Immunity boosted by low level of exposure to infection in an SIRS model

Asit K. Ghosh, J. Chattopadhyay, P.K. Tapaswi *

Embryology Unit, Indian Statistical Institute, 203, Barrackpore Trunk Road, Calcutta 700 035, India

Received 22 July 1994; accepted 6 January 1995

Abstract

The development of immunity in the susceptible class by a continuous low level of infection is a commonly observed phenomenon in many infectious diseases. This important feature has been incorporated in an SIRS epidemiological model with both the rates of incidence and increase of immunity being nonlinear in nature, instead of being bilinear, of the form $\beta_1 I^p S$ and $\beta_2 I^{p'} S$ ($0 < p' < p$, $p \neq 1$ and $p' \neq 1$) respectively. The local and global behaviour of the dynamics of the model have been investigated.

Keywords: Epidemiology; Incidence rates; Stability

1. Introduction

In the standard SIRS model where S, I and R stand for the fractions of susceptible, infective and immune (recovered) class respectively, the incidence rate is bilinear of the form βIS where β is the transmission rate (Bailey, 1975). By dropping the restriction to bilinear incidence rates, the system can have a much wider range of dynamical behaviour (for details see Capasso and Serio, 1978; Wang, 1978; Cunningham, 1979; Saunders, 1980; Hethcote et al., 1981; Liu et al., 1986,1987; Hethcote and van den Driessche, 1991).

The development of immunity in the susceptible class by a low level exposure to infection is an important and commonly observed phenomenon in many infectious diseases. Low level exposure to infection, in fact, acts as vaccination and develops immunity against the disease. In an endemic area of infectious diseases like malaria, kala-azar, sleeping sickness etc., people who use mosquito nets in the night are not generally infected by the disease, since the small number of mosquito bites they receive when they are outside the nets is not sufficient to cause the disease. On the other hand, they are subject to a small exposure to infection which gives them immunity.

But little attention has so far been paid on this commonly observed phenomenon. Recently

Ghosh and Tapaswi (1995) have incorporated this highly important qualitative feature of epidemic diseases, namely, the increase of immunity by low level of infection, in the generally discussed SIRS model. They have assumed that the incidence rate of the disease is nonlinear of the form $\beta_1 I^p S$ ($p > 1$) whereas the rate of increase of immunity is bilinear of the form $\beta_2 IS$. But in reality, the rate of increase of immunity should also be nonlinear. In this paper, we have considered the rate of increase of immunity by the form $\beta_2 I^{p'} S$ ($0 < p' < p$, $p \neq 1$ and $p' \neq 1$) in the generally discussed SIRS model. The global behaviour of non-zero equilibruim is also an open question in their paper. The global stability properties of this modified form have been discussed in this paper. If we take $\beta_2 = 0$, our model is similar with Liu et al. (1987) by setting $q = 1$ in their model. We have not considered the incidence rate of the form $\beta I^p S^q$, because choosing a q value different from 1 has by itself no major difference in dynamical behaviour (see, Liu et al., 1987).

2. The mathematical model

Let the fraction of the population that are susceptibles, infectives and immunes be denoted by S, I and R respectively. Those who are recovered from infection as well as the susceptibles who are subject to a low level of exposure to infection constitute the immune class R. Let β_1, β_2, γ and δ denote the transmission rate, rate of increase of immunity by low level of exposure to infection, rate of immunity loss and rate of recovery respectively. The birth and death rates are assumed to be equal and denoted by μ so that the population is in equilibrium. New-born individuals belong to the susceptible group.

