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STOCHASTIC MODELING OF CHICKENPOX EPIDEMICS ON NETWORKS

By

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Abstract

Chickenpox (also called varicella) is a highly contagious illness caused by an herpes virus called varicella zoster, and is one of the commonly reported childhood diseases. The objective of this study is to examine the effect of vaccination and human contact interactions on the transmission of chickenpox virus in general; and in particular, the plausibility of elimination of chickenpox disease in Idah Community, Kogi State, Nigeria. Data on contact interaction among 100 young children in Idah Community were collected. A graph representing this population and their interconnectedness was generated. The effects of different converges of vaccination on the epidemiology of chickenpox virus were investigated. The results show that when children reduce the number of contacts with their peers, the number of chicken pox cases reduces. However, based on the contact data collected, effective chickenpox vaccination is crucial for the elimination of chickenpox among the children in Idah community.

1. INTRODUCTION

Chickenpox (also called varicella) is an infectious disease characterized by fever and a blister-like rash caused by varicella-zoster virus (VZV). The varicella-zoster virus (VZV) causes two distinct clinical infectious diseases: chickenpox (varicella) and shingles (zoster). Chickenpox is the primary infection caused by the varicella-zoster virus. After recovery from chickenpox, the virus can be dormant in a nerve root. Reactivation of latent infection causes herpes zoster [1,2].A person of any age can contract chickenpox if there was no immunity through previous infection or vaccination [1,2].Varicella zoster virus is airborne and highly communicable. Approximately 90% of close contacts with those who are non-immune will contract chickenpox after exposure to persons with the disease. A person with varicella is contagious from 1-2 days before rash onset until the sores have crusted. The incubation period is approximately 10-21 (mean 14-16) days after exposure to the virus. VZV is spread from person-to-person through direct contact with the skin rash or through the air by inhaling respiratory droplets [1,2, 3].

Fever and malaise may occur 1 to 2 days before rash onset, particularly in adults. In children, the rash is often the first sign of the disease. The rash usually appears first on the head, chest and back; and then spreads to the rest of the body. The rash is itchy and progresses rapidly from flat sores to fluid-filled blisters before crusting. Asymptomatic infection (without rash or with very few blisters) may occur. Classically, lesions appear in crops, and are therefore present in different stages of development – some at the papule, others at the blister and still others at the crusted stage [3].

The varicella vaccine is highly effective in preventing varicella [1,2] and treatment of uncomplicated varicella in children is usually confined to symptomatic relief [1].

In this paper, we present and simulate a stochastic network model of chickenpox epidemic. The plan of this paper is as follows. Graphs and modeling is presented in section 2. Section 3 is devoted to model description. Data collection is presented in section 4. Simulation, results, discussion and conclusive remarks are passed in sections 5, 6, 7 and 8 respectively.

2. Graphs and Modeling

Classical epidemiological models ignore the importance of the complex patterns and structures of social interactions on the spread of diseases. So, most of the earlier epidemiological models trivialize the social aspects of disease transmission. However, since the middle of the twentieth century, sociologists, mathematicians have been studying social networks and have come up with a large literature spanning many different aspects of social networks from empirical, conceptual and methodological points of view [[4]

Graphs used in the literature can be classified based on the properties of interest. From the dynamism point of view, graphs or networks can be classified as static or dynamic depending on whether their structures change with time. From the field of application perspective, we have social networks, information networks, technological networks, epidemic networks, to mention a few. Each of these types of networks can be narrowed to specific networks. Graph classifications based on degree distribution exist. For instance, scale-free graphs, Poisson graphs. Graphs such as unipartite, bipartite or multipartite are based on the node types. For a general knowledge of graphs and their theory, refer to [5, 6,7, 8, 9, 10, 11, 12, 13, 14, 16].

Real world network are large, and in most cases it is virtually impossible to describe them in detail or to give an accurate model for how they came to be. To circumvent this problem, random graphs have been considered as network models. The field of random graphs was established in late 1950s and early 1960s. For detail, see [5].

