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Social Network – A New Perspective in Mathematical Epidemiology

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***Abstract*— In this paper we introduce a modified mathematical model to represent spread of disease, which adheres to the structure and dynamics of social network. We also discuss the importance and effectiveness of social network based study in the prediction and control of epidemics.**

***Index Terms*— Mathematical epidemiology; Social network; Mathematical models; Core.**

I. INTRODUCTION

First remarkable study in mathematical epidemiology is the study of smallpox by Daniel Bernoulli in 1760. But widely accepted models in mathematical epidemiology were proposed by public health physicians such as Sir Ross, R. A., W. H. Hamer, A. G. McKendric and W. O. Kermak between 1900 and 1935. These models are called compartmental models because they are obtained by dividing the host population into different compartments such as Sunciptible (S), Infected (I), Recovered (R) etc. For a detailed study of this subject the text edited by Fred Brauer et al. [1] is a better choice. The compartmental models such as SIS, SIR etc. are oversimplified with the assumption that the host population is homogenous and contacts between members are same throughout the population and steady over time. As evident from the literature on mathematical study of epidemics, this simplification has led to much variation between theory and practice.

Diseases spread among members of a society due to many reasons. Person to person contact is one among them, which is the main focus of our study. Our society is heterogeneous and human contact depends on the personal factors such as likes, dislikes, needs etc. There are some external factors such as disease monitoring and control mechanism, mass campaigning about the disease, availability of drugs and preventive medicines etc. In the initial stage of the spread of a disease people do not have proper information about the occurrence of the disease and in many cases authority many be weak in implementing right preventive measures. People may not be aware of the nature and consequence spread. Later as the disease progresses to an epidemic or endemic, more and more people become aware of the situation and they tend to reduce contact with infected people and in some cases society itself declares quarantine against diseased. As disease become stronger in infected people, they normally have to take bed-rest which results in some kind social withdrawal. All the above mentioned factors greatly affect social contacts between infected and susceptible and hence control the spread of disease in the later stage. Many previous studies have shown the effect of contact network and human behavior in the spread of disease. One is the study about the spread of gonorrhoea in the United States [2], which showed 60% all infections were caused by a small group of individuals that constitute only 2% of the whole population. In this study we try to incorporate some of these factors while modifying existing models. The outbreak of SARS in the year 2003 also hints about the role of structure in mathematical epidemiology [3].

In the next section we review a simple models being used in epidemiology, the SIS model and discuss its characteristic features. We also discuss its disadvantage and weakness in explaining some phenomenon. Then we move to the third section in which some networks with different structures are selected and analyzed to prove the weakness of homogenous mixing models. A revised model is given in the fourth section and its effectiveness in the analysis of disease dynamics is discussed.

II. THE SIS MODEL

For a person who studies the mathematics of epidemics, SIS model is a starting point. This model is obtained by dividing the host population (N stands for the total size of the population) into two compartments; the Susceptible (S) and the Infected (I). Initially it is assumed that the whole population contains only susceptible people and a disease is newly introduced into the population. The disease spreads among the members when they come in contact other members. A third assumption is that people do not acquire permanent immunity against the disease. A person, who enters the compartment, returns to S after some time, which varies from disease to disease. So the SIS model is suitable to represent only those models which do not develop prolonged immunity. Let β be the average rate at which a member contacts with others. So the total contact one person can make in the population is βN. The probability of a random contact with a susceptible is S/N. Thus the number of new infections per individual per unit time is $(\beta N)(S/N)$ and the rate of new infection is obtained by multiplying this quantity by I. So the number of people who leave the compartment S to join I is βSI. If γ is the rate of recovery, γI is the number of people who are recovered per unit time. Thus we get the following differential equations which model the disease dynamics.

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$$\begin{aligned} \frac{dS}{dt} &= -\beta SI + \gamma I \\ \frac{dI}{dt} &= \beta SI - \gamma I \end{aligned} \qquad (1)$$

This model is built on the assumption that all members have same kind of contact with a specific percentage of other members. The above equations represent a network in which members have homogenous type of mixing with the rest of the population. Graph theoretic model of the network depicted above is strictly a regular graph. A regular graph is a graph in which all vertices have same number of degree. Next we proceed to discuss how variations in network structures affect dynamics of disease spread in contact networks.