Our hypothesis in this paper is that the incidence rate of the disease is nonlinear of the form $\beta_1 I^p S$ whereas the rate of increase of immunity is non-linear of the form $\beta_2 I^{p'} S$ (where, $0 < p' < p$, $p \neq 1$ and $p' \neq 1$), that is to say, a higher level of exposure during a certain interval of time is essential for infecting an individual whereas a lower level of exposure during that interval causes immunity in that individual. For viral/bacterial diseases p is the threshold concentration of viruses/bacteria which is to be reached in the environment to infect the susceptibles. A susceptible individual subject to a concentration of viruses/bacteria below this threshold value for a certain period will acquire immunity against the disease. For vectored diseases, p is the average number of infective mosquito bites which a susceptible individual has to receive to become infective or, p is the average number of infective individuals which the vector must attack to acquire a level of pathogens sufficient to make its next attack effective in transmitting the disease (Liu et al., 1986). High level exposure or multiple exposure does not necessarily imply contact of a susceptible with more than one infective, it may come through a high level of intimate contact with the infective and thus high exposure may occur even when a susceptible is subject to a high level of intimate contact with only one infective. In fact, p is a measure of the level of exposure to infection or effective cooperativity among infectives. Considering the above, p is the number of attacks of an infection of a certain strength during a certain interval of time required to infect an individual with the disease whereas, p' ($< p$) is the number of attacks of an infection of the same strength during the same interval of time insufficient to cause the disease but rather giving immunity against the disease.

The differential equations for the SIRS model can be written as

$$\left.\begin{aligned} \frac{dS}{dt} &= -\beta_1 I^p S - \beta_2 I^{p'} S - \mu S + \mu + \gamma R \\ \frac{dI}{dt} &= \beta_1 I^p S - (\delta + \mu) I \\ \frac{dR}{dt} &= \beta_2 I^{p'} S + \delta I - (\gamma + \mu) R \end{aligned}\right\} \qquad (1)$$

where $S + I + R = 1$, $0 < p' < p$, $p \neq 1$ and $p' \neq 1$.

System 1 can be reduced to the 2-dimensional system

$$\left.\begin{aligned}\frac{dI}{dt} &= \beta_1 I^p(1-I-R) - (\delta+\mu)I \\ \frac{dR}{dt} &= \beta_2 I^{p'}(1-I-R) + \delta I - (\gamma+\mu)R\end{aligned}\right\} \quad (2)$$

Introducing

$$K_1 = \beta_1/(\delta+\mu), \quad K_2 = \beta_2/\delta, \quad r = \delta/(\delta+\mu), \quad h = (\gamma+\mu)/\delta \quad (3)$$

and rescaling system 2 by setting

$$T = (\delta+\mu)t \quad (4)$$

We have

$$\left.\begin{aligned}\frac{dI}{dT} &= K_1 I^p S - I \\ \frac{dR}{dT} &= r\left[K_2 I^{p'} S + I - hR\right]\end{aligned}\right\} \quad (5)$$

3. Existence of equilibria

Let $z = p-1$ and $z' = p'-1$. System 5 always has a zero equilibrium (which is called the disease-free equilibrium, in this case $S=1$, $I=0$ and $R=0$). Any non-zero equilibrium (when disease is present) must satisfy

$$S_e = 1 - I_e/G(I_e) \quad (6)$$

$$R_e = \left[\left(1+K_2(1-I_e)I_e^{z'}\right)/\left(h+K_2 I_e^{z'+1}\right)\right] I_e \quad (7)$$

$$1/K_1 = I_e^z\left(1-I_e/G(I_e)\right) \quad (8)$$

where

$$G(I_e) = \left(h+K_2 I_e^{z'+1}\right)/\left(1+h+K_2 I_e^{z'}\right) \quad (9)$$

The basic assumption in our model is $p > p'$, since otherwise, the model will be unrealistic. Since all the parameters are positive, every root I_e of Eq. 8 will be positive if $(1-I_e/G(I_e)) > 0$. This implies that $I_e < I_{max}$, where $I_{max} = h/(1+h)$. If I_e is specified, S_e and R_e are determined by Eqs. 6 and 7, respectively.

Using the same method as in Liu et al. (1986,1987), we can determine the number of non-zero equilibria. Let $f(I_e) = I_e^z(1-I_e/G(I_e))$. Since $I_e/G(I_e)$ is an increasing function of I_e, i.e., $(1-I_e/G(I_e))$ is a decreasing function of I_e when $I_e \in [0, I_{max}]$. We can see that when $-1 < z < 0$, i.e., $0 < p < 1$, there is always a unique non-zero equilibrium since, I_e^z is a decreasing function of I_e on $(0, I_{max}]$ i.e., $f(I_e)$ is a monotonically decreasing function of I_e on $(0, I_{max}]$, and $f(0) = \infty$. When $z > 0$, i.e., $p > 1$, I_e^z is an increasing function of I_e in $(0, I_{max})$, where $f(0) = 0$ and $f(I_{max}) = 0$. Therefore, $f(I_e)$ has a unique interior maximum at $I_e = I_m$ (say). In this case, there are two, one or no non-zero equilibria according to whether K_1 is larger than, equal to, or smaller than K_1^*, where

