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Mathematical Analysis and Simulations of a Model with Latent Population

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ABSTRACT

In this paper, we analyzed a generic model with susceptible, latent, infected, and recovered population. It is a system of four non linear differential equations which are solved analytically and computationally. The techniques used to study the model are stability analysis, next generation matrix, and numerical simulations in Matlab. We calculated R_0 and observed the effect of various parameters on the population dynamics.

1. Introduction

Infectious disease epidemiology can be understood by mathematical modeling in a better way. The transmission of disease, the mathematical representation of disease parameters affecting disease and analysis of emerging dynamics are basic parts of mathematical modeling [1], [2], [3] and [4]. Communicable disease have always been a principal part of human history. There have been diseases that have invaded human population causing deaths. Because of poor health care facilities in the developing countries, every year millions of people die of diseases like measles, diarrhoea and many others [10]. Many diseases like cholera, sleeping sickness and malaria are endemic in many parts of the world. The aim of epidemiologists is to firstly understand the disease, its cause, to find the ways to control it by different methods.

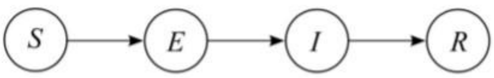

Figure 1: Flow diagram of the system

The first mathematical model in epidemiology was given by Daniel Bernoulli [11] for smallpox. In the formation of mathematical model, a few things are assumed about the transmission of infection. The diseases are spread through virus or bacteria. W.H. Hamer [13] proposed that spread of any disease depend on the number of susceptible and number of infectious. The models are formulated in the form of ordinary differential equations as initial value problem. Factors affecting disease transmission like infectious agent, latent period, incubation period, vector to host interaction, area where the disease prospers, mode of transmission, human reaction to media[9], and immunity are incorporated in the model as parameters [8]. If N(t) be the total population of species at times t, then

$$dN/dt = birth - death + migration$$

In the sample model, if migration term is not considered, then $dN/dt = aN - bN$

Where a, b are constants and $N(0) = N_0$ is the initial condition. If $a > b$, the population grows and if $a < b$, the population dies out. In compartmental modeling like in SIR model in many diseases, infection return to the susceptible class after recovery if the disease does not confer any immunity against disease. The disease which does not confer immunity are represented by terminology SIS. The possible model can also be SIRS, SEIR, SEIS, Where E is exposed period between being infected and becoming infective[5]. For diseases like tuberculosis, the progression of individuals into infected state is different for various individual. Also drug resistance is also considered in many cases with disturbed drug schedule. For influenza, a fraction of population gets infected but are asymptotic. In case of rotavirus, the pathogen attacks gastrointestinal tract in young children below age of five years. In case of chikungunya, the transmission is through a mosquito like *aedes albopictus* or *aedes ageypti* which also transmits dengue, again a vector borne disease [12].

2. Mathematical Model

The total population is divided into four compartments. S denotes susceptible population, E denotes the population exposed to infection, I denotes infectious class and R denotes recovered class. The transfer between the compartments is through various parameters. The susceptible human population is increased by birth at a constant rate *b*. Many researchers also add factor of immigration to increase the susceptible population. It is being assumed that all the humans in the S compartment are naive. From all the compartments, the population is decreased by deaths at a constant rate *μ*. The disease is transmitted from infected to susceptible at a constant rate *β*. Individuals move from exposed class to infected class with progression rate *γ*. From infected compartment individuals die due to disease at a rate *δ*. The susceptible are being vaccinated with efficacy to join the recovered compartment. It is assumed that susceptible and infected mixes deliberately. Under these assumptions the model is represented in the form of differential equations as:

$$\begin{aligned}
\dot{S} &= bN - \beta\frac{SI}{N} - \mu S - \epsilon S,\\
\dot{E} &= \beta\frac{SI}{N} - \gamma E + \mu E,\\
\dot{I} &= \gamma E - \delta I - \mu I,\\
\dot{R} &= \epsilon S - \mu R.
\end{aligned}$$

2.1 Positivity of the Model

Before analysis of the model, we will explore the basic features. As the model deals with the dynamics of humans, all parameters in the above model are assumed to be non- negative. Let

$$T = \{(S, E, I, R) \in R_+^4 : S \geq 0, E \geq 0, I \geq 0, R \geq 0\}$$

Assuming that the initial conditions for the given model to lie in region *T* there exists a unique solution which lies in *T* for all *t* ≥ 0.

Table 1: Table for parameters.

