On the interaction between social opinion dynamics and epidemic propagation

by

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Abstract

The response to an epidemic, in terms of the degree to which healthcare protocols are followed, is not the same for all individuals. Some may follow these strategies to the fullest extent, while some may dismiss them as superfluous. We shall be referring to these responses as opinions regarding the healthcare policies(or protocols). A spectrum of such opinions can be observed in society ranging from fully following to does not follow at all. Intuitively, one could deduce that it is these opinions that determine the longevity of an epidemic. Moreover, these opinions are not static. Each individual possesses the potential to update their opinion based on the opinions of their neighbors[1][2][3][4]. In this research, we derive a relationship between the dynamics of infection propagation and the opinion dynamics concerning healthcare policies. In other words, we wish to observe how these two quantities vary with respect to one another. We construct three models- the opinion dynamics model - independent of the number of infections, the epidemic propagation model- dependent on the opinions of individuals, and the opinion dynamics model -as a function of the number of infections in a given neighborhood. We then perform a stability analysis on each of these models to prove convergence. Also, we attach simulation results to show how these models progress with time.

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Chapter 1

Introduction

The dissemination of infections is significantly influenced by the degree to which each individual in a population follows the preventive measures set by the government[5]. These can be in the form of healthcare strategies such as maintaining social distancing, vaccination,wearing face gear,and using hand sanitizers. The extent to which an individual adopts these measures determines his/her vulnerability towards an infection.

Epidemiology is a widely studied domain targeted to understand the spread of infections in society[6][7][8][9]. Several models have been proposed to understand the dynamics of infection spread [10][11][12][13]. These include the SIS, SIR, SEIR, and SAIR models[14][15][16][17]. In the SIS model, the population is divided into two − the infected and the susceptible. For the SIR model, an individual is capable of transitioning between the susceptible, infected, and recovered states. SAIR and SEIR constitute the category of larger compartmental models with additional states such as exposed, asymptotic, and removed.

A key factor in the study of epidemic propagation is the basic reproduction number or the basic reproductive number denoted by R_0[18]. R_0 gives a measure of

the degree to which an emerging infection can spread in a population. Mathematically, an R_0 value less than one implies that the epidemic would eventually die out. Conversely, an R_0 value greater than one implies that the epidemic would become endemic[19].

Several factors affect the R_0 value. Some of these are the duration of infectivity, number of susceptible individuals that come in contact with that infected, and infectiousness of the disease. Healthcare measures such as vaccination, social distancing, use of protective gears,also have an impact on R_0. The Australian Department of Health adds the absence of "any deliberate intervention in disease transmission" to the definition of basic reproduction number[6].

It can be observed that in the absence of intervention strategies, such as the ones mentioned above, the life of infection can be dictated by the laws of nature alone. Looking at this from the perspective of a controls engineer, we can say that the system lacks control inputs[20][21]. And these control inputs are the intervention strategies that could allow the population to reach a disease-free state.

A volume of research can be found dedicated to studying the impact of preventive protocols like social distancing and vaccination on the propagation of infections[18] [22] [23] [24]. A known way of incorporating these measures is by treating them as additional states to the compartmental model[22]. This approach seems reasonable when dealing with one or two measures. But, as the number of preventive measures increases, it becomes computationally cumbersome to analyze each of them independently. Moreover, the kind and the degree to which each individual adopts these measures differ. For example, a person following social distancing might not believe in vaccination, or a vaccinated person might not follow social distancing. Also, these opinions could change with time, resulting in a random assortment of adoption levels in the population.

An interesting line of study in the form of dynamics of influence spread or influence maximization exists devoted to understanding the spread of ideas in a social network[25] [26] [27]. Initially, it was used as a marketing tool to solve the problem of maximizing the diffusion of a product[27][28][29]. The problem statement being, '' If a product is to reach/be adopted by a large section of the society, who should the initial customers be." Another study performed in this domain is that of opinion propagation. Opinion dynamics is an opinion evolution process of a group of agents, where the final opinion distribution tends to three stable states: consensus, polarization, and fragmentation[30].