In this article, our interest is in social networks and how they affect the epidemiology of diseases. A social network is a social structure made up of individuals (or organizations) called nodes which are connected by some specific types of interdependency, such as friendship, enmity, common interest, financial exchange, dislike, sexual relationship or relationship of beliefs, knowledge or prestige. For detail of social network analysis, refer to [16].

3. Methodology

3.1 Model Description

We construct a graph or network model, wherein each individual is represented by a node and the edges are the links between the individuals. A Poisson distribution is used to generate degree sequence; and the graph is constructed using the mechanism of configuration model.
We simulate epidemics on our graph based on the following procedure.

1. Specify the proportion already vaccinated at initial time t_0.
2. Specify the total population $T = N$.
3. Specify the degree distribution as a Poisson distribution with the parameter value λ.
4. Generate the graph by the mechanism of configuration model.
5. At each time step, apply the infection operator ξ_1. A susceptible node may be exposed by neighbouring infected nodes with probability p_1, which is determined by the number of infected nodes $i, i = 1, \dots, k$.
6. At each time step, apply local progression operatorξ_2. An exposed individual progresses to an infectious state p_2.
7. At each time step, an infectious individual recovers with probabilityp_3.
   Repeat these steps until statistical significance is obtained.

4. Data Collection

For the purpose of our simulations, we collected data on children's numbers of friends. We took a sample of 100 children between the ages of 3 and 10 years and the numbers of their friends were recorded. These data were tested for goodness of fit. It was discovered that the data follow a Poisson distribution. See below.

Table 1: Contact Distribution

No. of friends (x)	***Frequency (f)***	fx
3	9	27
4	13	52
5	17	85
6	12	72
7	13	91
8	14	112
9	11	99
10	11	110

$Mean\ contact = \sum fx / \sum f = \frac{648}{100} = 6.48$

We performed goodness-of-fit test using Chi-Square Test as follows.

Hypothesis

H_0: *The data follow a Poisson distribution*

H_1: *The data do not follow a Poisson distribution*

Table 2: *Goodness –of-fit Test*

contacts	***Observed Frequency*** (O_i)	***Expected Frequency*** (e_i)	$(o_i - e_i)^2/e_i$
3	9	7	0.444444444
4	13	11	0.307692308
5	17	15	0.235294118
6	12	15	0.75
7	13	14	0.076923077
8	14	12	0.285714286
9	11	9	0.363636364
10	11	6	2.272727273

$X^2_{cal} = \sum (\boldsymbol{o_i - e_i})^2/\boldsymbol{e_i} = 4.736431869$ and

$X^2_{n-1} = X^2_{9,\ 0.05} = 16.9.$

Since $X^2_{cal} < X^2_{9,\ 0.05}$, we accept H_0 and conclude that, the data follow a Poisson distribution.

5. Simulation

We use the following parameter values in Table 1.

Table 3: *parameters for numerical simulations*

parameters	Definition	Parameter value	source
p_1	Transmission probability per day	0.09	[1, 3]
p_2	probability of progression from exposed to infectious state	0.07	[1, 3]
p_3	Fixed probability for recovery	0.143	[1, 3]
p	Proportion vaccinated before start of outbreak	$0 - 0.95$	variable

6. Results

Our simulation results are as follows.

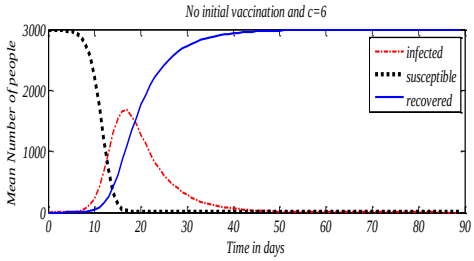

Figure 1: *Graph showing mean sample paths for infected, susceptible and recovered individualss without initial vaccination and when the average number of neighbors is* 6

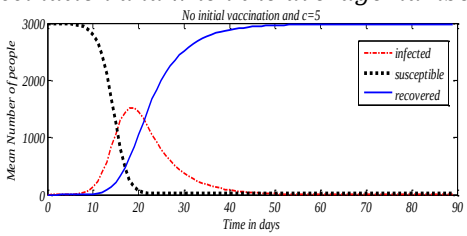

Figure 2: *Graph showing mean sample paths for infected, susceptible and recovered individualss without initial vaccination and when the average number of neighbors is* 5