Parameters	Description
β	Transmission probability of disease from mosquito to human
γ	Rate at which individuals move from susceptible class to exposed class
μ	Natural death rate
ϵ	Effective vaccination rate of susceptible humans
b	Birth rate of humans
δ	disease induced death rate
N	Total human population

3. Analysis of Model

3.1 Dimensionless Transformation

We will transform the equations in the model into normalized quantities by scaling the populations in each compartments by total populations *N*. We make the transformations as

$$s = \frac{S}{N}, e = \frac{E}{N}, i = \frac{I}{N}, r = \frac{R}{N}$$

The system of equations in the above model become

$$\dot{s} = b - \beta is - \mu s - \epsilon s$$
$$\dot{e} = \beta is - \gamma e + \mu e$$
$$\dot{i} = \gamma e - \delta i - \mu i$$
$$\dot{r} = \epsilon s - \mu r$$

The total population N can be determined from

$N = S + E + I + R.$

Also upon adding all the equations in the model for human populations, we get

$N^{\cdot} = bN - \delta I - \mu N.$

Here N is the total population. S is the susceptible class. E is the exposed class. I is infectious class. R is the recovered class. Similarly, there can be many models in which different parameters like temperature, immunity, weather etc can be considered to find their dependence on each other and also how they can affect the disease transmission.

3.2 Disease free equilibrium

The disease- free equilibrium of the model, that is, when disease is not present in the society is given by

$e^* = 0, i^* = 0, r^* = 0.$

Substituting in the first equation of model after making it dimensionless, we get,

$$b - 0 - \epsilon s - \mu s = 0$$
$$b = (\epsilon + \mu)s$$
$$s = \frac{b}{\epsilon + \mu}$$

Disease free equilibrium is

$$E^*\left(\frac{b}{\epsilon+\mu}, 0, 0, 0\right)$$

3.3 Stability Analysis at Disease free equilibrium

The local stability at this point is established from jacobian of the system given by

$$J_0 = \begin{pmatrix} -\beta i - \epsilon - \mu) & 0 & -\beta s & 0 \\ \beta i & -(\gamma + \mu & \beta s & 0 \\ 0 & \gamma & -(\delta + \mu) & 0 \\ \epsilon & 0 & 0 & -\mu \end{pmatrix}$$

To find the eigen values and substituting the disease-free equilibrium, we get,

$M = J_0 - \lambda I$ becomes

$$M = \begin{pmatrix} -\epsilon - \mu - \lambda & 0 & -\beta s^* & 0 \\ 0 & -(\gamma + \mu) - \lambda & \beta s^* & 0 \\ 0 & \gamma & -(\delta + \mu) - \lambda & 0 \\ \epsilon & 0 & 0 & -\mu - \lambda \end{pmatrix}$$

We get one value of $\lambda = -\mu$

Other two values of λ are given by $(\lambda + (\epsilon + \mu) = 0$ and

$\lambda^2 + \lambda(\gamma + \delta + 2\mu) + (\gamma + \mu)(\delta + \mu) - \beta\gamma s^* = 0$

3.4 The Basic Reproduction Number, R_0

The most important factor in the analysis of disease models, that is, epidemiological models is to determine the behaviour of the solution depending upon the stability of the concerned equilibrium point. Generally the model has one disease-free equilibrium and at least one point of endemic equilibrium. The local stability at DFE (disease-free equilibrium)is determined by threshold parameter known as basic reproduction number denoted by R_0 [6] and [7]. Theoretically R_0 is calculated by the approach given by Van den Driessche .We consider only those terms in which infection is in progress. It is calculated by next generation matrix. Hence, the following system is taken

$e^{\cdot} = \beta is - \gamma e + \mu e,$

$i^{\cdot} = \gamma e - \delta i - \mu i.$

Coefficient matrix is given by

$$Matrix = \begin{pmatrix} -(\gamma + \mu) & \beta s^* \\ \gamma & -(\delta + \mu) \end{pmatrix}$$

The above matrix can be decomposed into two matrices T and Σ, where

$$T = \begin{pmatrix} 0 & \beta s^* \\ 0 & 0 \end{pmatrix}$$

$$\Sigma = \begin{pmatrix} -(\gamma + \mu) & 0 \\ \gamma & -(\delta + \mu) \end{pmatrix}$$

After calculating Σ^{-1}, $T\Sigma^{-1}$ is calculated, which is given by

$$T\Sigma^{-1} = \begin{pmatrix} \frac{s^*\gamma\beta}{AC} & \frac{\beta s^*}{C} \\ 0 & 0 \end{pmatrix}$$

Where $A = (\gamma + \mu)$ and $C = (\delta + \mu)$

Upon calculating the eigen values, we get

$$\lambda = 0 \text{ and } \lambda = \frac{b\beta\gamma}{(\delta+\mu)(\epsilon+\mu)}$$

The dominant eigen value of the above matrix is the value of basic reproduction number. Therefore

$$R_0 = \frac{b\beta\gamma}{(\delta+\mu)(\epsilon+\mu)}$$

4. Numerical Simulations

Computational experiments are performed using Matlab ODE solver ode45 and simulations ran for 10 years. We varied parameters and measured different quantities. In Figure 1, there is no disease present initially and there is no disease present for $t \geq 0$. There is recovered population present in the system. In Figure 2, we assume that $S_0 = 1000, E_0 = 1, I_0 = 0, R_0 = 0$, we observed that the disease establishes itself and the susceptible population goes to zero. In Figure 3, we varied the parameters β and δ over a wide range of values and observed its effect of the susceptible population. We observed that as the disease transmission parameter increases, the susceptible population decreases. The population decreases less when the parameter δ increases.

