Modelling the SARS epidemic by a lattice-based Monte-Carlo simulation

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Abstract—We have analyzed the SARS data and the effect of the control measure in HongKong, based on a spatial Monte-Carlo model (SEIR) with susceptibles, exposed(latent), infective, and recovered. The SARS data can be well fit by numerical simulations. The control measure is effective to decreasing the transmission by reducing the contact rate. The average value of the reproductive number is consistent with many of the previous models.

I. INTRODUCTION

The study of the SARS transmission and the implementation of control measures is still a hot issue [1], [2], [3], [4], [5], [6], [7], [8] due to its high case-fatality rate [9] as well as the possibility of its outbreak in the future. The control measures [1], [2], [3], [6], [7] have been shown to be an effective method to control the outbreak of epidemic. Although the mathematics of epidemiology has been examined by many researches over a long period of time [10], Lipsitch et al. [2] and Riley et al. [3] gave the first quantitative assessment of the epidemic potential of SARS, and the effectiveness of control measures based on stochastic models. Chowell et al. [7] proposed a model of susceptible, exposed, infective, diagnosed, and recovered classes of people to extract average properties and rate constants for those populations in Ontario (Toronto) in Canada, HongKong in China and Singapore outbreaks. Shi presented his Monte-Carlo model based on the data in Vietnam [11]. Donnelly et al. have reviewed the recent progress on the epidemiological and genetic analyses [8].

As indicated by Dye and Gay [1], the next generation of SARS models should include the spatial and stochastic process. The importance of spatial effect became also aware in the study of other epidemics [12], [13], [14]. If the heterogeneity of subpopulation is taken into account, the rate of the cases increase depends on the local population susceptible rather than the global population susceptibles in the full system. The density of local population susceptible usually decline considerably when the epidemic is underway although the decrement of the proportion of the population susceptibles might be very small. Very recently, we have presented a lattice-based Monte-Carlo model (SEIR), in which individuals may be susceptibles, exposed(latent), infective, or recovered [15]. The predictions fit well to the data from Singapore. It is found that super-spreading events (SSE’s) [1] can happen even when the virulences are equal for all the infective individuals. The long latent periods play a critical role in the appearance of SSEs. In this study, the model is used to analyze the data from the outbreaks in HongKong, Guangzhou in China.

In order to characterize the spreading speed of an epidemic and measure the ability of reducing the spreading, the reproductive number, $R$, is introduced [2]. $R$ is defined as the number of secondary cases generated by an infectious case once an epidemic is underway. The mean value of $R$, denoted by $\bar{R}$, can be expressed as

$$\bar{R} = kbD,$$

where $k$ is the number of contacts each infectious individual makes per unit time, $b$ is the probability of transmission per contact between an infectious case and a susceptible person, and $D$ is the mean duration of infectiousness. $k$ depends on the local population density susceptible near the infectious case. During the course of epidemic, $\bar{R}$ declines because of depletion of susceptibles in the population, and because of the implementation of specific control measures. To stop outbreak, $\bar{R}$ must be maintained below one.

II. THE MODEL

The Monte-Carlo model is shown in Fig.1. Individuals may be susceptibles, exposed(latent), infective, or recovered (SEIR). Presently there is no direct evidence of transmission from an asymptomatic person [2]. We assume that infectiousness begins just before the onset of symptoms and remains constant during the symptomatic phase. The exposed individuals are those infected but in latent periods. The recovered individuals are those who will never transmit SARS to others from the infective phase. They include those fully recovered, dead, immune or effectively isolated. Initially susceptible individuals $N_s$ are randomly distributed in a system with size $L_x \times L_y$ and periodic boundary conditions, and no more than one is located in any node. During each time-step an individual can move from its current node to any of the four nearest neighbor nodes with a same probability $p_m$ or remains where it is with a probability $p_r$. $p_r + 4p_m = 1$. Initially $p_r = p_m = 0.2$ is usually assumed. A contact is defined as two individuals falling in a same node. The probability of transmission per contact between...
an infective case and a susceptible is $b$. $\gamma$ distributions are assumed for latent periods [9] and infective periods [16]. The mean latent period of the disease is always $t_{\alpha}=5$ days [17] and the mean infective period is $t_{\beta}$. Changes in the onset-to-hospitalization distribution and effective control to shorten the infectiousness in hospital are treated by the reducing of the infective period as an input to the model. In the simulation, each day corresponds to 90 timesteps, $b=0.2$.