$$1/K_1^* = f(I_m) = \frac{I_m^{z+1}\left[G(I_m) - I_m\left|\frac{dG}{dI_e}\right|_{I_e=I_m}\right]}{z\left(G(I_m)\right)^2}$$

and

$$S_m = 1 - I_m/\left[G(I_m)\right] = \frac{I_m\left[G(I_m) - I_m\left|\frac{dG}{dI_e}\right|_{I_e=I_m}\right]}{z\left(G(I_m)\right)^2}$$

When $K_1 > K_1^*$, the two non-zero equilibria can be ranked as smaller if $0 < I_e < I_m$ and larger if $I_m < I_e < I_{max}$. If $K_1 \to K_1^*$ from the above, then both two non-zero equilibria approach the non-zero equilibrium I_m.

4. Local stability analysis

In this section we shall study the local behaviour of zero and non-zero equilibria respectively. The case $z < 0$ and $z' < 0$, i.e., $p < 1$ and $p' < 1$. For this case, let $U = I^{-z}$. Using this substitution, we can transform system 5 to the following form:

$$\frac{dU}{dT} = -z(K_1 S - U)$$

$$\frac{dR}{dT} = r\left[K_2 U^{-(z'+1)/z} S + U^{-(1/z)} - hR\right]$$

where

$$S = 1 - U^{-(1/z)} - R.$$

We observe that $U = R = 0$ is not even an equilibrium, we conclude that the zero equilibrium in system 5 must be unstable. Thus, solutions which start near (0,0) in system 5 can not tend to it asymptotically. The case $z > 0$ and $z' > 0$, i.e., $p > 1$ and $p' > 1$. In this case, the Jacobian matrix J_0 at $I = R = 0$ is

$$J_0 = \begin{pmatrix} -1 & 0 \\ r & -hr \end{pmatrix}$$

Thus, the eigenvalues of J_0 are all negative (real). Hence, the zero equilibrium is locally asymptotically stable (LAS).

By Eq. 8, the Jacobian matrix of system 5 at the non-zero equilibrium (I_e, R_e) can be written as

$$J_e = \begin{pmatrix} z - \dfrac{I_e}{S_e} & -\dfrac{I_e}{S_e} \\ r\left[K_2 I_e^{z'}((z'+1)S_e - I_e) + 1\right] & -r\left(h + K_2 I_e^{z'+1}\right) \end{pmatrix}$$

Therefore, the characteristic equation of the matrix J_e is

$$\lambda^2 + \sigma_1 \lambda + \sigma_2 = 0$$

where

$$\sigma_1 = -\left[(z - hr) - \left(K_2 r I_e^{z'+1} + \frac{I_e}{S_e}\right)\right]$$

and

$$\sigma_2 = -r\left[\left(h + K_2 I_e^{z'+1}\right)z - \left((z'+1)K_2 I_e^{z'+1} + (1+h)\frac{I_e}{S_e}\right)\right].$$

Since I_e/S_e is an increasing function of I_e, $I_e/S_e \to \infty$ as $I_e \to I_{max} = h/(1+h)$. Thus, it is clear that σ_1 and σ_2 are two increasing functions of I_e in $(0, I_{max})$. It is obvious that $\sigma_2 = 0$ at $I_e = I_m$. Thus, $\sigma_2 < 0$ when $I_e \in (0, I_m)$ and $\sigma_2 > 0$ when $I_e \in (I_m, I_{max})$.

