Global stability results of a "susceptible–infective–immune–susceptible" (SIRS) epidemic model

P.K. Tapaswi *, J. Chattopadhyay

Embryology Research Unit, Indian Statistical Institute, 203, B.T. Road, Calcutta 700 035, India

Received 16 August 1994; accepted 9 February 1995

Abstract

In this paper, we have investigated the global behaviour of the SIRS epidemic model under the assumption that some portion of immunes are infective as proposed by J.L. Aron and the immunity is lost at a constant rate. We have shown that existence of local stability properties guarantees their global stability.

Keywords: Epidemiology; Immunity; Stability

1. Introduction

Aron (1988) has proposed and investigated the dynamical behaviour of boosted immunity in a simple SIRS epidemic model with vital dynamics. The whole population is divided into three classes: S representing the susceptible class, I representing the infective class and R representing the recovered and immune class. Immunity is not permanent. The immune class enters the susceptible class after a specific time, and the cycle SIRS is completed. Aron in his paper has worked out the criteria for existence and local stability character of the zero and nonzero equilibrium. However, the problem of global stability has been an open question in that paper. In this paper the problem has been tackled using Liapunov direct method, Bendixon–Dulac criteria and Bendixon–Dulac criterion as a special case, under the assumption that the duration of immunity is independent of exposure to infection.

2. The mathematical model

We assume the fractions of the populations that are susceptibles, infectives and immunes are denoted by S, I and R, respectively. Susceptibles become infected by mass action contact between susceptibles and infectives. The mass action term incorporates a coefficient of mixing β, the proportion of infected who are infective f, and the proportion of immunes who are infective f', where $f' < f$. Infective individuals in class I recover at a rate q to enter the immune class.

Immune individuals become susceptible again at a constant rate γ. Deaths occur at a rate μ, unaffected by age or disease status. Life expectancy is then $1/\mu$. Deaths are balanced by births into the susceptible class so that the population size remains constant.

With the assumptions the model mechanism is then of the form

$$\begin{aligned}\frac{dS}{dt} &= -\beta(fI+f'R)S+\gamma R+\mu-\mu S\\ \frac{dI}{dt} &= -\beta(fI+f'R)S-(q+\mu)I\\ \frac{dR}{dt} &= qI-(\gamma+\mu)R\end{aligned} \tag{1}$$

where $S+I+R=1$, $0\le f'\le f$ and $0\le f\le 1$.

Aron (1988) has worked out the basic reproductive rate R_0, which is given by

$$R_0=\frac{\beta f(\gamma+\mu)+\beta f'q}{(q+\mu)(\gamma+\mu)} \tag{2}$$

and shown that if $R_0<1$, the zero equilibrium is locally asymptotically stable and there is no other equilibrium. On the other hand, if $R_0>1$, there exits two equilibrium points: (i) the zero equilibrium which is locally unstable and (ii) the nonzero equilibrium which is locally stable as shown by Aron (1988). In the following section we shall prove the global dynamics of the above mentioned cases.

3. Global stability

Theorem 1. *If $R_0<1$, the existence of local stability implies its global stability.*

Proof. We define the positive definite function

$$V=(\gamma+\mu)I+\beta f'R \tag{3}$$

The time derivative along solution of Eq. 1 is

$$\begin{aligned}\frac{dV}{dt} &= I\{\beta f(\gamma+\mu)+\beta f'q-(q+\mu)(\gamma+\mu)\}\\ &\quad -(I+R)(\gamma+\mu)\beta(fI+f'R)\\ &= (\gamma+\mu)\left[I(q+\mu)(R_0-1)\right.\\ &\quad \left.-\beta(I+R)(fI+f'R)\right]\\ &\quad (\text{as } R_0<1)\end{aligned}$$

Hence the theorem.

Now, if $R_0>1$, the zero equilibrium is locally unstable as mentioned in Section 2. In the next theorem, we shall show that there can not be any closed orbit around this equilibrium by using Bendixon–Dulac criterion.

Theorem 2. *There will be no closed trajectory in the feasibility region*

$$A=\{I\ge 0,\ R\ge 0,\ I+R\le 1\}$$

Proof. Let

$$h(I,R)=1 \tag{4}$$

Obviously $h(I,R)>0$, if $I>0$ and $R>0$. We denote

$$\begin{aligned}k_1(I,R) &= \beta(fI+f'R)(1-I-R)-(q+\mu)I\\ k_2(I,R) &= qI-(\gamma+\mu)R\\ \Delta(k_1,k_2) &= \frac{\delta(k_1h)}{\delta I}+\frac{\delta(k_2h)}{\delta R}\end{aligned}$$

Then

$$\begin{aligned}h(I,R)k_1(I,R) &= \beta(fI+f'R)(1-I-R)\\ &\quad -(q+\mu)I\\ h(I,R)k_2(I,R) &= qI-(\gamma+\mu)R\end{aligned}$$

and

$$\begin{aligned}\Delta(k_1,k_2) &= \beta f-2\beta fI-\beta fR-(q+\mu)\\ &\quad -(\gamma+\mu)\end{aligned} \tag{5}$$

From Eq. 5 it is clear that when $I=1$, $R=0$, then $\Delta(k_1,k_2)<0$ or when

$I=0$, $R=1$ then $\Delta(k_1,k_2)<0$.

