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THEORETICAL ANALYSIS OF EPIDEMIC MODELS WITH RANDOM COEFFICIENTS

K. Anitha[1], R. Malinidevi[2], O. M. Kirthiga[1] and L. Rajendran*[1]

[1]Department of Mathematics, the Madura College, Madurai – 625 011, Tamilnadu, India.

[2]The Standard Fireworks Rajaratnam College for Women, Sivakasi – 626 123, Tamilnadu, India.

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ABSTRACT

Mathematical model of epidemics is discussed. The aim of epidemic modeling is to understand and if possible control the spread of disease. Epidemic models thus express scientific hypotheses. In this paper, an approximate analytical solution for the nonlinear equations that describe epidemiology is derived by employing the Homotopy perturbation method (HPM). The obtained analytical results are compared with simulation results and found to be in satisfactory agreement.

Keywords: *SIR epidemic models, Vaccination, Homotopy perturbation method, Mathematical modeling, Non-linear equations.*

1. INTRODUCTION

Epidemiology is concerned with patterns in space and time of the occurrence of disease. Epidemics of infectious disease are generally caused by a change in the ecology of the host population (e.g. increased stress or increase in the density of a vector species), a genetic change in the parasite population or the introduction of a new parasite to a host population (by movement of parasite or host).

Important control problems nowadays related to life sciences are the control of ecological models like, for instance, the population growth or the regulated harvesting quota as well as the disease propagation via vaccination control. The diseases that motivated the development of modern epidemiological theory are arguably those due to childhood infections, most notably measles [1]. Recently epidemic model has been received a great attention in mathematical ecology. To describe the development of an infectious disease, compartmental models have been given to separate a population into various classes based on the stages of infection [2].

The first model of an epidemic was suggested by Bernoulli in 1760. He used this model to explain the basic control effects obtained through population immunization and the advantages of vaccination in order to prevent an epidemic [3-4]. Analysis of the structure of epidemic models is vital because of the scarcity of good data and the sensitive dependence of results on assumptions [5-6].

Mathematical models have become important tools in analyzing the spread and control of infectious disease. Mathematical models and computer simulations are useful experimental tools for building and testing theories, assessing quantitative conjectures, answering specific questions, determining sensitivities to changes in parameter values, and estimating key parameters from data [7-8]. Mina *et al.* [9] proposed to model the spread of epidemics on networks and also study the role of the whole spectrum of the network. Joshi *et al.* [10] develop a new mathematical modeling approach on immunotherapies and cancer vaccination protocols. Ball *et al.* [11] is concerned with a stochastic SIR model for the spread of an epidemic amongst a population of individuals, with a random network of social contacts that is also partitioned into households. Ye *et al.* [12] studied the interaction of self and cross diffusion can be considered as an important mechanism for the appearance of complex spatiotemporal dynamics in epidemic models. Arenas *et al.* [13] present an epidemiological model to study the transmission dynamics of toxoplasmosis in a cat population under a continuous vaccination schedule.

Corresponding author: L. Rajendran[1]*
[1]Department of Mathematics, The Madura College, Madurai – 625 011, Tamilnadu, India.
E-mail: dr.rajendran.l@gmail.com

In particular the SIR model is a standard compartmental model that has been used to describe many epidemiological diseases. In this model the population divided into three groups: the susceptible (S), infective (I), and the recovered (R). The spread of an infectious disease in a population depends not only on the character of the disease, but also on the structure of the population. Often there are some individuals who have many interactions with others in the population while others have only a few interactions.

Recently, Chen *et al.* [14] studied the time evolution of a system of ordinary differential equations for epidemic models with random inputs using polynomial chaos. To the best of our knowledge, till date no exact analytical solutions of the model have been reported. The purpose of this paper is to present an analytical expression for the concentrations of susceptible group (S), infected group (I), and removed group (R) for all values of parameters using Homotopy perturbation method.