In this research, we utilize the techniques of opinion dynamics and infection propagation to develop a problem formulation of the title. We begin by posing a model for the spread of influence, in the form of a state-space equation, independent of the propagation dynamics of infections(a). Next, we present the propagation dynamics of infections taking into account the individual beliefs or opinions regarding health care policies(b). Finally, we rewrite the influence spread model as a function of the state each individual is in (c)- in the range of healthy to infected.

For the Influence spread model, we follow [25], except, the existence of an edge does not necessarily imply physical interaction/acquaintance. It merely points at a node that lies in the neighbourhood of the node in question, that influences the opinion of that node, depending on the weight[31] assigned to it.

We model each node's opinion on health care policies as a measure of the degree to which it physically follows these policies. And this value, proceeding as per [25], is represented as a continuous scalar ranging from 0 to 1, with 0(1) representing a fully follows situation(does not follow at all situation). All the values in-between correspond to the partially follows case.

Each node has an initial opinion, which it updates by observing the opin-

ions of those in the neighbourhood. Also, each node possesses some degree of stubbornness[25] with respect to its initial opinion.

In the analysis of part (a) of the paper, we let the stubbornness index be an arbitrary constant. For the part(c), we treat the stubbornness index as a varying quantity, dependent on the number of infections in the neighbourhood of the node i. We assume that the stubbornness of a node changes(lowers) as the number of infections around it increase.

The state of a node is defined in the range of healthy(0) to infected(1). State 0(1) implying nil contagiousness(maximum contagiousness). The intermediate values correspond to the different stages of infectivity. A population is said to be devoid of the infection when all the nodes reach state 0.

The usefulness(validity) of the proposed models is tested by analysing their steady state behaviours. We conclude our work by presenting results from the simulations.

Chapter 2

Related Work

In this chapter, we discuss the works in the literature that are related to the scope of this thesis. In [32], the effect of social impact on epidemics was investigated using the techniques of mean-field analysis and extensive simulations. For the influence spread model, each node was considered to be either in the active or the passive state. Subsequently, the behavioral patterns of these nodes were modeled according to the social impact theory of [33]. It was shown how the opinions of individuals on public healthcare strategies affect an infection spread. But, a converse to it, the impact of an infection spread on these opinions was not shown. Also, the stubbornness indices were ignored.

In our work, we will be studying the simultaneous variation of the dynamics of epidemic propagation with the opinion dynamics associated with the degree to which each node follows the health-care policies.

[22] and [23] investigated the impact of vaccination and social distancing on the spread of epidemics, respectively. [22] treated vaccination as an additional state to the considered compartmental model. And based on that, derived the basic reproduction number. Consequently, it was checked to see if the disease-free equilibrium

and the endemic equilibrium converge.

In [23], a mobility analysis was performed to study the effect of social distancing on the basic reproduction number(R_0) by constructing a mobility function that included the average mobility of a given population before, in transition, and after the implementation of social distancing.

[25] has addressed the convergence issues of opinion dynamics in the presence of stubborn agents.In the works of [25], each individual was treated as an agent in a social network. These agents were initially given a scalar value from 0 to 1 - representing an opinion regarding a commodity. Subsequently, an update rule was formulated for the change in the opinion of an agent i in response to the opinions of his neighbors. Finally, it was checked to see if these opinions converged to a finite value.

Chapter 3

Models and definitions

In this section we will be presenting the influence spread model - independent of the propagation dynamics of infection, the infection model- dependent on the opinion dynamics concerning the adoption of healthcare policies, and the influence spread model - dependent on the propagation dynamics of infection.

3.0.1 Influence Spread Model(a)

Consider a social network with n nodes, denoted by a graph $G(V, E)$ where nodes are the vertices and edges indicate the pairs of nodes that have interactions. For each node i, define its neighborhood δ_i as the set of nodes that node i interacts with. Let k(0)=$[k_1(0)....k_n(0)]^T$ denote the vector of initial adoption levels.

