

Stochastic Model in Epidemic

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ABSTRACT

This paper gives in detail about stochastic epidemic model. A simple stochastic epidemic model is defined and exact and asymptotic model properties are presented. The purpose of modelling is given by detailing the effects of vaccination and also in terms of inference procedures for important parameters, such as the basic reproduction number and the critical vaccination coverage. Also this paper gives about the initial phase of the epidemic approximated by homogeneous branching process.

Keywords: Basic reproduction number, Stochastic epidemic, Ultimate number, Branching Process.

INTRODUCTION

Bernoulli (1760) aimed at evaluating the effectiveness a certain method for variolation against smallpox. That is early modelling of infectious disease is confined to a specific disease. Similarly Ross (1911) modelled the transmission of malaria. Early models were often deterministic. Kermack and Mckendrick formed a general and more rigorous study on the spread of the epidemic. In the deterministic model of epidemic, we study about how the outbreak of infectious disease affect the whole population, how many will get infected if the epidemic takes off. Also we study about the vaccination prior to the arrival of the disease. As problems were resolved, the models were generalized in several ways towards making them more realistic. Some of such extensions were for different types of individual, allowing for non-uniform mixing between individuals, for example due to social or spatial aspects and to allow seasonal variations. Another generalization of the initial simple deterministic epidemic model was to study stochastic epidemic models.

DETERMINISTIC MODEL

One simple model the deterministic general epidemic model can be defined by two differential equations. It is assumed that at any time point an individual is either susceptible (s), infected and infectious (i) or recovered and immune (r). Such individuals are from now called susceptible, infective and recovered respectively.

The model makes the following assumptions only susceptible individuals can get infected and after having been infective for some time, an individual recovers and become completely immune for the remainder of the period. Finally we assume there are no births, deaths, immigration or emigration during the study period.

Let $s(t)$, $i(t)$, $r(t)$ respectively denote the community fractions of susceptible, infective and recovered. Since these are fractions and the community is closed we assume that $s(t) + i(t) + r(t) = 1$ for $t \geq 1$. From the assumptions mentioned above, together with the assumption of the community being homogeneous and people mixing homogenously the deterministic general epidemic model is defined by the following set of differential equations.

$$\left. \begin{aligned} s'(t) &= -\alpha s(t) i(t) \\ i'(t) &= \alpha s(t) i(t) - \beta i(t) \\ r'(t) &= \beta i(t) \end{aligned} \right\} \quad (1)$$

These differential equations, together with the starting coefficient $s(0) = 1 - \varepsilon$, $i(0) = \varepsilon$ and $r(0) = 0$ defines the model.

The initial fraction infective $\varepsilon > 0$ is often assumed to be small as indicated by the notation ε , it must however is positive, otherwise all differential equations are constant and equal to 0. The reason for assuming that $r(0) = 0$ is that initially immune individuals play no part in the dynamics, so up to a normalizing constant, initially immune individuals may simply be ignored.

The term $\alpha s(t) i(t)$ in equation (1) comes from the fact that susceptible must have contact with infective in order to get infected. So the assumption about uniform mixing implies that infective occur at a rate proportional to $s(t) i(t)$. By studying the differential equations it is straightforward to show that $s(t)$ is monotonically decreasing down to $s(\infty)$ say, and $r(t)$ is monotonically increasing up to $r(\infty)$. The differential equation for $i(t)$ can be written as $i'(t) = i(t) (\alpha s(t) - \beta)$. So if $\alpha s(0) > \beta$ then $i(t)$ initially increases but eventually when $s(t)$ has decreased enough $i(t)$ starts decreasing. If on the other hand

as $0 < \beta$ then $i(t)$ decreases already from the start with the effect that little will happen as t tends to infinity.

The ratio $R_0 = \frac{\alpha}{\beta}$ is of fundamental importance and can be interpreted as the average number of new infections caused by an infectious individual before recovering. This ratio is often referred to as the basic reproduction number and denoted by R_0 .

$$R_0 = \frac{\alpha}{\beta} \tag{2}$$

When $R_0 > 1$ the epidemic takes off and when $R_0 < 1$ there is no epidemic. The differential equation (1) can also be used to obtain a balance equation for the final state $s(\infty), r(\infty)$.