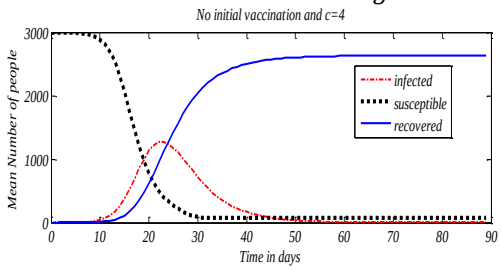

Figure 3: *Graph showing mean sample paths for infected, susceptible and recovered individuals without initial vaccination and when the average number of neighbors is* 4

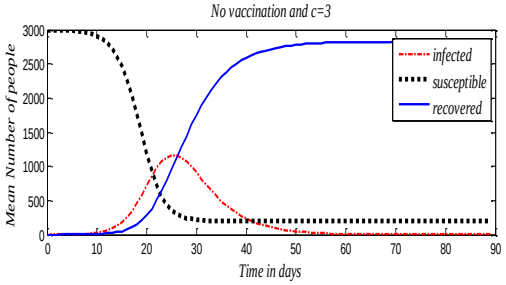

Figure 4: *Graph showing mean sample paths for infected, susceptible and recovered individuals without initial vaccination and when the average number of neighbors is* 3

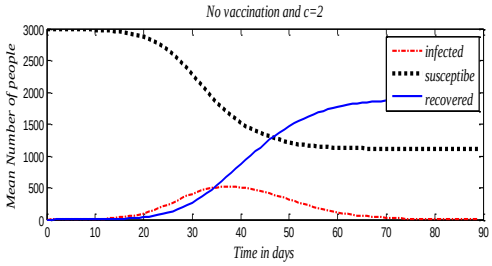

Figure 5: *Graph showing mean sample paths for infected, susceptible and recovered individualss without initial vaccination and when the average number of neighbors is* 2

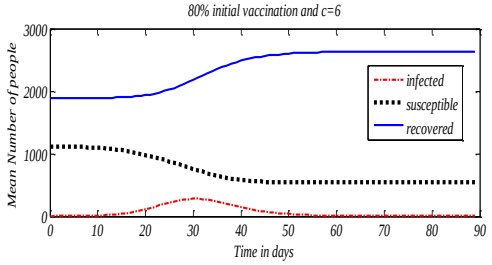

Figure 6: *Graph showing mean sample paths for infected, susceptible and recovered individualss with* 80% *initial vaccination and when the average number of neighbors is* 6

7. Discussion

In this section we discuss the results of our simulations. In the first case, a situation involving a mean of 6 neighbors (based on our data) without initial vaccination before introduction of an index case was investigated. Figure 1 depicts the dynamics of the infected, susceptible and recovered individuals over time. The result shows a very high level of epidemic. Basedon our data, invasion of an index case without initial vaccination is expected to cause a very highepidemic.Episodes two, three, four and five consider situations where there is no initial vaccination before invasion of an index case and the mean numbers of neighbors are respectively 5, 4, 3 and 2.The results of our simulations are shown in Figures 2, 3, 4 and 5 respectively. The results show that epidemic size decreases if the number of neighbors an infected person has contact with decreases. However, in respect of our data on the number of neighbors, the control of epidemic is not feasible if the children are not vaccinated. We, therefore, consider a situation where there is effective and efficient vaccination. The result of our experiment can be seen in Figure 6. The result shows that effective vaccination of children, in particular Idah children, is crucial for elimination of chickenpox disease.

8. Conclusion

In this paper, a network model of chickenpox epidemics is presented and simulated under different scenarios. For the purpose of application, data on contact interaction in Idah Community, Kogi State, Nigeria were collected. Simulation results show that without effective initial vaccination, high level of chickenpox epidemic is expected to occur in Idah. The results further show that it is feasible to bring the epidemic level low if we can reduce the mean number of neighbors to 2 (see Figure 5). Furthermore, based on our data, effective vaccination is required to bring to cause a low epidemic of chickenpox disease. Our simulation results, show that effective vaccination with mean of 6 neighbors can guarantee a low epidemic.

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