Each node tries to approach the average of the adoption levels(of the healthcare strategies)in his neighbourhood, weighted by strength of each edge. Also, each node possess some degree of stubbornness towards his initial opinion.

Based on that, we assume each node i has a cost function of the form

$$U(k_i, k_{\delta_i}) = \frac{1}{2}(k_i - \sum_{j \in \delta_i} \frac{\theta_{ij} k_j}{\sum_{j \in \delta_i} \theta_{ij}})^2 + \frac{K_i}{2}(k_i - k_i(0))^2 \tag{3.1}$$

that he tries to minimise[25].

Where K_i refers to the stubbornness of node i and θ_{ij} corresponds to the strength of the edge ij, $\theta_{ij} \neq \theta_{ji}$, $\theta > 0$. The first term accounts for the distance between k_i and the centre of mass of the neighbouring system of k_j's,weighted by their strengths.

Upon minimizing k_i we obtain:

$$k_i = \sum_{j \in \delta_i} \frac{\theta_{ij} k_j}{(1 + K_i) \sum_{j \in \delta_i} \theta_{ij}} + \frac{K_i k_i(0)}{1 + K_i} \tag{3.2}$$

We use a game theory approach to formulate the best response strategy for node i. We consider the following continuous-time best response strategy

$$k_i'(t) = -k_i(t) + \sum_{j \in \delta_i} \frac{\theta_{ij} k_j}{(1 + K_i) \sum_{j \in \delta_i} \theta_{ij}} + \frac{K_i k_i(0)}{1 + K_i} \tag{3.3}$$

Similar best response dynamics for discrete time analysis can be seen in the works of [25][34].

3.0.2 Infection Spread Model(b)

We consider a social network of n nodes denoted by $G(V, E)$. The vertices(nodes) represent the individuals in a population, and an edge indicates a pairwise interaction. Each node, initially, is either in the healthy state(x = 0) or the infected state(x = 1). Also, we consider the adoption levels of each node while formulating the propagation dynamics of the infection. These k values multiplied with the state give the effective contagiousness of that node.

Assumption 2

1. G is an undirected graph (we can apply the same analysis on any undirected subgraph $Gc \subseteq G$)

2. Transmission of infection occurs when at least one of the neighbours of the node i is infected and does not completely follow the suggested healthcare policies ($k > 0$). Also, the node i should be in the healthy state.

Let λ be the transmission coefficient[35] associated with the transition from the healthy to the infected state and γ be the recovery rate.

Based on the above assumptions, we can model the infection dynamics as

$$x_i'(t) = \lambda \sum_{j \in \delta_i} k_j(t) x_j(t) k_i(t)(1 - x_i(t)) - \gamma x_i(t) \tag{3.4}$$

3.0.3 Influence Spread Model with dependency on the fraction of nodes infected

We use the same model as in Part a, except we consider a stubbornness index of the form

$$Q_i(x_j) = \begin{cases} K_i - K_i(\sum_{j \in \delta_i} \frac{x_j(t)}{d_i} - b_i), \sum_{j \in \delta_i} \frac{x_j(t)}{d_i} - b_i > 0 \\ K_i, \ \sum_{j \in \delta_i} \frac{x_j(t)}{d_i} - b_i < 0 \end{cases} \tag{3.5}$$

Where b_i, $0 \leq b_i \leq 1$ corresponds to the threshold for i to lower its stubbornness index in response to the number infections in his surroundings.

The revised update rule for k would be,

$$k_i'(t) = -k_i(t) + \sum_{j \in \delta_i} \frac{\theta_{ij} k_j(t)}{(1 + Q_i(x_j)) \sum_{j \in \delta_i} \theta_{ij}} + \frac{Q_i(x_j) k_i(0)}{1 + Q_i(x_j)} \tag{3.6}$$

Chapter 4

Analysing the steady state behaviour

In this section we analyse the steady state behaviour of the models presented above.