III. Regular Graphs and Spread of Disease

To simplify the analysis, we assume that initially one individual is infected ($I_0 = 1, S_0 = N-1$) and in turn he infects $\beta SI = \beta(N-1)$ others in unit time. If we fix one unit of time as period, during the next period, we have the following values for N, I and S respectively.

$$\begin{aligned} N_1 &= N \\ I_1 &= \beta(N-1)1 \\ S_1 &= (N-1)+\gamma \end{aligned} \qquad (2)$$

Again repeating this for the next period, we get a more complicated set of equations, which are functions of N. Generalizing the above steps, we can develop a set of recurrence relations, which give the values of N_i and S_i .

$$\begin{aligned} N_i &= N \\ I_i &= \beta(S_{i-1})I_{(i-1)} - \gamma I_{(i-1)} \\ S_i &= \beta(S_{i-1})I_{(i-1)} + \gamma I_{(i-1)} \end{aligned} \qquad (3)$$

By substituting the values of I and S obtained in previous steps, we can show that all compartments are expressed in terms of the total population.

Now we consider a strict binary tree structure (fig: 2) for the contact network. A strict binary tree is a binary tree, in which each vertex, except the leaves have two branches. Assume that a person occupying the position v_1 is infected. He then infects a portion of individuals in his neighbourhood. These infected people infect the other people in their neighbourhood union and so on. In graph theory, an open neighborhood of a vertex v which is denoted by $N(v)$ is the set of all vertices which are connected to that vertex. The closed neighbourhood of a vertex, $N[v]$ is the union of its open neighbourhood and the vertex itself (i.e., $N[v] = N(v) \cup \{v\}$). In a binary tree if the infection is at the root, the disease has to pass through four steps to infect all members. On the other hand, if a leaf vertex is infected, eight steps are necessary to cover the entire population. Thus time taken by the disease to infect the whole population depends on who is infected and not on the size of the population. Next we consider another contact network known as an n-dimensional hypercube graph. We can label the vertices in a hypercube, which contains 2^n vertices using the $n-$ tuple $(x_1 x_2 \ldots x_n)$, where $x_i = 0$ or 1 . Two vertices are adjacent if and only if there is exactly one change in the values of x_i in the corresponding positions. In this network time taken to cover the entire network is same irrespective of the position from where we start. So this network is highly homogenous unlike the previous network.

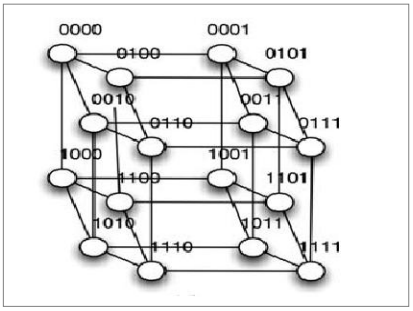

Figure 1. Three dimensional Hypercube.

Disease can spread equally in all directions in this type of networks. The standard deviation of the degrees of vertices is almost zero in both. Clearly S. D. of degree of vertices is not a factor which decides the propagation of disease in a network.

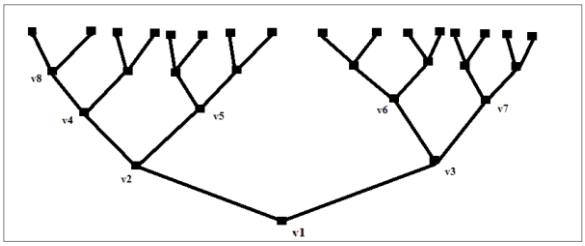

Figure 2. A strict binary tree.

Another highly homogenous regular graph network is a complete graph network. In this network every member is connected to every other member. An n vertex complete graph contains $n(n-1)/2$ edges and it is regular with degree $(n-1)$. If a vertex in a network having complete graph structure is infected, then it can transmit disease to all other members in single step and can produce an epidemic within a short period. This character of the vertex is due to the fact that the closed neighbourhood of every member is the entire population. It motivates us to define the core of a network. It is the set of vertices in a network, such that the union of the closed neighbourhoods of the members of the set spans the entire network. Each member in a complete graph network is a core. Core is a very crucial idea in the study of epidemic. If an infection occurs in the core, it can definitely spread rapidly in one or two steps and thus it can cause a pandemic.

