

## RESEARCH

# A toy article based on "A double epidemic model for the SARS propagation and CHIME

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### Abstract

**Attention:** Some of the information in this document is not factually correct and was only included to test the functionality of the alignment component of the Automates project developed at the UArizona.

**Background:** An epidemic of a Severe Acute Respiratory Syndrome (SARS) caused by a new coronavirus has spread from the Guangdong province to the rest of China and to the world, with a puzzling contagion behavior. It is important both for predicting the future of the present outbreak and for implementing effective prophylactic measures, to identify the causes of this behavior.

**Results:** In this report, we show first that the standard Susceptible-Infected-Removed (SIR) model cannot account for the patterns observed in various regions where the disease spread. We develop a model involving two superimposed epidemics to study the recent spread of the SARS in Hong Kong and in the region. We explore the situation where these epidemics may be caused either by a virus and one or several mutants that changed its tropism, or by two unrelated viruses. This has important consequences for the future: the innocuous epidemic might still be there and generate, from time to time, variants that would have properties similar to those of SARS.

**Conclusion:** We find that, in order to reconcile the existing data and the spread of the disease, it is convenient to suggest that a first milder outbreak protected against the SARS. Regions that had not seen the first epidemic, or that were affected simultaneously with the SARS suffered much more, with a very high percentage of persons affected. We also find regions where the data appear to be inconsistent, suggesting that they are incomplete or do not reflect an appropriate identification of SARS patients. Finally, we could, within the framework of the model, fix limits to the future development of the epidemic, allowing us to identify landmarks that may be useful to set up a monitoring system to follow the evolution of the epidemic. The model also suggests that there might exist a SARS precursor in a large reservoir, prompting for implementation of precautionary measures when the weather cools down.

**Keywords:** SARS; SIR; infection

### Content

Review on the standard SIR model Consider a disease that, after recovery, confers immunity (which includes deaths: dead individuals are still counted). We assume that there is no entry into or departure from the population. The population can then be divided into three distinct classes; the susceptibles,  $S$ , who can catch the disease; the infectives,  $I$ , who have the disease and can transmit it; and the removed

class, R, namely those who have either had the disease, or are recovered, immune or isolated until recovered. Here we follow the definition of the class R given in [8]. However, we would like to draw the readers' attention that this definition of the class R is different from those given in [7], [11] which do not include isolated infectives in the class R. The progress of individuals is schematically described by  $S \rightarrow I \rightarrow R$ . Let  $S(t)$ ,  $I(t)$  and  $R(t)$  be the number of individuals in each of the corresponding class at time  $t$ . Note that usually only  $R(t)$  can be known. It is often considered that  $R(t)$  is the cumulative number of patients admitted to hospitals. With some reasonable assumptions (which will be explained in details in the next section), we can show that these three functions are governed by the following system of nonlinear ordinary differential equations (see next section for the derivation of these differential equations):

where  $r$  is the infection rate and  $a$  the removal rate of infectives. The parameters  $r$  and  $a$  characterize the propagation of the disease and can also be used as control parameters in order to stop the epidemic. In general, the functions  $S$ ,  $I$  and  $R$  behave as the three curves in Figure 1. The characteristics of these curves are as follow.

- The function  $I$  first increases exponentially and reaches the maximum value at time  $t_0$  and then decreases to zero. The time  $t_0$  is the critical point of the function  $I$  at which  $\frac{dI}{dt} = 0$

$$\begin{aligned}\frac{dS}{dt} &= -rS(t)I(t) \\ \frac{dI}{dt} &= rS(t)I(t) - aI(t) \\ \frac{dR}{dt} &= aI(t) \\ \frac{dS}{dt} &= -rS(t)I(t) - r_P S(t)I_P(t)\end{aligned}$$

## 1 Reproductive number

An important descriptive parameter is the basic reproduction number, or  $R_0$ . This represents the average number of people who will be infected by any given infected person. When  $R_0$  is greater than 1, it means that a disease will grow. A higher  $R_0$  implies more rapid transmission and a more rapid growth of the epidemic. It is defined as  $R_0 = \beta/\gamma$

$R_0$  is larger when the pathogen is more infectious people are infectious for longer periods of time the number susceptible people is higher.

A doubling time of 6 days and a recovery time of 14.0 days imply an  $R_0$  of 2.71 mm.

$$\begin{aligned}I_P(0) &= 500000 \\ S(0) &= 6300000 \\ I_P(0) &= 500000 \\ E(0) &= 100 \\ I(0) &= 50 \\ r_P &= 7.079 \times 10^{-8}\end{aligned}$$

## 2 Miscellaneous text blocks 1

... which is the transmissibility  $\tau$  multiplied by the average number of people exposed  $c$ . The transmissibility is the basic virulence of the pathogen. The number of people exposed  $c$  is the parameter that can be changed through social distancing. The number of people exposed is hard-coded to 300 people.