4.0.1 Influence Spread Model(a)

Consider the best response dynamics of 3.3,

Define an $n \times n$ matrix T such that

$$T_{ij} = \begin{cases} \frac{\theta_{ij}}{(1+K_i)\sum_{j\in\delta_i}\theta_{ij}}, (i,j) \in E \\ 0, \ otherwise \end{cases} \tag{4.1}$$

Also, define an $n \times n$ matrix B such that $B = \frac{K_i}{1+K_i}, 1 \leq i \leq N$.

The update rule in the matrix form is given by

$$k'(t) = Ak(t) + Bk(0), \text{ for } A = -I + T$$

We make the following assumptions for analysing the steady state behaviour,

Assumption 1

1. G is a directed graph (we can apply the same analysis on any directed subgraph ,i.e, $G_c \subseteq G$).
2. At least one of the nodes is stubborn, i.e.,$K_i > 0$, for at least one $i \in V$
3. No nodes in the system are isolated.

Proposition 1

At $t \to \infty$, we get the steady state value of k as

$$k(\infty) = -A^{-1}Bk(0)$$

Proof

T is substochastic matrix under assumption 1 with the row sum of at least one row less than one. Let $\rho_1(T) = max_i|\lambda_i(T)|$ denote the spectral radius of T. Then, $\rho_1(T) < 1$ for a substochastic matrix[25]. Hence, we can infer,

$$\lambda(A) < 0$$

following

$$\lambda(A) = \lambda(-I + T)$$

We conclude that as all the eigenvalues of A are negative, our system is asymtotically stable. Also as all the eigenvalues of A are non-zero, A^{-1} exists.
The equilibrium value(steady state value of k) can be calculated as

$$k'(\infty) = Ak(\infty) + Bk(0)$$

At equilibrium $k'(\infty) = 0$,

$$k(\infty) = -A^{-1}Bk(0)$$

4.0.2 Infection Spread Model(b)

Consider the propagation dynamics of 3.4,

The equilibrium points of (3.4) can be calculated by finding $x_1'(t) = x_2'(t)... = 0$ By making the above substitutions, we arrive at a system of N equations and N unknowns. Thus, a solution in the form of $x^* = [x_1^* \ x_2^* \dots]$ exists, which is the equilibrium point of the system. Let $k^* = [k_1^* \ k_2^* ...]$ be the equilibrium point for the influence propagation model.

By linearizing the system around x^*,we obtain

$$\frac{\partial f_i}{\partial x_j} \mid_{x=x^*} = \lambda k_j^* k_i^* (1 - x_i^*), i \neq j$$

$$\frac{\partial f_i}{\partial x_i} \mid_{x=x^*} = -\lambda \sum_{j \in \delta_i} k_j^* x_j^* k_i^* - \gamma$$

as the non-diagonal and the diagonal entries of the Jacobian, J, respectively.

For the Infection propagation model to reach a disease free equilibrium ($x^* = 0$, for all i), a sufficient condition is given by

$$\gamma > \lambda k_i^* \sum_{j \in \delta_i} k_j \tag{4.2}$$

4.0.3 Influence Spread Model with dependency on the fraction of nodes infected

Consider equation 3.6,

Let Q^* be the equilibrium point for $Q(x_j)$.

Linearizing around $k = k^*$, we obtain

$$\frac{\partial f_i}{\partial k_i} \mid_{k=k^*} = -1, i \neq j$$

$$\frac{\partial f_i}{\partial k_j} \mid_{k=k^*} = \frac{\theta_{ij}}{(1+Q_i^*)\sum_{j\in\delta_i}\theta_{ij}}$$

as the diagonal and the non-diagonal entries of the Jacobian, J, respectively.

J can be rewritten as $J = -I + P$, for

$$P_{ij} = \begin{cases} \frac{\theta_{ij}}{(1+Q_i^*)\sum_{j\in\delta_i}\theta_{ij}}, (i,j) \in E \\ \\ 0, \ otherwise \end{cases} \tag{4.3}$$

P is an substochastic matrix under assumption 1 with the row sum of at least one row less than one. Let $\rho_1(P) = max_i|\lambda_i(P)|$ denote the spectral radius of P. Then, $\rho_1(P) < 1$ for a substochastic matrix[25]. Hence, we can infer,

$$\lambda(J) < 0$$

following

$$\lambda(J) = \lambda(-I + P)$$

We conclude that as all the eigenvalues of J are negative,hence, our system is asymtotically stable.