2. MATHEMATICAL FORMULATION AND SOLUTION OF THE PROBLEM

The SIR model is easily written using ordinary differential equations (ODEs), which implies a deterministic model (no randomness is involved, the same starting conditions give the same output), with continuous time (as opposed to discrete time). Comparable to the principles of reaction kinetics, we assume that encounters between infected and susceptible individuals occur at a rate proportional to their respective numbers in the population. The rate of new infections can thus be defined as bSI, where b is a saturation parameter for infectivity. Infected individuals are assumed to recover with a constant probability at any time, which translates into a constant per capita recovery rate that we denote with a, and thus an overall rate of recovery aI. Based on these assumptions the scheme of the model is given Fig.1. The scheme can also be translated into a set of following differential equations [14]:

$$\frac{dS}{dt} = -bSI + cR \text{ (Susceptible)} \tag{1}$$

$$\frac{dI}{dt} = bSI - aI \text{ (Infective)} \tag{2}$$

$$\frac{dR}{dt} = aI - cR \text{ (Removed)} \tag{3}$$

where a, b and c are saturation parameters respectively. A summary of the process is drawn in a flow chart in Fig.1.

The initial conditions at $t = 0$ is given by

$$S(0) = S_t, \quad I(0) = I_t, \quad R(0) = R_t \tag{4}$$

From the eqns. (1-3), the following results are obtained.

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0 \tag{5}$$

$$S(t) + I(t) + R(t) = S_t + I_t \tag{6}$$

3. ANALYTICAL EXPRESSION OF THE PROBLEM USING HOMOTOPY PERTURBATION METHOD

In recent times, many authors have applied the Homotopy perturbation method (HPM) to solve the non-linear problem in physics and engineering sciences [14-17]. This method is also used to solve some of the non-linear problem in physical sciences [18-20]. This method is a combination of Homotopy in topology and classic perturbation techniques. Ji-Huan He used to solve the Light hill equation [18], the Duffing equation [19] and the Blasius equation [20-21]. The HPM is unique in its applicability, accuracy and efficiency. The HPM uses the imbedding parameter p as a small parameter, and only a few iterations are needed to search for an asymptotic solution. The basic concept of Homotopy perturbation method is given in Appendix A. Using this method [22-27] (Appendix B), we can obtain solution of the Eqns. (1) – (3) as follows:

when $c = 0$

$$S(t) = S_t + \left(\frac{S_t I_t b}{a}\right)\left(e^{-at} - 1\right) \tag{7}$$

$$I(t) = I_t\, e^{-at}\left(1 + b\, S_t\, t\right) \tag{8}$$

$$R(t) = \left(\frac{b\, S_t\, I_t}{a}\right)\left(1 - e^{-at} - a\, t\, e^{-at}\right) + R_t \tag{9}$$

4. NUMERICAL SIMULATION

The non-linear differential Eqns. (1)-(3) is also solved by numerical methods using Scilab software. Its numerical solution is compared with Homotopy perturbation method in Figs. (2)– (6) and it gives satisfactory result for all small values of the dimensionless parameters. The Scilab program is also given in Appendix C.

5. RESULTS AND DISCUSSIONS

Eqns. (7-9) are the approximate analytical expressions of susceptible group $S(t)$, infected group $I(t)$, and removed group $R(t)$ using homotopy perturbation technique for the initial conditions (4). The average period of infectiousness at six days, so that would suggest $a = 1/6$. Each infected would make a possibly infecting contact every two fifty six days, then b would be 1/256 [9]. In Fig.2-6 shows the solution curves for these choices of b and a .

From Fig.2.and Fig.3 we can be observed that the value of susceptible decreases gradually from its initial value and reaches the steady state value when $t \geq 15$. For all values of parameters, the infective I increase gradually from its initial value and attains maximum in the interval [0, 10] and decreases gradually, after that attains steady state value when $t \geq 35$ for all values of parameters. The removed R increases slowly from the initial value, attains maximum value in the interval [10, 20] and attains steady state value when $t \geq 20$ The value of susceptible decreases gradually when time increases and it reaches the steady state value for various values of b . The removed R increases slowly when time increases and attains steady state value for various values of a as shown in Fig 4. Fig.5-6 describes the infective versus time t . If time increases the infective I is also increases slowly and it reaches the maximum level at $t = 5$ then it decreases gradually and it reaches the steady state level.

