Homotopy Perturbation Method for Solving SIR Infectious Disease Model by Incorporating Vaccination

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ABSTRACT

In this paper, we propose a general SIR mathematical model for infectious diseases dynamics for a given constant population. These mathematical models are described by nonlinear first order differential equations. We first find the analytical solution which is obtained using Homotopy Perturbation Method (HPM). The results of the numerical simulation show that the disease has the tendency of dying out over a period of time.

(Keywords: SIR infectious disease model, epidemic model, homotopy perturbation method, HPM)

INTRODUCTION

In spite of the improvement in sanitation, developments of antibiotics and vaccines, infectious diseases still contribute significantly to deaths worldwide. While the earlier recognized diseases like cholera or the plague still sometimes create problems in underdeveloped countries erupting occasionally in epidemics, in the developed countries new diseases are emerging like AIDS (1981) or hepatitis C or E (1989-1990). Additionally, some of the diseases that we generally seem to have been under control like tuberculosis, pneumonia, or gonorrhea develop antibiotic-resistant strains.

Malaria or yellow fever continues to be a major problem in regions with climate changes. New threads constantly appear like the recent bird flu (SARS) epidemic in Asia or the very dangerous Ebola virus in Africa. Overall, infectious diseases continue to be one of the most important health problems worldwide (Hethcote, 1994, 2000).

Infectious diseases are the world's biggest killer of people and account for thousands of deaths per year. There are many infectious diseases in which infection is transmitted by direct contact of susceptible populations with the infected, while there are some diseases like tuberculosis, which are also transmitted indirectly by the flow of bacteria from the infected into the environment.

The poor environmental conditions existing in the densely populated cities of the third world countries have the greatest impact on the spread of bacterial diseases. If the environment is conducive to the growth of bacterial population, then it further helps in the spread of infectious diseases (Ghosh *et al*., 2005, 2006; Naresh *et al*., 2008, 2009; Peter *et al.,* 2018).

One of the most powerful methods to approximately solve linear and non-linear differential equations is the Homotopy Perturbation Method (HPM). The HPM method is based in the use of a power series, which transforms the original non-linear differential equation into a series of linear differential equations. Two continuous functions from one topological space to another are called homotopic if one can be "continuously deformed" into the other, such a deformation is called a homotopy between the two functions which provides analytical approximate solution, is applied to various linear and non-linear equations (He, 1999). The HPM is a series expansion method used in the solution of non-linear partial differential equations (Jiya, 2010).

MATERIALS AND METHODS

In 1927, W.O. Kermach and A.G. McKendrick proposed a mathematical model in which they considered a fixed population with three compartments, Susceptible Class, Infected Class, and Recovered Class. The compartment used in this model consists of three subclasses:

$S(t)$ is used to represent the number of individuals in a population that are prone to the disease at time t, or those susceptible to the disease. $I(t)$ denote the number of individuals in a population who have been infected with the disease and are capable of spreading the disease to those in the susceptible class. $R(t)$ is the compartment used for those individuals in a population who have been infected and then recovered from the disease. Those in this category are not able to be infect those in the susceptible class again or transmit the infection.

The SIRS epidemic models are extensions of SIR models that describe the infection spread when recovery from infection does not lead to permanent immunity. The dynamics of the SIRS epidemic processes differ from the SIR processes in that susceptible become infected, removed by death or recovery and then become susceptible again upon recovery.

FORMULATION OF THE MODEL

The Susceptible subpopulation is generated from daily recruitment of birth at rate β. It is increased as a result of loss of immunity due to recovery and vaccination at the rate σ and decreases due to vaccination and natural death at the rate ρ and μ, respectively.

Sucseptible individuals acquired infection and move to the Infected class via interaction of the Infected and the Susceptible at the rate α.

The Infected class are generated from Susceptible individual that acquired infection. The subpopulation reduced through recovery from infection, disease induced death at the rate γ, δ, respectively.

Furthermore the Recovery subclass are generated from vaccinated Susceptible subpopulation and recovered Infected individuals at the rate ρ and γ respectively. They are reduced due to loss of immunity from recovery and natural death at the rate γ and μ, respectively.