When $z < 0$. The unique non-zero equilibrium is LAS. When $z > 0$. In this case, when $K_1 > K_1^*$, we have two non-zero equilibria, one which is smaller is always unstable saddle and the other which is larger is LAS for $z \le hr$. When $z > hr$. Let $\sigma_1 = B(I_e)/[h - (1+h)I_e]$, where

$$B(I_e) = K_2[(1-r) - hr]I_e^{z'+2} + K_2 hr I_e^{z'+1} + [h + (1+h)(z - hr)]I_e - h(z - hr)$$

(using Eqs. 6 and 9). Therefore, $B(I_e = 0) = -h(z - hr) < 0$ and $B(I_{max} = h/(1+h)) = I_{max}^2[(1+h) + K_2((1-r)I_{max} + hr(1 - I_{max}))I_{max}^{z'-1}]$ which is greater than zero. Since σ_1 is an increasing function of I_e in $(0, I_{max})$, we can find a unique positive root I_1 (say) of the equation $\sigma_1(I_e) = 0$ in $(0, I_{max})$. Thus, $\sigma_1 > 0$ if $I_e \in (I_1, I_{max})$. Hence, the larger equilibrium is always LAS if $I_1 < I_m$, when $z > hr$, i.e., $p > 1 + hr$, where I_m is the unique positive root of the equation $df(I_e)/dI_e = 0$ in $(0, I_{max})$.

5. Global stability analysis

First, we shall study the global stability of the zero equilibrium and then the stability of the non-zero equilibria.

If $I = 0$, then from system 5, we see that $R \to 0$ as $T \to \infty$. Let us consider the Lyapunov function, $L = I$. So,

$$\frac{dL}{dT} = I\left[K_1 I^z(1 - I - R) - 1\right] \tag{10}$$

We have

$$K_1 I^z(1 - I - R) \le K_1 I^z(1 - I) \tag{11}$$

Let $g(I) = I^z(1 - I)$. So, $g(I)$ attains a maximum value $1/K_1^1$ (say) at $I = z/(z+1)$, where

$$\frac{1}{K_1^1} = g\left(\frac{z}{z+1}\right) = \frac{z^z}{(z+1)^{z+1}}.$$

Therefore, $K_1^1 = (z+1)^{z+1}/z^z$. Thus, Eq. 11 can be expressed as

$$K_1 I^z(1 - I - R) \le K_1 I^z(1 - I) \le \frac{K_1}{K_1^1},$$

when $K_1 < K_1^1$. In this case, from Eq. 10 we get $dL/dT \leq 0$. It is obvious from Eq. 10 that $I = R = 0$ is the largest invariant subset in the set where $dL/dT = 0$. So by a Lasalle extension of the Lyapunov theorem (see, e.g., Hale, 1969; Miller and Michel, 1982, p. 227), the zero equilibrium (when $K_1 < K_1^1$) is globally asymptotically stable (GAS). Hence, we conjecture that the zero equilibrium is GAS when $K_1 < K_1^r$.

We shall now find out the criterion for the global stability of the non-zero equilibrium (I_e, R_e). We define the following positive definite function $V(I,R)$ as:

$$V(I,R) = I - I_e - I_e \log\frac{I}{I_e} + \left(\tfrac{1}{2}R - R_e\right)^2$$

where (I_e, R_e) is the non-zero equilibrium. Note that $V(I,R)$ is a positive definite function for all $(I,R) \neq (I_e, R_e)$. The derivative along the solutions of system 5 is