Chapter 5

Results and Simulations

We perform the simulations on a social network comprising of 3 nodes. We consider a fully connected graph for all the models. Thus, each node has two neighbours.

For the transmission coefficient, $\lambda = 0.8$, and the recovery rate, $\gamma = 0.7$, the results of the simulations are as follows:

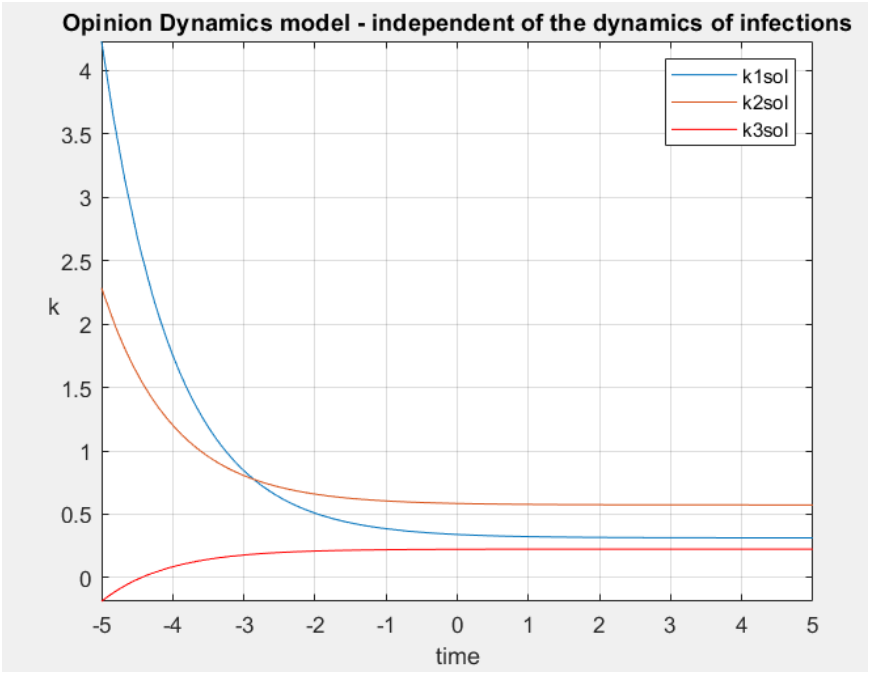

Figure 5.1: Influence spread model(a)