### 3 Discrete-time SIR modeling of infections/recovery

The model consists of individuals who are either Susceptible (S), Infected (I), or Recovered (R). The epidemic proceeds via a growth and decline process. This is the core model of infectious disease spread and has been in use in epidemiology for many years.

### 4 Miscellaneous text blocks 2

Transmissibility is measured in millimeters. For example, if we take the total initial population,  $S(0) = 6.8$  millions (e.g. the case of Hong Kong) and an initial infected class of  $I(0) = 10$  persons, together with  $a = 2/3$  and  $r = 9.788 \times 10^{-8}$ . Transmissibility is set to 32 mm. But other times,  $\tau = 450$  kg.

For many deterministic epidemic models, an infection can get started in a fully susceptible population if and only if  $R_0 \geq 1$ . The population can then be divided into three distinct classes; the susceptibles, S, who can catch the disease; the infectives, I, who have the disease and can transmit it; and the removed class, R, namely those who have either had the disease, or are recovered, immune or isolated until recovered. Here we follow the definition of the class R given in [8]. However, we would like to draw the readers' attention that this definition of the class R is different from those given in [7], [11] which do not include isolated infectives in the class R. The progress of individuals is schematically described by  $S \rightarrow I \rightarrow R$ . Let  $S(t)$ ,  $I(t)$  and  $R(t)$  be the number of individuals in each of the corresponding class at time  $t$ . Note that usually only  $R(t)$  can be known. It is often considered that  $R(t)$  is the cumulative number of patients admitted to hospitals.

$$1/a + 1/b = 11.83$$

The gain in the exposed class E is at a rate proportional to the number of people in the infective class I and that of the susceptibles S, that is  $rS(t)I(t)$ , where  $r > 0$  is a constant. (iii) The gain in the infective class I P is at a rate proportional to the number of people in the infective class I P and that of the susceptibles S, that is  $rPS(t)IP(t)$ , where  $rP > 0$  is a constant.

The rate of removal of the people in class E to the infective class I is proportional to the number of people in class E, that is  $bE(t)$ , where  $b$  is a positive number. The exposed class is measured in people.  $E = 30$ . The number of people in exposed class is equal to 35.

$$I_{t+1} = I_t + \beta S_t I_t - \gamma I_t$$

A makes contact sufficient to transmit infection with  $rN$  others per unit time, where  $N$  is the total population. Note that the probability that a random contact by an infective with a susceptible, who can then transmit infection, is  $S/N$ , therefore the number of new infections in unit time is  $(rN)(S/N)I = rSI$ . One can also interpret  $rP$  in a similar way. Here  $r$  and  $rP$  are related to the infection rate of disease A and B respectively, while  $a$ ,  $aP$  and  $b$  are the removal rate of individuals in class I,  $IP$  and  $E$  respectively. The last two equations follow from the assumption that the population number equals 200. this amounts to only consider a fraction of the initial population in the Susceptibles class S (and to set  $IP(0) = 0$  because the spread has already taken place); equivalently this can be implemented by putting at the initial time some of the population in the Removed class R (i.e.,  $R(0) \geq 0$ ).

## 5 Misc paragraph 4

The total population was taken to be  $S(0) = 23.67$  millions; the other parameters (including the initial conditions) were optimised to ensure a good agreement with the data and were obtained to be  $E(0) = 2$ ;  $IP(0) = 137638$ ;  $I(0) = 32$ ;  $R(0) = 0$ ;  $RP(0) = 0$ ;  $r = 1.62 \times 10^{-8}$ ;  $rP = 3.87 \times 10^{-8}$ ;  $a = 0.120$ ,  $aP = 0.272$ ;  $b = 7.644$ .

$$\beta = \tau \times c$$

The parameters  $a$  and  $aP$  describe the removal from the classes  $I$  and  $IP$  to the classes  $R$  and  $RP$  respectively. Since the removed classes  $R$  are considered to contain the individuals with infections the parameters  $a$  and  $aP$  characterize the identification rate of potential cases; it is then rather related to the health policy than to the disease itself (e.g.  $a$  is not the mortality rate!). Since we don't know these things, we can extract it from known doubling times  $T_d$ . The AHA says to expect a doubling time  $T_d$  of 7-10 days.