III. SIMULATIONS AND RESULTS

We begin with the data on the 1425 cases by the date of admission to hospital in the first 9 weeks of the epidemic from Feb 20, 2003. The HongKong epidemic has been characterized by two large clusters of cases, at least 125 people were infected on or soon after March 3rd in the Price of Wales Hospital (PWH) and over 300 people becomes infected in the Amoy Gardens (AG) estate, together with ongoing transmission to close contacts [3]. The epidemic due to ongoing transmission to close contacts will be analyzed by our model and two large clusters in PWH and AG will be discussed separately.

The controls in HongKong includes by reducing local contact rates, by shortening the delay between onset of symptoms and hospitalization, by improving the effectiveness of isolation measures in hospitals, and imposing restrictions on longer range movements/contacts [3]. Voluntary drops in the local contact rates are easily attributed to increased awareness of the infection, corresponding to decreasing of $p_r$. The mean time from symptom onset to hospital admission fell to 79% of its original value by 26th March, and to 76% by April 1st [3]. Improving the effectiveness of isolation measures in hospitals reduces the infectious time of symptomatic individuals. In the present model, the infectious time period is effectively including in the $\gamma$ distribution of the period from symptomatic onset to hospital admission. We will consider the case with $t_{\text{onset1}}=6$ days, larger than that reported in HongKong [3]. Reduction of $p_r$ can also effectively decrease the longer range movements/contacts. Consequently, the control measures are We assume that $p_r$ decreases linearly from 0 at $t_{q0}$ to a constant $p_r^0$ in a day.

$$t_{\text{inf}} = \begin{cases} t_{\text{inf}}^0 & \text{for } t < t_{q0} \\ t_{\text{inf}}^0 t_{q0} - t_{\beta} t_{q0} & \text{for } t_{q0} \leq t \leq t_{b} \\ t_{\text{inf}}^0 / 2 & \text{for } t > t_{b} \end{cases}$$

(2)

and

$$p_r = \begin{cases} 0.2 & \text{for } t < t_{0} \\ 1 + S(t - t_{0})/(t_1 - t_0) & \text{for } t_{0} \leq t \leq t_{1} \\ 1 + S & \text{for } t > t_{1} \end{cases}$$

(3)

where $t_{0}$, $t_{1}$, $t_{\alpha}$, $t_{\beta}$ and $S$ are parameters.
The control measure in Eq. 3 reduces the contact rate considerably. Fig. 3 displays the average numbers of contacts for each infectious individual in the above settings and parameters (a), comparing with those with \( p_r \) fixed to 0.2. When the \( p_r \) increases from March 29, the average numbers of contacts for each infectious individual decreases considerably.

The number of contacts depends on the definition of contact, which relates to the probability of transmission per contact between an infectious case and a susceptible person \( b \). In the present model, the number of contacts depends both on \( p_r \) and the susceptibles \( N_s \). For a fixed \( p_r \), the larger the \( N_s \), the larger the number of contacts. In the early period of epidemic, the fraction of infected individuals is very small in the susceptibles so that the decreasing of the susceptibles can be ignored. As the epidemic develops, the susceptibles \( N_s \) deplete, decreasing the contact rate.

Fig. 3. The average numbers of contacts for each infectious individual for \( p_r = 0.2 \) (Black) and \( p_r \) changes according to Eq. (3) (Red). \( N_s = 6000, b = 0.2, t_{onset}^0 = 4.9 \) days.

The early exponential growth, i.e., the cases before March 27, is independent of the control measures. There are infinite combination of \( N_s, b \) and \( t_{onset}^0 \) to obtain the similar simulation results. For any such combination, a measure on \( p_r \) can be found if the change of \( t_{onset} \) is determined as the three examples displayed in Fig. 2.

It should be noted that Riley et al. [3] used the 125 first-generation cases in PWH and other 70 cases, giving a total of 195 infections assumed to occur on 3 March as the seeding infection event in their analysis of the epidemic, while Chowell et al [7] started with an infectious individual in their model for the SARS outbreaks in Ontario, HongKong, Singapore. It is clear that different simulation results can be obtained with different seeding infection events. However, The data in HongKong can be well fit by our model with adjusting the parameters. Moreover, if the parameters are known, we can predict the transmission of the SARS. Since the present model is spatial relevant, the position of each seed should be known in the simulation.

**REFERENCES**

The period from onset to hospital admission can be approximated by a $\gamma$ distribution [9] while there is no report on the distribution of the infectious period after hospital admission.