A Study of Effects of Disease Caused Death in A Simple Epidemic Model

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Abstract- This paper is concerned with various effects of disease caused death on the host population in an epidemic model of SIR type. Various effects of disease caused death on the host population are studied in this epidemic model. The basic problem discussed in this paper is to be describing the spread of an infection caused death within a population. It is further assumed that there is no substantial development of immunity and that removed infectious are in effect cured of disease. The rate of natural birth and death is assumed to be balanced.

Key Word - Mathematical modeling, Population size , Birth- rate , Death- rate & Infection rate.

INTORDUCTION

Anderson and May [7] studied the effects of disease caused death on the population size in model for a disease which spreads through direct infection within a population whose size is allowed to very in time. Two important new phenomena a was revealed by their study.

A threshold for the population size exists that determines whether the population can sustain an epidemic; fatal diseases are found to have a regulating effect on the growth of the population. Many subsequent works have fallowed this line of research [5]. Another characteristic of this body of research is that the emphasis is on the interplay between the net intrinsic growth rate r and the rate of disease caused death α : it $r > \alpha$ then the disease it likely to become endemic. To explain this phenomenon, potential mechanisms to endemicity other then a large intrinsic growth rate r need to be studied. In recent study, we discovered that a long incubation period incorporated into a SIR model may provide an explanation for concurrence of high pathogenicity and long life span of infectiousness. We took different approach to study of epidemic models by assuming that the population a small intrinsic growth rate r so that disease caused mortality rate
α is relatively large. This approach has following advantage.

1. If greatly reduces the technicality in mathematical analysis, One may start with the case r = 0 and then consider the case of small positive r.
2. If enables us to isolate those effects on the population that directly related to the disease e.g. we discovered our essential difference between a model that incorporates an incubation period and one that does not. Even in a simple model that does not contain an incubation period, this new approach leads to the discovery of several interesting details not found in literature.
3. By keeping the mathematical technicalities at its minimum, this approach may allow our models more accessible to field epidemiologist and hence encourage of application of mathematics in epidemiological studies.

In the present work we demonstrate our approach through a very simple model. We assume that the disease spreads through direct contact among the hosts, the disease has no incubation period as considered in most of previous works [2,4] and that the intrinsic growth rate of host population is zero i.e r = 0 so that in absence of disease, the population size remains constant. The mathematical analysis of model is very elementary, and it provides epidemiologically interesting details about an epidemic. Also, we demonstrate that the kind of phenomenon one many observe in the case of a small positive intrinsic growth is essentially the some as we obtained here. In particular, this seems to suggest that, an SIR model is essentially a model for an epidemic; it does not provide an epidemiologically relevant mechanism for disease endimicity.
For other studies on epidemological models with varying population size closely related to the one we consider her, see Greenhalgh [1] and Mena- Lorca and Hethcote [6] and references there on. Other models with varying population and disease- caused death have been studies by Brauer [2], Bremerman and Thieme [3], Gao and Hethcote [4], and Hethcote [6], and Pugliese [7].

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FORMULATION OF MATHEMATICAL MODEL :

The population is partitioned into classes of susceptible, infectious and immune individuals, with population N(t) is

$$n(t) = x(t) + y(t) + z(t) \qquad (1)$$

Let us consider the per capital birth rate is a constant 'b' and all newborns are susceptible. The per capital natural death rate assumed to be 'b' so that total population remains constant in absence of disease. Suppose the disease spreads through direct contact between susceptible and infections individuals. We assume that the transmission coefficient per unit time by β x(t)y(t). This is equivalent to assuming that the contact rate between individual is β n(t), proportional to n (t).

The disease is assumed to causes death to infected individuals, with a death rate constant α . Let the average infectious period for an infectious individual be $\frac{1}{\gamma}$ so that transfer from infectious class to immune class is at a constant rate γ . It is also assumed that the disease confers permanent immunity so that no transfer from infectious class to immune class exists. Since vaccination is one of the major means of control and prevention of many viral infections, the effect of a vaccination strategy is also considered. All susceptible individuals are vaccinated at a constant per capita rate 'p'. Based on these modeling hypotheses, the following set of differential equations is derived.

$$x' = bn - \beta xy - bx - px$$
$$y' = \beta xy - (b + r + \alpha)\, y$$
$$z' = ry - bz + px \qquad (2)$$

and

$$n(t) = x(t) + y(t) + z(t)$$

The model (2) was one of many model proposed and studied in [1], in which only a local analysis is carried out. It is also related to a model studied in [5] which does not consider vaccination.

Adding the equations in (2) gives

$$x' + y' + z' = bn - \beta xy - bx - px + \beta xy - (b + r + \alpha)\, y + ry - bz + px$$

$$n' = bn - \beta xy - bx - px + \beta xy - by - ry - \alpha y + ry - bz + px$$

$$n' = -\alpha y \qquad (3)$$

which implies that total population always decreases.