To indicate this mathematically, we have:

$$\frac{dS}{dt} = \beta - \alpha SI - (\rho + \mu)S + \sigma R$$

$$\frac{dI}{dt} = \alpha SI - (\gamma + \delta + \mu)I$$

$$\frac{dR}{dt} = \gamma I - (\mu + \sigma)R + \rho S$$

Where,

$\sigma =$ rate of loss of immunity after recovery/vaccination

$\gamma =$ rate of recovery from infection

$\rho =$ vaccination rate

$\alpha =$ contact rate

$\beta =$ natural birth rate

μ natural death rate

$\delta =$ typhoid fever induced death rate

SOLUTION OF THE MODEL EQUATION BY HOMOTOPY PERTURBATION METHOD

Fundamentals of Homotopy Perturbation Method (HPM) were first proposed by He (2000a).To illustrate the basic ideas of this Method, the following nonlinear differential equation was considered:

$$A(u) - f(r) = 0, r \in \Omega \quad \textbf{(1)}$$

Subject to the boundary condition:

$$B\left(u, \frac{\partial u}{\partial n}\right) = 0, r \in \Gamma \quad \textbf{(2)}$$

Where A is a general differential operator, B a boundary operator, $f(r)$ is a known analytical function and Γ is the boundary of the domain Ω. The operator A can be divided into two parts L and N, where L is the linear part, and N is the nonlinear component. Equation (1) may therefore be rewritten as:

$$L(u)+N(u)-f(r)=0,\ r\in\Omega \qquad \textbf{(3)}$$

The homotopy perturbation structure is shown as follows:

$$H(v,p)=(1-p)\left[L(v)-L(u_0)\right]+p\left[A(v)-f(r)\right]=0 \qquad \textbf{(4)}$$

Where:

$v(r,p):\Omega\in[0,1]\to R$ In eqn. (3) $p\in[0,1]$ is an embedding parameter and u_0 is the first approximation that satisfies the boundary condition. It can be assumed that the solution of eqn. (1) can be written as power series in *p* as follows:

$$v=v_0+pv_1+p^2v_2+..... \qquad \textbf{(6)}$$

and the best approximation for the solution is:

$$u=\lim_{p\to 1}v=v_0+pv_1+p^2v_2+..... \qquad \textbf{(7)}$$

The series (7) is convergent for most cases. However, the convergence rate depends on the nonlinear operator A(v).

Consider the system:

$$\frac{dS}{dt}=\beta-\alpha SI-(\rho+\mu)S+\sigma R \qquad \textbf{(8)}$$

$$\frac{dI}{dt}=\alpha SI-(\gamma+\delta+\mu)I \qquad \textbf{(9)}$$

$$\frac{dR}{dt}=\gamma I-(\mu+\sigma)R+\rho S \qquad \textbf{(10)}$$

With the following initial conditions:

$$S(0)=S_0,\ I(0)=I_0,R(0)=R_0$$

Applying HPM to (8) we have:

$$(1-p)\frac{dS}{dt}+p\left[\frac{dS}{dt}+\alpha SI+(\rho+\mu)S-\sigma R-\beta\right]=0 \qquad \textbf{(11)}$$

Let

$$S(t)=x_0+px_1+p^2x_2+..... \qquad \textbf{(12)}$$

$$I(t)=y_0+py_1+p^2y_2+... \qquad \textbf{(13)}$$

$$R(t)=z_0+pz_1+p^2z_2+... \qquad \textbf{(14)}$$

Substituting Equations (12), (13) and (14) into (11):

$$(1-p)(x_0'+px_1'+p^2x_2'+.....)+ p\left[\begin{array}{l}\left(x_0'+px_1'+p^2x_2'+...\right)+\alpha\left(x_0+px_1+p^2x_2+....\right)\\ \left(y_0+py_1+p^2y_2+...\right)(\rho+\mu)S-\sigma R-\beta\end{array}\right] \qquad \textbf{(15)}$$

Collecting the coefficient of the powers of p we have:

$$p^0:x_0' \qquad \textbf{(16)}$$

$$p^1:x_1'+\mu z_0+\alpha x_0y_0-\rho x_0+\mu x_0-\sigma z_0-\beta=0 \qquad \textbf{(17)}$$

$$p^2:x_2'+\mu z_0+\alpha x_0y_1+\alpha x_1y_1+\rho x_1+\mu x_1-\sigma z_1=0 \qquad \textbf{(18)}$$

$$p^3:\alpha x_0y_2+\alpha x_1y_1+\alpha x_2y_0+\rho x_2+\mu x_2-\sigma z_2=0 \qquad \textbf{(19)}$$

$$p^4:\alpha x_2y_2+\alpha x_2y_1=0 \qquad \textbf{(20)}$$

Applying HPM to (9) we have:

$$(1-p)\frac{dI}{dt}+p\left[\frac{dI}{dt}-\alpha SI+(\gamma+\delta+\mu)I\right]=0 \qquad \textbf{(21)}$$