Hence the Bendixon's negative criterion does not hold. Thus there can not be any closed trajectory in the feasibility region

$$A=\{I\ge 0,\ R\ge 0,\ I+R\le 1\}$$

Hence the theorem.

Thus we note that local instability of the zero equilibrium implies its global instability also.

If $R_0>1$, the nonzero equilibrium is locally asymptotically stable as mentioned in Section 2.

In the following theorem we shall show that the nonzero equilibrium is globally asymptotically stable by using Bendixon–Dulac criterion as a special case.

Theorem 3. *The model system 1 has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region H.*

Proof. We consider the feasibility region

$$H = \{S \geq 0,\ I \geq 0,\ R \geq 0,\ S + I + R \epsilon R_3^+ :\ S + I + R = 1\}$$

and we define $H_0 = H - \{1,0,0\}$.

Our proof is based on the following result, which includes the well-known Dulac criterion as a special case (see Busenberg and van den Driessche, 1990, theorem 4.1, p. 268).

Let

$$g(S,I,R) = \{g_1(S,I,R),\ g_2(S,I,R),\ g_3(S,I,R)\}$$

be a vector field which is piecewise smooth on compact subsets contained in the interior of H and which satisfies the conditions $g \cdot F = 0$ and $(\text{curl } g) \cdot (1,1,1) < 0$ on $H_0 = H - \delta H$, where δH is the boundary of H and $F(f_1,f_2,f_3)$ is a Lipschitz continuous field on H_0. Then the differential equation system $dS/dt = f_1$, $dI/dt = f_2$, $dR/dt = f_3$ has no periodic solutions, homoclinic loops, or oriented phase polygon in H_0.

Let f_1, f_2 and f_3 denote the right hand side of system 1 respectively and use the relation $S + I + R = 1$ to rewrite these in the equivalent forms:

$$f_1(S,I) = -\beta\{fI + f'(1-S-I)\}S + \gamma(1-S-I) + \mu - \mu S$$

$$f_1(S,R) = -\beta\{f(1-S-I) + f'R\}S + \gamma R + \mu - \mu S$$

$$f_2(S,I) = \beta\{fI + f'(1-S-I)\}S - (q+\mu)I$$

$$f_2(I,R) = \beta(fI + f'R)(1-I-R) - (q+\mu)I$$

$$f_3(S,R) = q(1-S-R) - (\gamma+\mu)R$$

$$f_3(I,R) = qI - (\gamma+\mu)R$$

Let $g = (g_1,g_2,g_3)$ be a vector field, where

$$g_1 = -\frac{f_3(S,R)}{SR} - \frac{f_2(S,I)}{SI}$$

$$= \frac{q}{SR} - \frac{q}{R} - \frac{\gamma}{S} - \beta f - \frac{\beta f'}{I} + \frac{\beta f' S}{I} + \beta f'$$

$$g_2 = \frac{f_1(S,I)}{SI} - \frac{f_3(I,R)}{IR}$$

$$= -\beta f - \frac{\beta f'}{I} + \frac{\beta f' S}{I} + \beta f' + \frac{\gamma}{SI} - \frac{\gamma}{S} + \frac{\mu}{SI} - \frac{q}{R}$$

$$g_3 = \frac{f_2(I,R)}{IR} - \frac{f_1(S,R)}{SR}$$

$$= \frac{2\beta f}{R} - \frac{\beta f I}{I} - \frac{\beta f S}{R} - 2\beta f + \frac{\beta f'}{I} - \frac{\beta f' R}{I} - \frac{q}{R} - \frac{\gamma}{S} - \frac{\mu}{SR}$$

Clearly, $g \cdot F = 0$ on H_0, since the alternate forms of f_1, f_2 and f_3 are equivalent on H.

A few computations yield the expression

$$(\text{curl } g) \cdot (1,1,1) = -\frac{2\beta f' - \beta f'(S+I+R)}{I^2} - \frac{q}{SR^2} - \frac{\mu}{S^2R} - \frac{\gamma+\mu}{S^2I} < 0,$$

since $S + I + R = 1$

Remark: Since the region H is invariant and for $R_0 > 1$, the zero equilibrium is unstable, by Poincaré–Bendixon theorem and Theorem 3 above, the existence of local asymptotic stability of this model system ensures its global asymptotic stability.

4. Conclusion

Thus if $R_0 < 1$, the zero equilibrium –which is the only equilibrium –is globally asymptotically stable, i.e., the disease does not persist, whatever be the initial numbers of infectives in the populations.

On the other hand, if $R_0 > 1$, the zero equilibrium is globally unstable and the nonzero equilibrium is globally asymptotically stable, i.e., the disease is endemic in the global sense.

Biologically, the above result implies that if the basic reproductive rate is below a threshold value, the disease cannot spread and dies out, for any initial size of the infectives in the population. On the other hand, if the basic reproductive rate is above that threshold value the disease will certainly spread and will attend a stable nonzero value called the endemic equilibrium, whatever small the size of infectives the population may initially have.

References

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