By dividing the first by the last we get $\frac{ds}{dr} = -R_0 S$, which implies that $s(t) = s(0) e^{-R_0 r(t)}$.

The fact that $i(\infty) = 0$ implies that $s(\infty) = 1 - r(\infty)$ at the end of the epidemic there are no infective, only susceptible and recovered.

We have $R_0 < 1$ then there will be small outbreak or no epidemic and if $R_0 > 1$ there will be a major outbreak of epidemic. The above results is true when the community or population is homogeneous and that the individuals mix uniformly with each other.

Even if the assumption of a homogeneous uniformly mixing community or population are accepted this deterministic model may not be suitable in some cases. So we need stochastic epidemic model.

STOCHASTIC MODEL

We now define the standard stochastic SIR epidemic model.

Let $S(t), I(t)$ and $R(t)$ respectively denote the number of susceptible, infective and recovered at time t and suppose that at time $t=0$ there numbers are given by $S(0) = n - m, I(0) = m$ and $R(0) = 0$. The dynamics of the model are defined as follows, Infectious individuals have close contact with other individuals randomly in time at constant rate α and each such contact is with a randomly selected individual, all contacts of different infective being defined to be mutually independent.

Infectious individuals remain infectious for a random time T after which they stop being infectious, recover and become immune to the disease. The infectious periods are defined to be independent and identically distributed having distributions F_1 and mean $E T = \frac{1}{\beta}$.

The epidemic starts at time $t=0$. As the epidemic evolves, according to the rules above new individuals get infected and eventually recover, up until the first time T when

there are no infective in the population. Then no further individuals can get infected implying that the epidemic stops. The final state of the epidemic is described by the ultimate number $R T$. The final number of infected $R T$ will consist of those m who were initially infected plus those z , who were infected during the outbreak.

EXACT DISTRIBUTION

We derive a recursive formula for the final size of the epidemic. This formula is based on the fact that in order not get infected an individual must escape infection from all those who did get infected during the outbreak.

The derivation of the recursive formula for the final size uses two main ideas; a Wald’s identity for the final size and the total infection pressure and the interchangeability of individuals making it possible to express the probability of getting i additional infections among the initially $m - n$ in terms of getting i infected in a smaller subset.

We start with the latter result. To this end fix n and write $\alpha' = \alpha / n$. Let Z denote the final number infected excluding the initial infective, so the possible values of Z are $0, \dots, n - m$. Since individuals are interchangeable we can label the individuals according to the order in which they get infected. The initial infective are labelled $- m - n, \dots, 0$ then according to time of infection: $1, \dots, Z$ and those who avoid infection according to any order $Z + 1, \dots, n - m$ with labelling we define the total infection pressure A .

$$A = \alpha' \sum_{i=-m-1}^Z I_i \tag{3}$$

Now let $p_i^{n-m} = P Z^{n-m} = i$ denote the probability that exactly i susceptible get infected during the outbreak, explicitly showing the number of initial susceptible but suppressing the dependence on the initial number of infective m . Then using the interchangeability of individuals and reasoning in terms of subsets among the initially susceptible, it can be shown that for any $i \leq k \leq n - m$ it holds that

$$\frac{P_i^{n-m}}{\binom{n-m}{i}} = \frac{P_i^k}{\binom{k}{i}} E e^{-n-m-k A^k} \Big| Z^k = i \tag{4}$$

The probability that i get infected among the initially $m - n$ susceptible equals the product of the probability of having i infected in a smaller subset of size $k \leq m - n$ multiplied by the probability that no one in the remaining set gets, infected conditioning on the first event. The notations A^k and Z^k are hence the total pressure and final size starting with k susceptible and m infective.