$$\begin{aligned}
\frac{dV}{dT} &= (I - I_e)\frac{1}{I}\frac{dI}{dT} + (R - R_e)\frac{dR}{dT} \\
&= K_1(I - I_e)\left[I^{p-1} - I^p - I^{p-1}R - I_e^{p-1}\right. \\
&\quad \left. - I_e^p + I_e^{p-1}R_e\right] + r(R - R_e) \\
&\quad \times \left[K_2 I^{p'} - K_2 I^{p'+1} - RK_2 I^{p'} - K_2 I_e^{p'}\right. \\
&\quad \left. + K_2 I_e^{p'+1} + R_e K_2 I_e^{p'} + (I - I_e)\right. \\
&\quad \left. - h(R - R_e)\right] \\
&= K_1(I - I_e)\left[(I - I_e)f_1(I) - (I - I_e)f_{11}(I)\right. \\
&\quad \left. - R(I - I_e)f_1(I) - I_e^{p-1}(R - R_e)\right] \\
&\quad + r(R - R_e)\left[K_2(I - I_e)f_2(I)\right. \\
&\quad \left. - K_2(I - I_e)f_{22}(I) - K_2 I_e^{p'}(R - R_e)\right. \\
&\quad \left. - K_2 R(I - I_e)f_2(I) + (I - I_e)\right. \\
&\quad \left. - h(R - R_e)\right] \\
&= K_1(I - I_e)\left[(I - I_e)(1 - R)f_1(I)\right. \\
&\quad \left. - (I - I_e)f_{11}(I) - I_e^{p-1}(R - R_e)\right] \\
&\quad + r(R - R_e)\left[K_2(I - I_e)(1 - R)f_2(I)\right. \\
&\quad \left. - K_2(I - I_e)f_{22}(I)\right. \\
&\quad \left. - (R - R_e)(K_2 I_e^{p'} + h) + (I - I_e)\right] \\
&= -\left[K_1(I - I_e)^2\left(f_{11}(I) - (1 - R)f_1(I)\right)\right. \\
&\quad + (R - R_e) \\
&\quad \times (I - I_e)\left(K_1 I_e^{p-1} - rK_2(1 - R)f_2(I)\right. \\
&\quad \left. + rK_2 f_{22}(I) - r\right) \\
&\quad \left. + r(R - R_e)^2(K_2 I_e^{p'} + h)\right]
\end{aligned}$$

Clearly $f_1(I)$, $f_2(I)$, $f_{11}(I)$ and $f_{22}(I)$ are positive. Eq. 12 can be written as

$$\frac{dV}{dT} = -X^T J X$$

which is negative definite if the matrix J is positive definite, where $X = (I - I_e, R - R_e)$, X^T denotes the transpose of the matrix X, and

$$J = \begin{pmatrix} K_1[f_{11}(I) - (1 - R)f_1(I)] & (1/2)[K_1 I_e^{p-1} \\ & + r(K_2 f_{22}(I) \\ & - K_2(1 - R)f_2(I) - 1)] \\ (1/2)[K_1 I_e^{p-1} + r(K_2 f_{22}(I) & \\ - K_2(1 - R)f_2(I) - 1)] & r(K_2 I_e^{p'} + h) \end{pmatrix},$$

Since $r(K_2 I_e^{p'} + h) > 0$, the matrix J will be positive definite if $det(J) > 0$.

To show $det(J) > 0$, for simplicity, we assume that $p = p' + 1$. Then we have

$$\begin{aligned}
f_3(I) &= f_{11}(I) - (1 - R)f_1(I) \\
&= f_{22}(I) \quad (1 - R)f_2(I).
\end{aligned}$$

Now, expanding $det(J)$, we obtain

$$det(J) = \tfrac{1}{4}\left[-a_0(f_3(I))^2 + a_1 f_3(I) - a_2\right]$$

where

$$a_0 = K_2^2 r^2$$

$$a_1 = 2r\left[K_2 r + K_1(2h + K_2 I_e^{p'})\right]$$

$$a_2 = (K_1 I_e^{p'} - r)^2.$$

$det(J)$ is a quadratic in $f_3(I)$ having two roots F_1 and F_2 at $I = I'$ and $I = I''$ (say) respectively for some $p'(= p - 1)$, where $0 < I' < I'' < 1$. Therefore, $det(J) > 0$ when $I \in (I', I'')$.

Hence, the non-zero equilibrium is GAS for all initial values of $I \in (I', I'')$ and $p = p' + 1$.

6. Discussion

The main idea behind this paper is that small doses of infection during a certain interval of time do not infect the susceptibles, but on the other hand, give immunity to them whereas when the level of infection during that interval of time crosses a certain threshold value, the individuals become infected. Exploring this commonly observed phenomenon we have proposed a modified SIRS model incorporating nonlinear rates of incidence and increase of immunity of the form $\beta_1 I^p S$ and $\beta_2 I^{p'} S$ ($0 < p' < p$, $p \neq 1$ and $p' \neq 1$) respectively. There are certainly abundant examples of such types of disease spread. It has been observed that people living in a certain region where a particular disease is endemic get generally accustomed to the disease in the sense that some of them acquire immunity against the disease by a long exposure to a low level of infection (below a certain threshold value) whereas susceptible persons from outside the region who come to visit may become infected with the disease by being suddenly exposed to a high dose of infection (above the threshold value).

