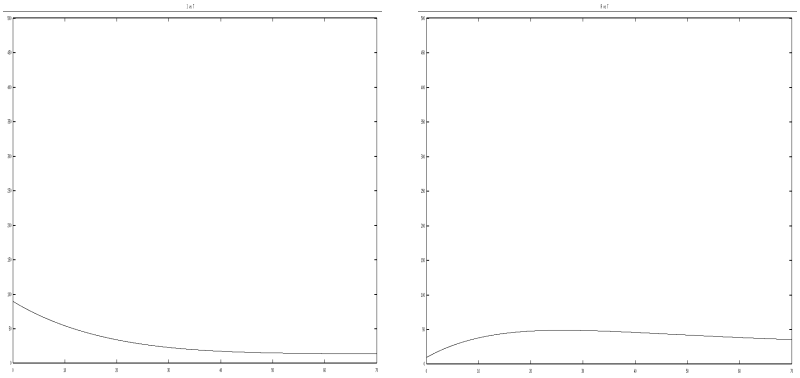


Figure 8: I vs T and R vs T for params: g(0) = 0.00001, C = 10000, $\gamma = 0.3$, $\nu = 0.02$, d = 0.007 ($R_0 < 1$)

5 Proposing a Network Centrality based Intervention Model

5.1 Introducing Percolation Centrality[5]

Percolation of a 'contagion' occurs in complex networks in a number of scenarios. For example, viral or bacterial infection can spread over social networks of people, known as contact networks. The spread of disease can also be considered at a higher level of abstraction, by contemplating a network of towns or population centres, connected by road, rail or air links.

In the epidemiological domain, a few studies have successfully modelled epidemic spread as a specific example of percolation in networks. For instance, Newman and Watts[6] suggested using a site percolation model for disease spreading in which some fraction of the population is considered susceptible to the disease, and an initial outbreak can spread only as far as the limits of the connected cluster of susceptible individuals in which it first strikes. An epidemic can occur if the system is at or above

its percolation (epidemic) threshold where the size of the largest (giant) cluster becomes comparable with the size of the entire population.

In any context, if we need to stop the contagion from spreading further, we need to supply nodes with certain resources. For example, during a disease outbreak affecting a network of towns, medical staff, medicine and other resources need to be rushed to each town to stop the infection from spreading to other towns as well as to treat people in that town. Generally, there are limited resources (vaccines, drugs, medical staff, transport, etc.) to respond in time. Therefore, choices for early intervention in the affected network need to be precise. However, 'nodes' that are individually at the highest risk are not necessarily those which will contribute most to the contagion transmission. Hence, there is a need to identify nodes that are 'central' in terms of their impact on the spread. Moreover, we need to interpret the node's impact both in terms of their topological connectivity and their current infected (percolated) state.

In 2012, Piraveenan, Prokopenko, Hossain proposed a centrality measure, *percolation centrality*, a measure that quantifies the above extent, how critical a node is in the spread, based not only on the topological connectivity but also the percolation state of the nodes. Formally, it is defined as the proportion of 'percolated paths' that go through that node. A 'percolated path' is a shortest path between a pair of nodes, where the source node is percolated (e.g., infected).

$$PC^t(v) = \frac{1}{(N-2)} \sum_{s \neq v \neq r} \frac{\sigma_{s,r}(v)}{\sigma_{s,r}} \frac{R\left[x_s^t - x_r^t\right]}{\sum_{s \neq v \neq r} R\left[x_s^t - x_r^t\right]}$$

The percolation state of node i at time t is denoted by x_i^t. Specifically, $x_i^t = 0$ indicates a non-percolated state at time $t, x_i^t = 1$ indicates a fully percolated state at time t, while a partially percolated state corresponds to $0 < x_i^t < 1$. It uses the Ramp function R, defined as $R(x) = x$ for positive x and $R(x) = 0$ for negative x.