IV. Other Networks

A. Star Structured Networks

A star in graph theoretic terminology is a graph, which contains a vertex that is connected to all other vertices having degree one. So there is only one vertex having degree greater than one. This vertex is called hub in the terminology of network science.

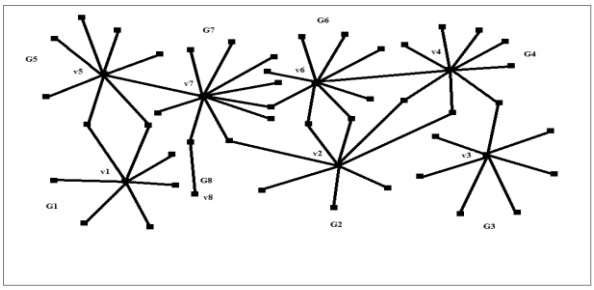

Figure 3. A star network

A star structured network is a union of many stars. So it can be decomposed into small star sub-graphs $G_1, G_2, \ldots, G_n$ having hub vertices $v_1, v_2, \ldots, v_n$. The core of the graph is $\{v_1, v_2, \ldots, v_n\}$. A star structured network is highly vulnerable to disease because it can spread over the whole network in one or two steps if the first infection is at all vertices in the core. On the other hand if only a part of the core is initially infected then the disease has to take many steps to spread over the network depending on the network topology.

B. Random Networks

Random networks are artificial networks. They do not possess any specific structure. So they are not representative of any natural networks. But they provide a platform for comparative study of structured networks. Random networks are identified with their degree distribution. They have either a Poisson degree distribution (if the network size is very big) or a Binomial distribution (if network size is very small).

C. Scale-free Network

Scale-free networks are the networks in which there are a large number of vertices of small degree and very small number of vertices of large degree. Thus the degree distribution obeys a power law. The large degree vertices are called hubs. So a scale-free network contains a small collection of hubs. The neighbourhood union of this set spans almost the entire network. So it behaves like a core. If disease affects a considerable number of members of the core, it quickly spreads in the networks. If it affects a set of members who have very law connectivity, the rate of spread may decrease and ultimately die out. So the question, which part of the population is affected by the disease is very important in the study.

V. The Model

In the light of above discussion, we propose a new model to represent spread of disease in a structured network. We assume that the process contains only infected and susceptible. Nobody is recovered permanently and birth and death does not occur in the population. Let T be the infective period for a disease. This is the average time an infected remains infective. We divide the whole period of time in subintervals having length T and T_i denote the i th interval. Let I_i, S_i and $N(I_i)=\cup_{x\in I_i} N(x)$ be the infected, susceptible and the open neighbourhood set of the infected members, respectively in the beginning of the i th interval. We assume that the population size is steady and I_0 is initial set of infected. Then, $S_0 = N - I_0$. $N(I_0)$ is the set of all members who are in contact with I_0. Due to many factors such as weak contact, natural resistance developed by internal mechanism against disease etc., all of them may not be infected. So we introduce a probability factor here. Let α be the probability of a contact resulting into an infection. Then the total number of infection is $\alpha|N(I_0)|$. By time T all infected might have become susceptible. Thus after time T we get,

$$\begin{aligned} I_1 &= \alpha|N(I_0)| \\ S_1 &= N - \alpha|N(I_0)| \end{aligned} \qquad (4)$$

After time $2T$,

$$\begin{aligned} I_2 &= \alpha|N(I_1)| \\ S_2 &= N - \alpha|N(I_1)| \end{aligned} \qquad (5)$$

Generalizing the equations, we get,

$$\begin{aligned} I_{(n)} &= \alpha|N_i(I)| \\ S_{(n)} &= N - \alpha|N_i(I)| \end{aligned} \qquad (6)$$

To get the total number of infections throughout the period of prevalence of disease, we can use the formula,

$$|I| = \sum_{i=0} I_i | \qquad (7)$$

and the probability of infection is roughly obtained by

$$\alpha = \frac{\sum_i I_i|}{\sum_i N(I)|} \cdot \qquad (8)$$

In the model given above, it assumed that the average time T_1 for transmission of disease from one group of infected to the set of individuals in its neighbourhood is same as the average rate of recovery T_2. If $T_2 < T_1$, an infected recovers faster than he makes a susceptible person, infected. So strength of the disease decreases and if $T_2 << T_1$, ultimately the disease dies out. On the other hand, if $T_1 < T_2$, disease spreads faster in the neighbourhoods and it grows gradually. If the inequality is very strong, then the disease turns out to be a pandemic.