Observing that the variable z does not appear in first two equations in (1) we may simply study the following system

$$x' = bn - \beta xy - bx - px$$
$$y' = \beta xy - (b + r + \alpha)\, y \qquad (4)$$
$$n' = -\alpha y$$

and determine the variable z from

$$z(t) = n(t) - x(t) - y(t)$$

The feasible region for (4) is

$$G = \{(x, y, n) \in R \quad x + y \le n\}$$

It can be checked that G is positively invariant under the dynamics of (4) which, together with the fact that the right hand side of (4) is analytic in (x, y, n), shows that the model is well posed.

EQUILIBRIUM POINT:

Setting the right hand side of (4) equal to zero, we obtain the set of equilibrium Eo

$$Eo = \{(x, y, n) \in G \quad y = 0, x = b/b+p\, n\} \qquad (5)$$

LOCAL STABILITIES:

To study the local stability of each equilibrium,

$$P^* = (bn^*/b+p,\ 0,\ n^*) \in Eo,$$

we linearize the vector field of (4) at P*. The corresponding Jacobian matrix is

$$J(P^*) = \begin{bmatrix} -b-p & -\beta bn^*/b+p & b \\ 0 & \beta bn^*/b+p - b - r - \alpha & 0 \\ 0 & -\alpha & 0 \end{bmatrix}$$

whose characteristic equation is $[J - \lambda I] = 0$

$$\begin{vmatrix} b - p - \lambda & \beta bn^*/\, b+p & b \\ 0 & \beta bn^*/\, b+p - b - r - \alpha - \lambda & 0 \\ 0 & -\alpha & 0 - \lambda \end{vmatrix} = 0$$

Or $(-b-p-\lambda)\,(\beta bn^*/b+p - b - r - \alpha - \lambda)\,(-\lambda) = 0$

whose eigen values are

$$\lambda_1 = -b - p$$

$\lambda_2 = \beta bn^*/b+p - b - r - \alpha$
$\lambda_3 = 0$ (6)

with corresponding eigen vetors
$v_1 = (1, 0, 0)$
$v_2 = (a_1, a_2, a_3)$
$v_3 = (b, 0, b+p)$ (7)

where
$a_1 = (b - \beta bn^* a_2 / b + p) / (\beta bn^*/b+p - p - r - \alpha)$
$a_2 = \beta bn^*/\alpha (b+p) - (b + r + \alpha) / \alpha$ (8)
set
$n = (b+p)(b + r + \alpha) / \beta b$ (9)

The local stability of $P^* = (x^*, y^*, n^*)$ is determined by the sings of λ_i's. These are two cases arises:

CASE I :

1. $P^* \in E_0$ and $n^* \neq n$, then P^* always has a 1-dimensional manifold
given by E_0.

2. If $n^* > n$, then P^* has a 1- dimensional stable and 1-dimensional
unstable manifold.

3. If $n^* < n$, then P^* has a 2- dimensional stable manifold.

CASE II :

If $P^* \in E_0$ and $n^* = n$ then we observe that subspace y=0 is invariant with respect to (6.2.4) and so is the line defined by y=0 and n= n* for each n*>0. In each of these lines P* is the global attractor in the line y=0, n = n*.

RESULT:

The parameter
$n = (b+p)(b + r + \alpha) / \beta b \equiv (b + p) x / b$
is the threshold for the total population to sustain an epidemic. Where the total population n(t) is much below this value so that x < x , any outbreak of infection, no matter its initial momentum y(0), will sustain itself and immediately decreases and continues to decrease monotonically with time until it dies out. On other hand, if the population above this threshold value, disease will sustain itself for certain time, reach its maximum extent while killing many of the infection and reducing the total population below the threshold n, and start to decline and eventually die out.
If we set, each solution x(t) , y(t), n(t) to (4)

$y(max) = \max y(t)$

Then y (max) is achieved where y'(t) = 0, so
$x(t) = (b + r + \alpha) / \beta = x$

where no vaccination is applied, namely P = 0 then x = n. This is the loss from the infected class divided by the rate of transmission of disease caused death. α increases. Once again this implies that a faster killing disease has less change to cause a large scale epidemic.
If y (max) increases as transmission coefficient β increases. This implies that diseases with greater transmission rate cause larger scale epidemics.
If p increases. y (max) decreases and it takes less time for y(t) to achieve its minimum, the faster the susceptible are removed to the immune class, the less virulent the disease becomes.
. Also x can used as an indicator as to whether the epidemic has reached its maximum extent. After the number of susceptible has decreased below x , the disease will start to die down and eventually die out. If y (max) decreases when the rate

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