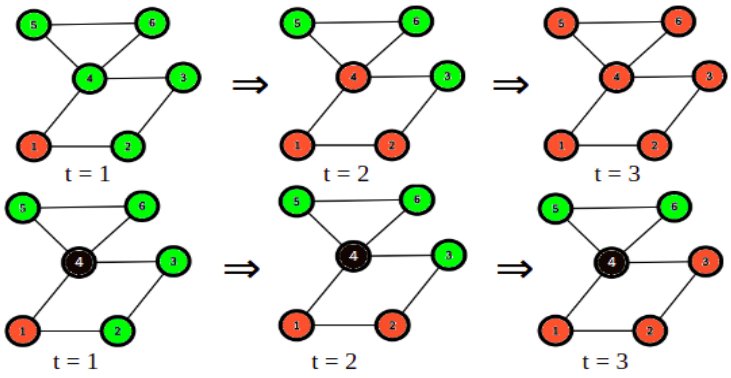


Figure 9: Red denotes a fully percolated (infected) node while green denotes an unpercolated one. Suppose the contagion spreads one unit at each timestep, node 4 shows high percolation centrality and should be shielded (black) to prevent spread.

5.2 Proposing PC based Intervention Model

The aim is to device an intervention model for a city which has been divided into zones, which have adopted individual intervention and have been observing respective infection forces, which might not be alike to other zones.

In a classical intervention model exploiting percolation centrality, mostly x_i^t, i.e., the percolation at zone i would have been taken as the ratio of infected individuals $(=\frac{I}{N})$ at time step t, maybe on the t-th day. Now on basis of the percolation centralities estimated at time t, the zones would

have been blocked or shielded for commute on t-th day. Rather than taking daily decisions and intervention measures at larger level as in the above scenario, if we could figure out a slightly long term solution keeping in mind the local scenario, how the infection force is turning out at each zone respectively, what are the feasible interventions at respective zones, it could be immensely helpful. The goal can be thought of as a way to distance the zones as much as possible and take local decisions regarding crucial interventions at certain periods at the zonal level while at the larger level, we are just to be worried about which zones to shield and still have a spanning path network in the city.

Our model is to adopt the following steps :

- Decide on the time T, at an interval of which the intervention measures are to be restrategised for the zones.
- We devise a percolation function on basis of the Non Linear Dynamic model. It is tough obtaining a closed form solution for the nonlinear couple system of ODEs we observed in section 4.1, i.e., on $\frac{dI}{dt}$ and $\frac{dR}{dt}$, hence we plan to use computer simulations to realise I, R, $\frac{dI}{dt}$ and $\frac{dR}{dt}$ at specific time points. We obey the following function :

$$x_i(I_i, R_i, C_i, T) = p \cdot \frac{I_i}{C_i} + q \cdot \frac{1}{N} \frac{d(I_i - R_i)}{dt}$$

  where I is the number of infected cases after a time period of T from the last instant the intervention measures were strategised, C is the total population expected after time period of T (may vary as per the birth rate and death rate), $\frac{dI}{dt}$ and $\frac{dR}{dt}$ are the rates of infection and recovery as previously defined after a period of T days. N is the total number of zones. p and q are the weights associated with proportion of infected individuals and difference in rates of infection and recovery after time period T. To put it in sense, the nonlinear dynamic model using the computer simulations helps as a predictive model how shall the situation be after a period of T days.
- Now estimate the percolation centralities of each of the zones using the Proposed algorithm to compute Percolation Centrality based on modified x_i (check Algorithm 1).

Algorithm 1 Proposed algorithm to compute Percolation Centrality based on modified x_i

1: Run *All pairs shortest path algorithm* to calculate the shortest distances from each of the nodes to others. *Floyd Warshall* is a suitable candidate for the same. Note that not just distance, we also require the count of shortest paths between every pair of nodes/zones. Let $dist_{i,j}$ denote the distance of zone i to j and $\sigma_{i,j}$ denote the count of shortest paths between i and j.
2: **for** each zone $i \in Zones$ **do**
3: **for** each zone $j \in Zones$ **do**
4: **for** each zone $k \in Zones$ **do**
5: **if** $dist_{i,k} + dist_{k,j} == dist_i, j$ **then**
6:

$$PC^T(k) = \frac{1}{N-2} \cdot \frac{\sigma_{i,k} \cdot \sigma_{k,j}}{\sigma_{i,j}} \cdot \frac{R[x_i(I_i, R_i, C_i, T) - R[x_j(I_j, R_j, C_j, T)]}{\sum_{s,r} R[x_s(I_s, R_s, C_s, T) - R[x_r(I_r, R_r, C_r, T)]}$$

- Note that we may run simulations to check which weights would be suitable for p and q in the above definition of $x_i()$. Also, this function is a simple prediction on what shall be the severity of the situation in a zone after T days, we could bring in a lot of other factors as well on basis of behaviour of the system in a zone.
- As percolation centralities are estimated, now decisions can be made on this basis, which nodes are to be shielded and which commute modes are to be blocked, i.e, if we find $PC^t(k) \geq \alpha$

(again a parameter), we can either choose to shield k or we can also choose to block the commute modes passing through k through which the infection is transmitted over from i to j through k, hence erasing the percolated paths through k, which lead to its high percolation centrality.