On the outbreak of a disease, through mass media and campaigning programmes, people are made aware of the disease. It affects the attitude of the people towards the disease and they resort to some self defensive mechanism. For example, they keep themselves away from the diseased person reducing their public contacts. Consequently, the associated network

shows changes in the structure and hence it becomes necessary to consider dynamic graphs instead of static one.

VI. Further On Core

Here we generalize the concept of core to incorporate the structure of a network and to make the idea more meaningful and applicable. The second order closed neighbourhood of a set $S \subseteq V(G)$ is the subset $N[S]=N[N[S]]$ and the third order closed neighbourhood is $N[S]=N[N[N[S]]]$. This concept can be generalized to obtain the n^{th} order closed neighbourhood of the set S $N[S]=N[N^{n-1}[S]]$ We define the span number of a graph $\alpha(G,S)$ with respect to a given subset $S \subseteq V.$ as the smallest number n such that $N^n[S]=V.$ If $G=K_n$ the complete graph containing n vertices, then $\alpha(G,\{v\})=1$ for all $v \in V.$ If $G=K_{(1,n)}$ where $n \geq 2.$ $V=\{v,v_1,v_2,\ldots v_n\}$ and $d(v)=n$ then $\alpha(G,\{v\})=1$ and $\alpha(G,\{v_i\})=2$ for all i. From the second example it is clear that for two subsets S_1 and $S_2,$ $\alpha(G,S_1)$ and $\alpha(G,S_2)$ may be different. We can prove the following results on closed neighbourhood and span number of subsets of $V.$

Lemma – 1: For any two subsets S_1 and S_2 of V

[1] $N[S]=N[S_1] \cup N[S_2]$

[2] $N[S]=N[S_1] \cap N[S_2]$

Proof of 1: For any two vertices v_1 and v_2 $N[v_1,v_2]=N[v_1] \cup N[v_2]$ Generalizing it for an arbitrary set, we get $N[S]=N[S_1] \cup N[S_2]$

Proof of 2: Applying 1.1 repeatedly n times, we get lemma 2.

Theorem – 2: For any two subsets S_1 and S_2 of V such that $S_1 \subset S_2$, $\alpha(G,S_2) \leq \alpha(G,S_1)$

Proof: Since $S_1 \subset S_2$, S_2 can span the whole graph in $\alpha(G,S_1)$ steps. But S_2 contains more vertices than $S_1.$ So, it may be possible to cover the graph in lesser number of steps.

The above relations are important in the determination of spread of disease. Correct information about the higher order neighbourhoods of a set of infected is very crucial in deciding the policies of disease control. Vaccinating all members of the neighbourhood of infected is a better choice than doing it for the whole population, as a temporary, quick and cost effective measure.

VII. Importance Of Network Structure In Control Of Disease

From the discussions so far, it is clear that the knowledge about the spread and related dynamics of disease is very important in all stages of control and management of disease. Spread of disease in the network is controlled by the network topology. While vaccinating against disease, vaccinating the whole population is costly and very difficult. But if the members of the core are vaccinated first, authorities could effectively control the disease. Widely used models like SIS and SIR do not account for network structure. Heterogeneity is of least importance in these models and uses the assumption that each member is likely to contact with any other member of the population. Analyses of original data have shown wide differences of theory from reality. This has motivated us to develop a new model, in which contact of a person in his neighbourhood and structure are the main concern. We can modify it further incorporating the factors affecting dynamism of network to get much improved models.

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K. R. Kumar has fifteen years of teaching experience and ten years of research experience. He has been supervising six students for Ph. D. Two scholars have completed their research for the award of Ph. D. He has ten international publications and five national publications in his credit.