6. CONCLUSION

In this paper, the non-linear equations in SIR epidemic model with constant vaccination strategy are solved analytically using the Homotopy perturbation method. An excellent agreement with the numerical result is noted. The information gained from this theoretical model can be useful for the kinetic analysis of the experimental results and the product distribution.

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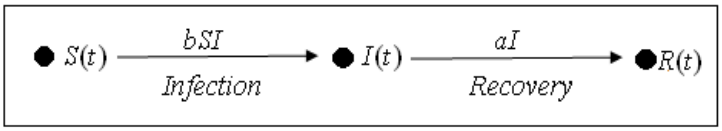

Fig.1: Flow chart for the SIR model.

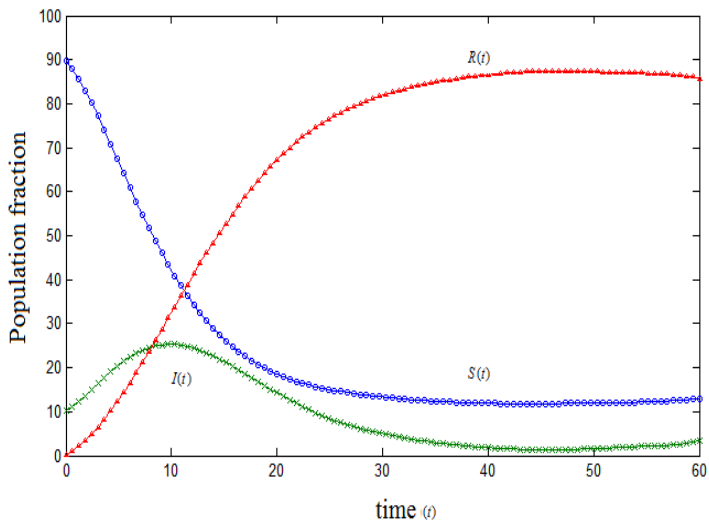

Fig.2: The effect of population fraction versus time for some fixed values of $a = 1/6$ and $b = 1/256$. The key to the graph**:** **( )** represents the analytical solution of the eqns. (7)-(9). (**ooo**) represents the normalized susceptible group S ; (+++) represents the numerical result for normalized infected group I ; and (▲▲▲) represents the numerical result for normalized removed group R .

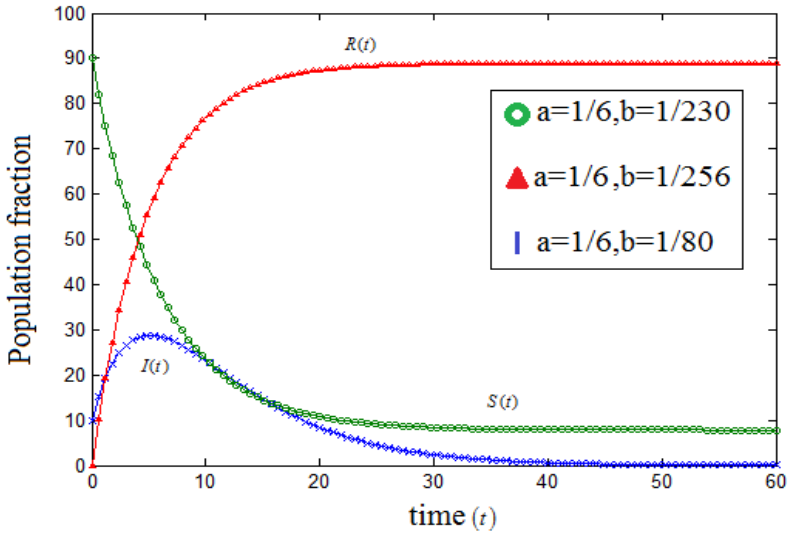

Fig.3: The effect of population fraction versus time for the various values of parameters. The key to the graph: **( )** represents the analytical solution of the eqns. (7)-(9). **(ooo)** represents the normalized susceptible group S; **(+++)** represents the numerical result for normalized infected group I; and **(▲▲▲)** represents the numerical result for normalized removed group R..