Based on Peeraveenan's paper, simulation of contagion spread using a simple real world network has been shown below :

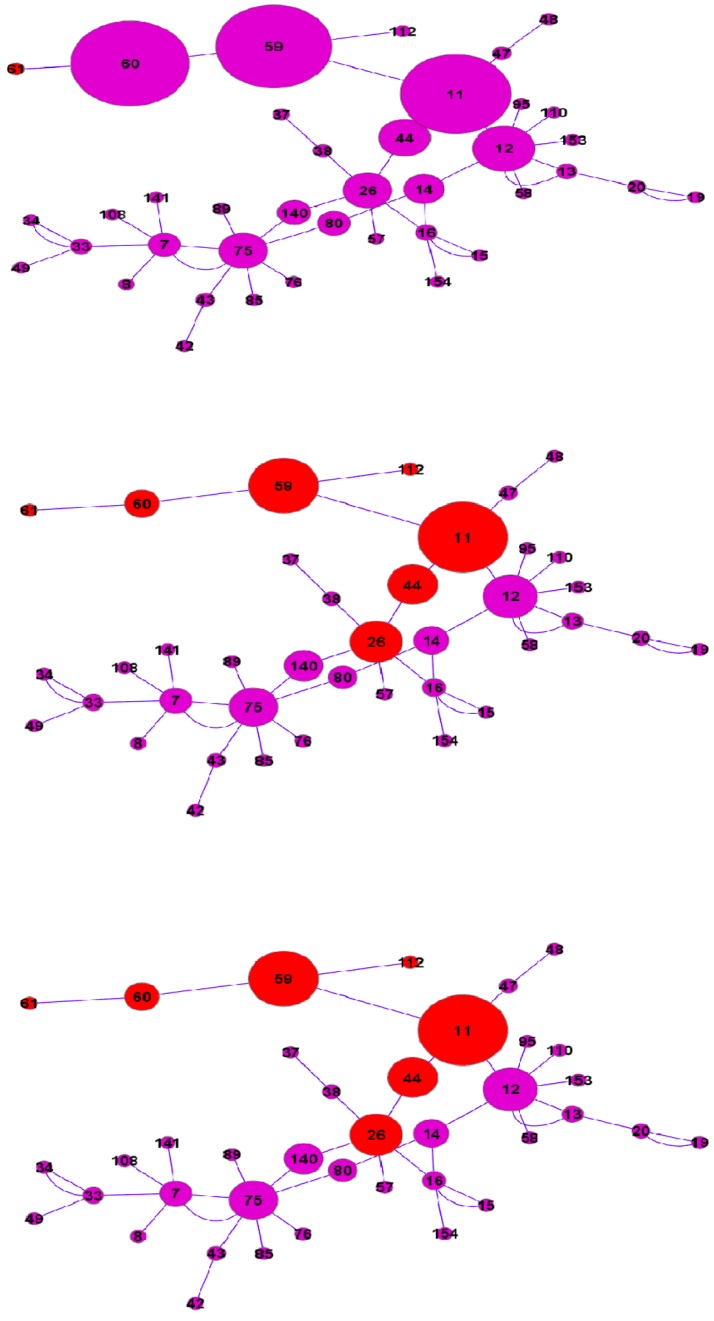


Figure 10: (a) The percolation centrality of nodes at $T = 1$. (b) The percolation centrality of nodes at $T = 7$. (c) Percolation centrality of nodes at $T = 20$, All these simulations have no intervention at all, hence the spread has been quite fast, transmission only possible through direct contact and with a probability 0.2.