We have assumed that p doses of infection in a certain interval of time are required to infect a susceptible whereas p' ($< p$) number of exposures of the infection during the same interval of time is not sufficient to cause the disease but rather it gives immunity against the disease.

We have shown that, whatever is the value of p (> 0), there always exists zero (disease-free) equilibrium. When $p < 1$, there is a unique non-zero (disease-present) equilibrium which is LAS for any value of p' ($< p$). It is also observed that when $p > 1$ and $p' > 1$, there are two, one or no non-zero equilibria according to whether K_1 is larger than, equal to, or smaller than K_1^*. In this case, the zero equilibrium is LAS whereas this equilibrium is also GAS if $K_1 < K_1^*$ and if $K_1 > K_1^*$ (i.e., when two non-zero equilibria exist) the smaller non-zero equilibrium is always an unstable saddle whereas the larger non-zero equilibrium is LAS if $p \leq 1 + hr$ and if $p > 1 + hr$, the larger equilibrium is always LAS when $I_l < I_m$ (I_l and I_m are the positive roots of the equations $\sigma_1(I_c) = 0$ and $\mathrm{d}f(I_c)/\mathrm{d}I_c = 0$, respectively).

Another remarkable point is the global stability of the non-zero equilibrium. By constructing a suitable Lyapunov function, we have observed that the non-zero equilibrium is GAS under some parametric conditions for all initial values of $I \in (I', I'')$ and $p = p' + 1$.

Finally, we may conclude that our findings also corroborate the results of Ghosh and Tapaswi (1995), Liu et al. (1987) and Mukherjee et al. (1993). By setting $p' = 1$, the results of Ghosh and Tapaswi (1995) follow. Also the results of Liu et al. (1987) follow when $\beta_2 = 0$ in our model case and setting $q = 1$ in the model of Liu et al. (1987). Mukherjee et al. (1993) demonstrated the global results of the model of Liu et al. (1987). These findings also follow from our global results by setting $\beta_2 = 0$ in our model and $q = 1$ in their model.

The stability of the non-zero equilibrium for $p' < p$ implying endemicity of the disease depends upon the values of p and p'. Thus, control of the disease may be achieved by controlling the value of p and p'.

References

Bailey, N.T.J., 1975. The mathematical theory of infectious diseases and its application. Griffin, London.

Capasso, V. and Serio, G., 1978. A generalization of the Kermack–McKendrick deterministic epidemic model. Math. Biosci., 42: 41–61.

Cunningham, J., 1979. A deterministic model for measles. Z. Naturforsch., 34C: 647–648.

Ghosh, A.K. and Tapaswi, P.K., 1995. An SIRS epidemiological model with immunity caused by low level of infection. Cybernetica (in press).

Hale, J.K., 1969. Ordinary Differential Equations. Wiley-Interscience, New York.

Hethcote, H.W. and van den Driessche, P., 1991. Some epidemiological models with nonlinear incidence. J. Math. Biol., 29: 271–287.

Hethcote, H.W., Stech, H.W. and van den Driessche, P., 1981. Nonlinear oscillations in epidemic models. SIAM J. Appl. Math., 40: 1–9.

Liu, W.M., Levin, S.A. and Iwasa, Y., 1986. Influence of nonlinear incidence rates upon the behaviour of SIRS epidemiological models. J. Math. Biol., 23: 187–204.

Liu, W.M., Hethcote, H.W. and Levin, S.A., 1987. Dynamical behaviour of epidemiological models with nonlinear incidence rates. J. Math. Biol., 25: 359–380.

Miller, R.K. and Michel, A.N., 1982. Ordinary Differential Equations. Academic Press, New York.

Mukherjee, D., Chottapadhyay, J. and Tapaswi, P.K., 1993. Global stability results of epidemiological models with nonlinear incidence rates. Math. Comput. Model., 18: 89–92.

Saunders, I.W., 1980. A model for myxomatosis. Math. Biosci., 48: 1–15.

Wang, F.J.S., 1978. Asymptotic behaviour of some deterministic epidemic models. SIAM J. Math. Anal., 9: 529–534.