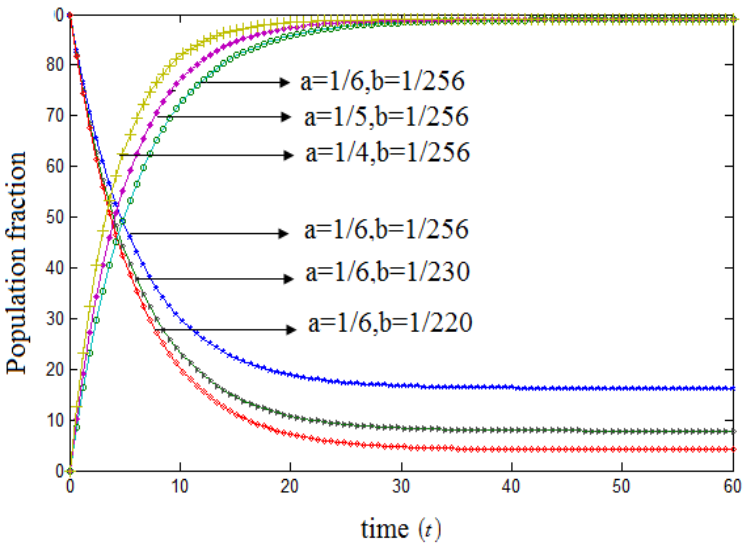

Fig.4: plot of susceptible and removed group versus time *t* using Eqn. (7) and Eqn. (9) for various values of parameters.

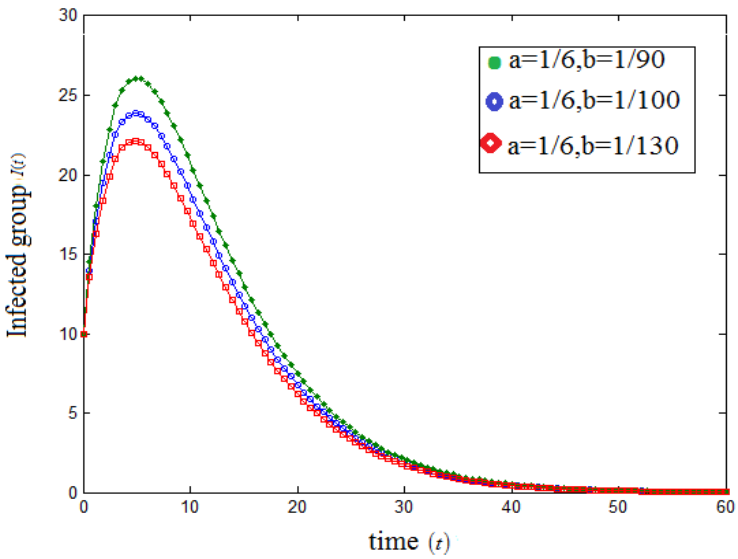

Fig.5: plot of infected group versus time using Eqn. (8) for various values of parameters.

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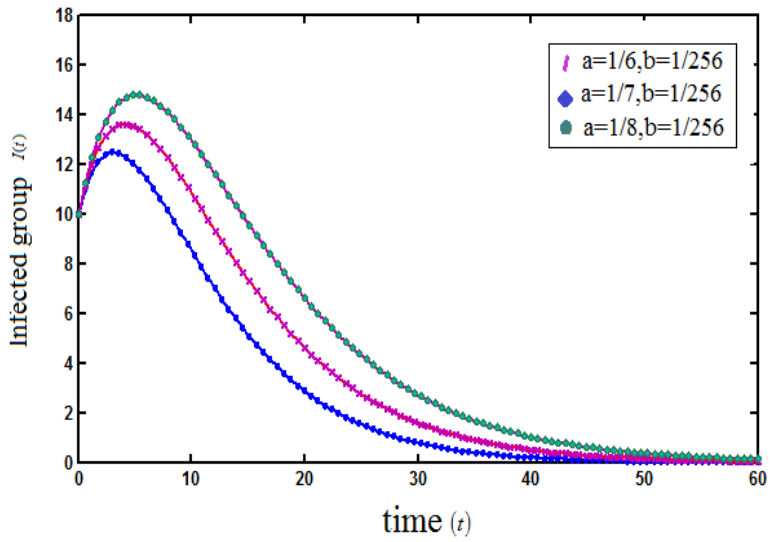