6 Future Scope

6.1 Spread under quadratic infection force

We have studied two different kinds of infection forces. One is when we have appropriate intervention measures. And the other is the case when infection force is a saturated one. On similar lines, we can also study a quadratic infection force with saturation[7]. Here our $g(I)$ is defined as:

$$g(I) = \lambda \frac{I}{1 + pI + qI^2}$$

Note that we had earlier derived these two equations:

$$\frac{dI}{dt} = Ig(I)(C - I - R) - (d + \gamma)I$$

$$\frac{dR}{dt} = \gamma I - (d + \nu)R$$

Substituting $g(I)$ here, we get:

$$\frac{dI}{dt} = \lambda \frac{I^2}{1 + pI + qI^2}(C - I - R) - (d + \gamma)I$$

$$\frac{dR}{dt} = \gamma I - (d + \nu)R$$

We can derive that there is no endemic equilibrium if:

$$\lambda C \leq p(d + \gamma)$$

It is found that equilibrium analysis is greatly affected by the term:

$$\frac{\lambda C}{d + \gamma}$$

Thus we define:

$$\mathcal{R}_1 = \frac{\lambda C}{d + \gamma}$$

From [8], $\mathcal{R}_1$ can be treated as the number of primary contacts of an infected person. The condition for no endemic equilibrium, then reduces to:

$$\mathcal{R}_1 < p + 2\sqrt{q + \lambda \frac{d + \gamma + \nu}{(d + \gamma)(d + \nu)}}$$

- Increase in population size (C), increase the possibility of an endemic equilibrium. This can be compared to daily rise in COVID cases in India as C is very large.
- Increase in parameters such as p and q makes the infection force less severe which increase the possibility of endemic to die out.
- Increase in γ means that more and more people are recovering which will also help in reducing the extent of epidemic.

6.2 Modelling the effect of vaccination[9]

Earlier we had looked how to take intervention policies under consideration by taking infection force $\beta(I)$ as $\lambda I/f(I)$. Here we assumed that all the intervention policies are collectively captured by this force. However we can also model measures such as vaccination separately. In such a case we would get $\beta(I)$ as a highly complex function which could capture various governmental effects separately in a precise manner. Though, we have avoided going into much details of it and run simulations of them, here's a brief overview of it.

There are 2 types of vaccination strategies, continuous vaccination strategy (CVS) and pulse vaccination strategy (PVS). For certain kinds of infectious diseases, PVS is more affordable and easier to implement than CVS. We need to introduce a variable to gauge the effect of vaccination on our model. To be simply put it follows :

$$S(t^+) = (1-p)S(t)$$
$$I(t^+) = I(t)$$
$$R(t^+) = R(t) + pS(t)$$

where, constant p is the vaccination rate. However in reality, vaccination is often restricted by medical resources. Moreover, vaccination success rate has a saturation effect, hence the vaccination rate can also be described as a saturation function :

$$p(t) = \frac{pS(t)}{S(t)+\theta}$$

where, p is the maximum pulse immunization rate and θ is defined as the half-saturation constant; that is, the number of susceptible when the vaccination rate is half to the largest vaccination rate. Hence $S(t^+)$ takes a changed form as:

$$S(t^+) = (1-p(t))S(t)$$

7 Summary of the Work

Through this project, we have come to learn about the mathematical models built for studying epidemics. Below is a summary on the work details involved in the project :

- We started with understanding the very basics of SIR models, without and with vital dynamics, understand what do SIR, SIRS, SEIR, SEIQR and SEIRS signify.
- Next we studied the survey paper "Epidemic Models with Nonlinear Infection Forces" by Wendi Weng, which had followed the previous work of Ruan and Weng, where we learnt of the effect of forces concerned with the intervention as well as a saturated infection force, studied the proofs of existence of the equilibrium points, disease free equilibrium and endemic equilibrium.
- *Constructing time course simulations using Xppaut* for the coupled system of nonlinear ODEs in each of the above models, we observed their dynamics.
- We studied and discussed briefly about percolation centrality, as proposed by Piraveenan, Prokopenko, Hossain and using that and the nonlinear dynamic models we studied so far, we propose an intervention model based on percolation centrality algorithm for a large area consisting of multiple smaller zones. However, the computation would require $O(N^3 \cdot S)$, where S is constant time to run the simulations on NLD model. Hence, we have *implemented a parallel percolation centrality computation algorithm in CUDA*.
- During the course of reading for the project, we explored some topics which we didn't go into greater depth and hence have put in the future scope section.

All the implementation work has been italicized above and can be found in the *code* folder.

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