Substituting Equations (12), (13) and (14) into (21):

$$(1-\rho)(y_0^{'}+py_1^{'}+p^2y_2^{'}+...)+$$
$$p\begin{bmatrix} y_0^{'}+py_1^{'}+p^2y_2^{'}+....-\alpha(x_0+px_1+p^2x_2+..) \\ (y_0+py_1+p^2y_2+.(\gamma+\delta+\mu) \\ (y_0+\rho y_1+\rho^2y_2+,,,). \end{bmatrix}=0$$

(22)

Collecting the coefficient of the powers of the power of p we have:

$$p^1: y_1^{'}-\alpha x_0y_0+(\gamma+\delta+\mu)y_0=0 \quad \textbf{(23)}$$

$$p^2: y_2^{'}-\alpha(x_0y_1+x_1y_0)+(\gamma+\delta+\mu)y_1=0 \quad \textbf{(24)}$$

$$p^3: (\gamma+\delta+\mu)y_2-\alpha(x_1y_1+x_2y_0-x_0y_2)=0 \quad \textbf{(25)}$$

$$p^4: -\alpha(x_1y_1+x_2y_1)=0 \quad \textbf{(26)}$$

Applying HPM to (10) we have:

$$(1-p)\frac{dR}{dt}+p\left[\frac{dR}{dt}-\gamma I+(\mu+\sigma)R-\rho S\right]=0 \quad \textbf{(27)}$$

Substituting Equations (12), (13) and (14) into (27):

$$(1-p)(z_0^{'}+pz_1^{'}+p^2z_2^{'}+...)+$$
$$p\begin{bmatrix} z_0^{'}+pz_1^{'}+p^2z_2^{'}+....-\gamma(y_0+py_1+p^2y_2+...) \\ +(\mu+\sigma)\begin{pmatrix} z_0+pz_1+p^2z_2+.. \\ -\rho(x_0+px_1^{'}+p^2x_2) \end{pmatrix} \end{bmatrix}=0$$

(28)

Collecting the coefficient of the powers of the power of p we have:

$$p^0: z_0^{'} \quad \textbf{(29)}$$

$$p^1: z_1^{'}+\mu z_0+\sigma z_0-\gamma y_0-\rho x_0=0 \quad \textbf{(30)}$$

$$p^2: z_2^{'}+\mu z_1+\sigma z_1-\gamma y_1-\rho x_1=0 \quad \textbf{(31)}$$

$$p^3: \mu z_2+\sigma z_2-\gamma y_2-\rho x_2=0 \quad \textbf{(32)}$$

Solving Equations (16),(17),(18),(19) and (20) we have:

$$S(t)=S_0+(\beta+\sigma R_0-\alpha S_0I_0-\rho S_0-\mu S_0)t+$$
$$[(\gamma I_0-\rho S_0-\mu R_0-\sigma R_0)\sigma-$$
$$(\beta+\sigma R_0-\alpha S_0I_0-\rho S_0-\mu S_0)(\mu+\rho+\alpha S_0I_0)-$$
$$\alpha S_0I_0(\alpha S_0-\gamma-\mu)\ ]\frac{t^2}{2} \quad \textbf{(33)}$$

Solving Equations (23),(24),(25),(26) and (27) we have:

$$I(t)=I_0+[\alpha S_0-\gamma-\delta-\mu]I_0+$$
$$\begin{Bmatrix} \alpha\left[\begin{matrix} S_0(\alpha S_0-\gamma-\delta-\mu)+ \\ (\beta-\sigma R_0-\alpha S_0I_0-\rho S_0-\mu S_0)\ \end{matrix}\right] \\ -(\gamma+\delta+\mu)(\alpha S_0-\gamma-\delta-\mu).\} \end{Bmatrix} I_0\frac{t^2}{2} \quad \textbf{(34)}$$

Solving Equations (29),(30),(31) and (32) we have:

$$R(t)=R_0+(\gamma I_0-\rho S_0-\mu R_0-\sigma R_0)t+$$
$$\begin{bmatrix} 2\mu(R_0\sigma+\rho S_0-\gamma I_0)\gamma I_0(\alpha S_0-\delta-\gamma)+ \\ R_0(\sigma^2-\sigma\rho-\mu^2)+ \\ \rho S_0(\alpha I_0+\rho+\mu)-(\sigma\gamma I_0+\beta\rho)\ \end{bmatrix}\frac{t^2}{2} \quad \textbf{(35)}$$

Equations (33), (34), and (35) are the general solutions of (8), (9) and (10), respectively.