We show a Wald's identity for Z^k and A^k . Let $\phi_\theta = E e^{-\theta I}$ denote the Laplace transform of the infection period I . We then have the Wald's identity

$$E \left(\frac{e^{-\theta A^k}}{\phi_{\theta \alpha'}^{m+Z^k}} \right) = 1, \quad \theta \geq 0 \tag{5}$$

The following steps prove the result

$$\begin{aligned} \phi_{\theta \alpha'}^{m+k} &= E \left[\exp \left(-\theta \alpha' \sum_{i=m-1}^k I_i \right) \right] \\ &= E \left[\exp \left(-\theta \left(A^k + \alpha' \sum_{i=Z^k+1}^k I_i \right) \right) \right] \\ &= E \left[e^{-\theta A^k} \phi_{\theta \alpha'}^{k-Z^k} \right] \end{aligned}$$

Where the last identity follows because the $k - Z^k$ infections periods I_{Z^k+1}, \dots, I_k are mutually independent and also independent of A^k . Dividing both sides by $\phi_{\theta \alpha'}^{m+k}$ gives the desired result. If we use Wald's identity with $\theta = n - m - k$ and condition on the value of Z^k we get

$$\sum_{i=0}^k \left[\frac{E e^{-(n-m-k) A^k} \mid Z^k = i}{\phi_{n-m-k \alpha'}^{m+i}} \right] p_i^k = 1 \tag{6}$$

Using equation (4) in the equation above we get

$$\sum_{i=0}^k \frac{\binom{k}{i} p_i^{n-m}}{\binom{n-m}{i} \phi_{n-m-k \alpha'}^{m+i}} = 1 \tag{7}$$

Simplifying the equation, returning to $\alpha = \alpha' n$ and putting p_k^{m-n} on one side, we obtaining the recursive formula for the final size distribution $p_k^{n-m}, k = 0, \dots, n - m$.

FINAL SIZE DISTRIBUTION

$$P_k^{n-m} = \binom{n-m}{k} \left[\phi \quad n-m-k \quad \alpha/n \right]^{m+k} - \sum_{i=0}^{k-1} \binom{n-m-i}{k-i} \left[\phi \quad n-m-k \quad \alpha/n \right]^{k-i} p_i^{n-m} \quad (8)$$

The above is a recursive formula. Solving (8) for $k = 0$ and then for $k = 1$ given

$$p_0^{n-m} = \phi \alpha^m, \\ p_1^{n-m} = n \phi \quad n-1 \quad \alpha/n \left(\left[\phi \quad n-1 \quad \alpha/n \right]^m - \left[\phi \quad \alpha \right]^m \right) \quad (9)$$

In order to compute p_k^{n-m} using (8) it is hence necessary to sequentially compute p_0^{n-m} up to p_{k-1}^{n-m} . As a consequence the formula is not very enlightening and it may be numerically very unstable when k is large that is when $n-m \geq k$. Even when it is possible to compute p_k^{n-m} using (8) with $n-m$ being large, an approximate formula can be more informative.

EARLY APPROXIMATION

When n is large, the initial phase of the epidemic can be approximated by a homogeneous branching process with birth rate α and life distribution $I \sim F_1$ having Laplace transform ϕs . If $R_0 = \alpha E I = \alpha/\beta \leq 1$, the final size of the epidemic is bounded in probability whereas if $R_0 > 1$, it is not. The approximation further tells us that when $R_0 > 1$ the epidemic will be minor (bounded) with a probability q^m and major (unbounded) with probability $p = 1 - q^m$, where q is the smallest solution to

$$q = E q^X = E E q^X | I = E e^{-\alpha I (1-q)} = \phi \alpha (1-q) \quad (10)$$

The distribution outbreak size in the minor case can also be determines from branching process theory.

CONCLUSION

There are many applications, to modelling of infectious disease spread, some disease that have received much modelling attention over the last decades are for example HIV, smallpox, foot and mouth disease, SARS and recently H1N1 influenza.

Even when trying to include as many realistic features in a model as possible there is a limit to how close a model can get to reality, and models can never completely predict what will happen in a given situation.

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