Fig.6: plot of infected group versus time using Eqn. (8) for various values of parameters.

APPENDIX: A

Basic concept of Homotopy perturbation method [14-27]

To explain this method, let us consider the following function:

$$D_o(u) - f(r) = 0, \ \mathrm{r} \in \Omega \tag{A.1}$$

with the boundary conditions of

$$B_o\left(u, \ \frac{\partial u}{\partial n}\right) = 0, \ \mathrm{r} \in \Gamma \tag{A.2}$$

where D_o is a general differential operator, B_o is a boundary operator, $f(r)$ is a known analytical function and Γ is the boundary of the domain Ω. In general, the operator D_o can be divided into a linear part L and a non-linear part N. Eqn. (A.1) can therefore be written as

$$L(u) + N(u) - f(r) = 0 \tag{A.3}$$

By the Homotopy technique, we construct a Homotopy $v(r, p) : \Omega \times [0,1] \to \Re$ that satisfies

$$H(v, p) = (1-p)[L(v) - L(u_0)] + p[D_o(v) - f(r)] = 0. \tag{A.4}$$

$$H(v, p) = L(v) - L(u_0) + pL(u_0) + p[N(v) - f(r)] = 0. \tag{A.5}$$

where $p \in [0, 1]$ is an embedding parameter, and u_0 is an initial approximation of Eqn. (A. 1) that satisfies the boundary conditions. From Eqn. (A.4) and Eqn. (A.5), we have

$$H(v,0) = L(v) - L(u_0) = 0 \tag{A.6}$$

$$H(v,1) = D_o(v) - f(r) = 0 \tag{A.7}$$

When p=0, Eqn. (A.4) and Eqn. (A.5) become linear equations. When p =1, they become non-linear equations. The process of changing p from zero to unity is that of $L(v) - L(u_0) = 0$ to $D_o(v) - f(r) = 0$. We first use the embedding parameter p as a "small parameter" and assume that the solutions of Eqn. (A.4) and Eqn. (A.5) can be written as a power series in p :

$$v = v_0 + pv_1 + p^2 v_2 + \ldots \tag{A.8}$$

Setting $p = 1$ results in the approximate solution of Eqn. (A.1):

$$u = \lim_{p \to 1} v = v_0 + v_1 + v_2 + \ldots \tag{A.9}$$

This is the basic idea of the HPM.

APPENDIX: B

To find the solution of the Eqns. (1) – (3), we construct the homotopy as follows:

$$(1-p)\left(\frac{dS}{dt}\right)+p\left(\frac{dS}{dt}+bSI\right)=0 \tag{B.1}$$

$$(1-p)\left(\frac{dI}{dt}+aI\right)+p\left(\frac{dI}{dt}-bSI+aI\right)=0 \tag{B.2}$$

$$(1-p)\left(\frac{dR}{dt}-aI\right)+p\left(\frac{dR}{dt}-aI\right)=0 \tag{B.3}$$