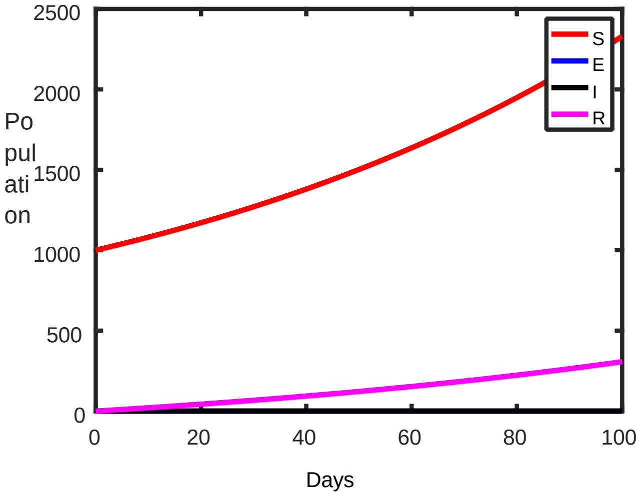

Figure 2: Time series of the population in the absence of disease.

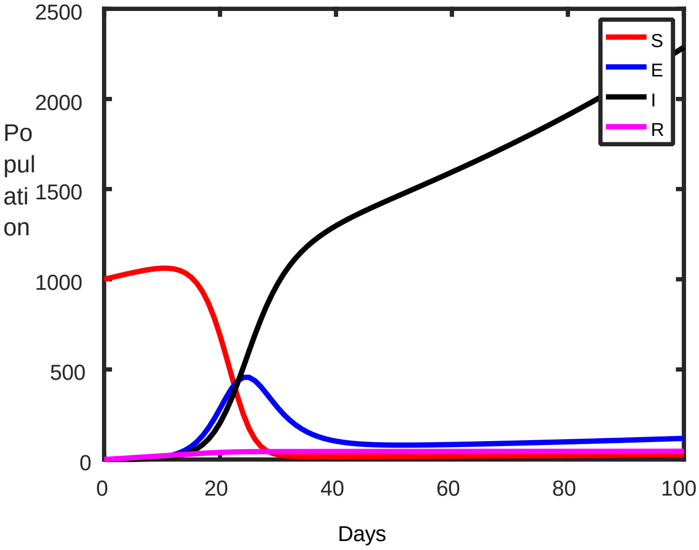

Figure 3: Time series of the population in the presence of disease.

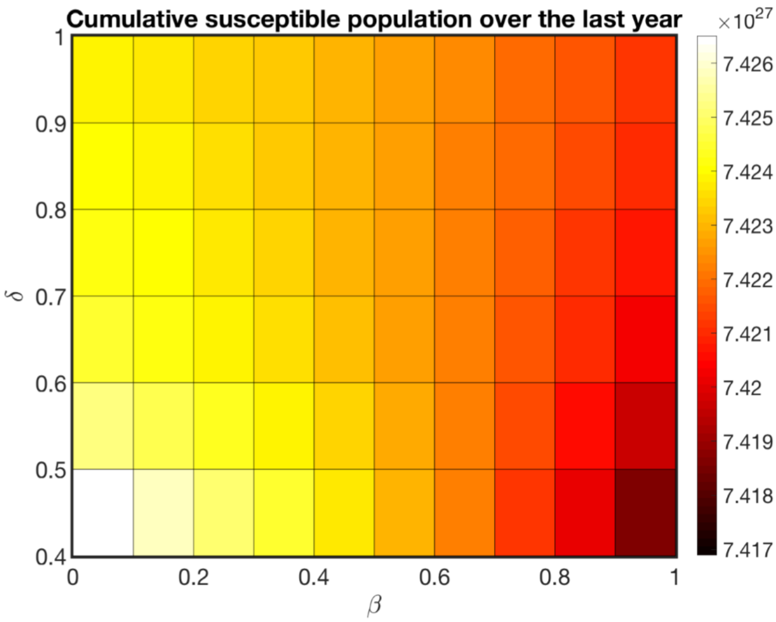

Figure 4: Effect of the parameters *β* and *δ* on the susceptible population at the steady state.

5. Conclusion

After running various simulation experiments, we got a better insight into the dynamics of the model. We observed the effect of the disease transmission parameter on the susceptible population. We also noticed the dynamics of the population at the steady in the absence and presence of the disease. It is quite clear from the expression of R_0, that as the transmission parameter β increases, the value of R_0 increases. The value of basic reproduction number also depends upon γ, which is progression rate. The parameter associated with vaccination affects the value of R_0 inversely. As the effective vaccination rate increases, it helps in reducing the value of R_0. It can be interpreted that by planning the vaccination coverage, its timings, the geographic area to be vaccinated, one can delay the epidemic. Our theoretical results are well in accordance with simulation results.

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