TABULAR AND GRAPHICAL PRESENTATION OF THE MODEL USING MAPPLE 18

This section shows the tables and graphs generated from the general solution of our Model using MAPLE.

We use hypothetical values to generate the tables and graphs for low contracting rate and high recovery rate, respectively.

Table 1: Low Contracting Rate and High Recovery Rate ($\alpha = 0.0001,\ \gamma = 0.16$).

t	S(t)	I(t)	R(t)
0	110	55	35
1	105	45	45
2	101	39	53
3	99	32	60
4	95	27	63
5	92	23	69
6	89	19	74
7	85	16	77
8	83	14	79
9	81	11	80
10	79	9	84

Simulated result for**:**

$\beta = 0.2, \sigma = 0.002, \delta = 0.01, \mu = 0.015, \rho = 0.02$

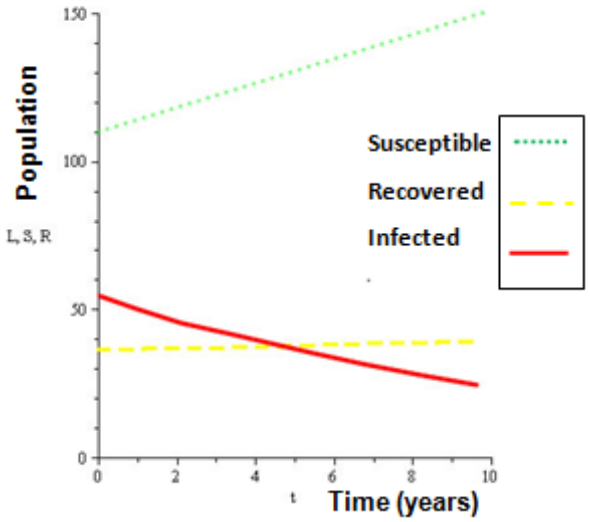

Figure 1: Graphical Profile for Low Contracting Rate and High Recovery Rate ($\alpha = 0.0001,\ \gamma = 0.16$).

Table 2: High Contracting Rate and High Recovery Rate ($\alpha = 0.001,\ \gamma = 0.16$)

t	S(t)	I(t)	R(t)
0	110	55	35
1	100	50	38
2	92	47	53
3	86	42	63
4	80	38	69
5	76	76	33
6	69	31	80
7	67	28	85
8	63	24	88
9	59	22	92
10	58	10	94

Simulated result for:

$\beta = 0.2, \sigma = 0.002, \delta = 0.01, \mu = 0.015, \rho = 0.02$

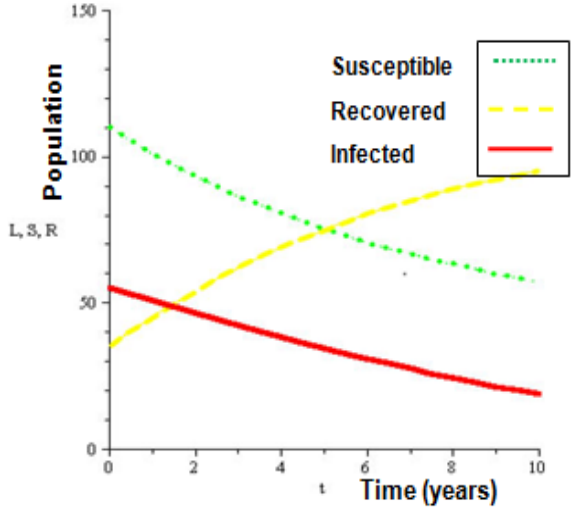

Figure 2: Graphical Profile for High Contracting Rate and High Recovery Rate ($\alpha = 0.001,\ \gamma = 0.16$).

Table 3: Low Contracting Rate and Low Recovery Rate ($\alpha = 0.0001,\ \gamma = 0.1$).

t	S(t)	I(t)	R(t)
0	110	55	35
1	105	48	42
2	103	43	47
3	97	39	53
4	96	33	57
5	91	31	62
6	88	27	66
7	85	23	69
8	82	21	70
9	80	18	73
10	78	17	77

Simulated result for:

$\beta = 0.2, \sigma = 0.002, \delta = 0.01, \mu = 0.015, \rho = 0.02$

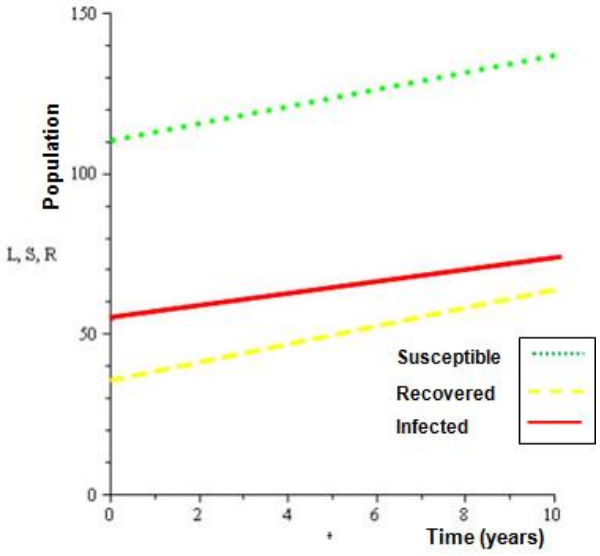

Figure 3: Graph for Low Contracting Rate and Low Recovery Rate $\alpha = 0.0001,\ \gamma = 0.1$.

Table 4: High Contracting Rate and Low Recovery Rate ($\alpha = 0.001,\ \gamma = 0.1$).

t	S(t)	I(t)	R(t)
0	110	55	35
1	100	53	42
2	91	52	47
3	85	50	54
4	78	48	59
5	72	45	66
6	66	43	70
7	62	40	74
8	57	38	77
9	54	35	82
10	51	33	85

Simulated result for:

$\beta = 0.2, \sigma = 0.002, \delta = 0.01, \mu = 0.015, \rho = 0.02$

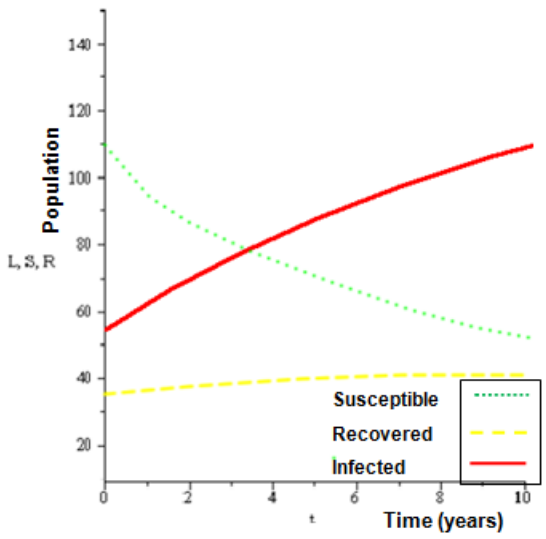

Figure 4: High Contracting Rates and Low Recovery Rate ($\alpha = 0.001,\ \gamma = 0.1$).

DISCUSSION OF RESULTS

The general solutions of the model equation were obtained using Homotopy Perturbation Method (HPM). The graphical profiles of the compartments are presented using MAPPLE computer software package.

Table 1 and Figure 1 are for Low Contracting Rate and High Recovery Rate $\alpha = 0.0001,\ \gamma = 0.16$ we can see from the graph that the population of the Susceptible was increasing, while the population of the infected was decreasing since the recovery rate is high we also noticed that the population of the recovered rise a bit we can conclude here that the disease is under control.

Table 2 and Figure 2 are for the High Contracting Rate and High Recovery Rate $\alpha = 0.001,\ \gamma = 0.16$. From the graph the susceptible population decreased more than when the contacting rate was low and the population of the infected decreases less than when the contracting rate was low. Also the population of the recovered decreases than when the contacting rate was low. The more people are infected the lesser the population of the susceptible and more effort are made to eradicate the disease from the population.

Table 3 and Figure 3 are for Low Contracting Rate and Low Recovery Rate $\alpha = 0.0001,\ \gamma = 0.1$; we noticed from the graph that the susceptible population increased more than when the contracting rate is high.

Table 4 and Figure 4 are for High Contracting Rate and Low Recovery rate $\alpha = 0.001,\ \gamma = 0.1$. The graph show us that the population of the susceptible decreased more than when the recovery rate was high; this shows that more people are infected.

CONCLUSION

Homotopy Perturbation Method (HPM) was used to attempt the approximate solution of each compartment of the Model. The approximate solution was use to present the Model graphically, which gives us the better understanding of the infectious disease dynamics. It is obvious from our results whether high or low contracting rate, the recovered population increases. Also, that increase in contact rate drops the population of the susceptible and the infected population grows exponentially. We can conclude here that the disease have the tendency of dying out.

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