The analytical solutions of (B.1), (B.2) and (B.3) is

$$S=S_0+pS_1+p^2S_2+..... \tag{B.4}$$

$$I=I_0+pI_1+p^2I_2+..... \tag{B.5}$$

$$R=R_0+pR_1+p^2R_2+..... \tag{B.6}$$

Substituting the eqns. (B.4), (B.5) and (B.6) in (B.1), (B.2) and (B.3) respectively

$$(1-p)\left(\frac{d\left(S_0+pS_1+p^2S_2\right)}{dt}\right)+p\left(\begin{array}{l}\frac{d\left(S_0+pS_1+p^2S_2\right)}{dt}+bS_0I_0+bpS_0I_1+bp^2S_0I_2+bpI_0S_1\\+bp^2S_1I_1+bp^3S_1I_2+bp^{\,2}I_0S_2+bp^3I_1S_2+bp^4S_2I_2\end{array}\right)=0 \tag{B.7}$$

$$(1-p)\left(\frac{d\left(I_0+pI_1+p^2I_2\right)}{dt}+aI_0\right)+p\left(\begin{array}{l}\frac{d\left(I_0+pI_1+p^2I_2\right)}{dt}-bS_0I_0-bpS_0I_1-bp^2S_0I_2-bpS_1I_0\\-bp^2S_1I_1-bp^3S_1I_2-bp^2S_2I_0-bp^3S_2I_1-bp^4S_2I_2\\+aI_0+apI_1+ap^2I_2\end{array}\right)=0 \tag{B.8}$$

$$(1-p)\left(\frac{d\left(R_0+pR_1+p^2R_2\right)}{dt}-aI_0-apI_1-ap^2I_2\right)+p\left(\frac{d\left(R_0+pR_1+p^2R_2\right)}{dt}-aI_0-apI_1-ap^2I_2\right)=0 \tag{B.9}$$

Comparing the coefficients of like powers of p in (B.7), (B.8) and (B.9) we get

$$p^0:\frac{dS_0}{dt}=0 \tag{B.10}$$

$$p^0:\frac{dI_0}{dt}+aI_0=0 \tag{B.11}$$

$$p^0:\frac{dR_0}{dt}-aI_0=0 \tag{B.12}$$

$$p^1:\frac{dS_1}{dt}+bS_0I_0=0 \tag{B.13}$$

$$p^1:\frac{dI_1}{dt}-bS_0I_0+aI_1=0 \tag{B.14}$$

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$$p^1 : \frac{dR_1}{dt} - aI_1 = 0 \tag{B.15}$$

Solving the eqns. (B.10) – (B.15) and using the initial conditions Eqn.(4) we obtain the following results:

$$S_0 = S_t \tag{B.16}$$

$$S_1 = \left(\frac{S_t\, I_t\, b}{a}\right)\left(e^{-at} - 1\right) \tag{B.17}$$

$$I_0 = I_t\, e^{-at} \tag{B.18}$$

$$I_1 = b\, S_t\, I_t e^{-at} t \tag{B.19}$$

$$R_0 = I_t\left(1 - e^{-at}\right) + R_t \tag{B.20}$$

$$R_1 = \left(\frac{b\, S_t\, I_t}{a}\right)\left(1 - e^{-at} - a\, t\, e^{-at}\right) \tag{B.21}$$

APPENDIX: C

Scilab program to find the solutions of the Equations (23)-(27)

```
function main1
options= odeset('RelTol',1e-6,'Stats','on');
Xo = [90;10;0];
tspan = [0,60];
tic
[t,X] = ode45(@TestFunction,tspan,Xo,options);
toc
figure
hold on
plot (t, X(:,1))
plot(t, X(:,2))
plot(t, X(:,3))
return
function [dx_dt]= TestFunction(t,x)
a=1/6;b=1/256;c=0;
dx_dt(1)=-b*x(1)*x(2)+c*x(3);
dx_dt(2)=b*x(2)*x(1)-a*x(2);
dx_dt(3)=a*x(2)-c*x(3);
dx_dt = dx_dt';
```

APPENDIX: D

Nomenclature

Symbols	Definition
S	No.of susceptible
I	No.of infective
R	No.of removed
a, b, c	Saturation parameter
t	Time
S_t	Initial value of susceptible
I_t	Initial value of infective

R_t	Initial value of removed
S_0	First iteration of susceptible
I_0	First iteration of infective
R_0	First iteration of removed
S_1	Second iteration of susceptible
I_1	Second iteration of infective
R_1	Second iteration of removed
p	Embedding parameter

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