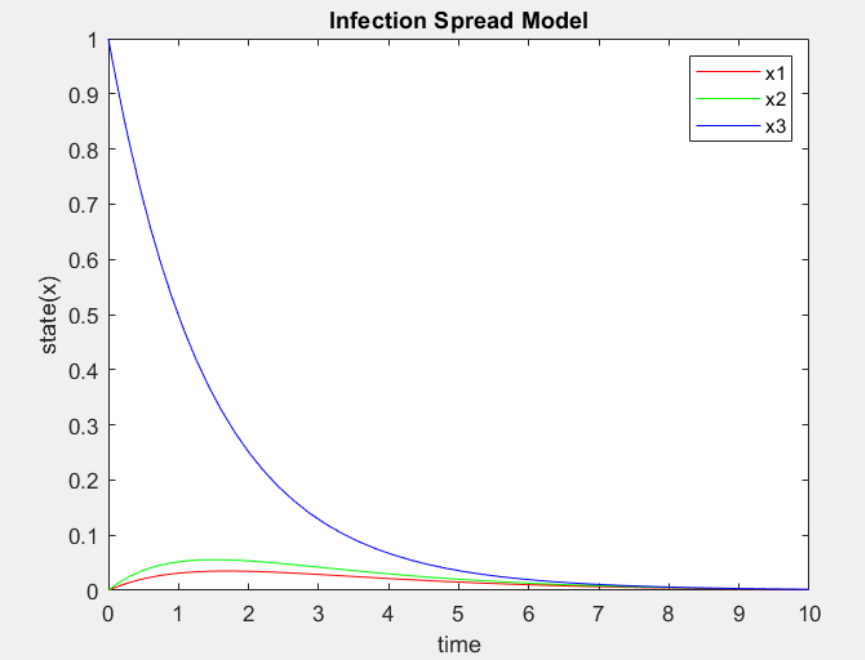

Figure 5.2: Infection spread model

We initialize the k values at [0.340 ; 0.585 ; 0.223]. We observe that, at $t \to \infty$, the influence spread model approaches a steady state value at the points $k = [0.3140, 0.5738, 0.2254]$ for the three nodes.

For the infection spread model, we initialize the states of the nodes at x = [0,0,1]. We start with two healthy and one infected node.

At $t \to \infty$, we can see that, the states corresponding to the infection model go to zero i.e., $x^* = 0$. Thus, we attain a disease-free equilibrium.

And as $t \to \infty$, $\quad Q_i^* = K_i$, from 3.5

Chapter 6

Conclusion

In this thesis, we have analysed the variation of the propagation dynamics of infections with the opinion dynamics associated with the degree of adoption of the healthcare policies and vice versa. We began by showing that the opinion dynamics, independent of the dynamics of infections, converged to a finite value under certain assumptions. Using similar assumptions, we were able prove that the propagation dynamics of infections, as a function of k, converged to a disease free state for a given range of γ. Lastly, by treating the stubbornness index as a function of the fraction of nodes infected in the surroundings of a node, we were able to observe how opinion dynamics varied with the propagation dynamics of infections. Also, we were able to prove that these revised opinion dynamics converged to a finite value. Further, through simulations performed on the first two models, we were able to see that the opinion dynamics, independent of x_j, converged to a finite value ,as calculated, and our infection model converged to a disease free state($x = 0$). And as the infection model converged to a disease free state, the stubbornness index, Q , as a function of x_j, converged to K.

Chapter 7

Scope for future work

We did not consider the existence of forceful agents- these are the individuals(nodes) that do not change their decisions, the analysis of which requires a different mathematical approach[25]. The nodes were incapable of migration. This migratory behavior of nodes changes the structure of the social network, resulting in the occurrence of different networks with time. The focus of our analysis was on deriving the sufficient conditions for the infection spread model to reach disease-free equilibrium. The case where an endemic equilibrium occurs was not analyzed. Moreover, birth rates and death rates were not considered.

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Chapter 8

Appendix

Proof for the sufficient condition on γ

For the system to be asymptotically stable at the equilibrium point x^*, all eigenvalues of J should be negative(i.e., J should be negative definite). Utilizing the Gershgorin Circle theorem, we get the centre at

$$c \equiv \lambda \sum_{j \in \delta_i} k_j^* x_j^* k_i^* - \gamma$$

And the radius

$$R \equiv \lambda k_i^* (1 - x_i^*) \sum_{j \in \delta_i} k_j^*$$

For asymptotic stability, all the eigenvalues of the system should lie in the negative half of the complex plane.

Thus,the condition,
Distance of the origin to the centre $> R$, holds

$$\lambda \sum_{j \in \delta_i} k_j^* x_j^* k_i^* + \gamma > \lambda k_i^* (1 - x_i^*) \sum_{j \in \delta_i} k_j^*$$

$$\gamma > \lambda k_i^* (1 - x_i^*) \sum_{j \in \delta_i} k_j^* - \lambda \sum_{j \in \delta_i} k_j^* x_j^* k_i^*$$

$$\gamma \ > \ \lambda k_i^* \sum_{j \in \delta_i} k_j^* ((1 - x_i^*) - x_j^*)$$

At disease-free equilibrium,

$$\gamma \ > \ \lambda k_i^* \sum_{j \in \delta_i} k_j, \text{as } x^* = 0$$