

The SIRS Model Approach to Host/Parasite Relationships

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Abstract

In this paper, we shall explore examples of host/parasite relationships, specifically viral infections, and how they can be described by the SIRS model.

1. The SIRS Model For Disease

When considering the many problems that have faced human society, one of the most deadly and devastating is that of disease. Mathematical models have often been used as attempts towards greater understanding of diseases and epidemics, what factors are involved, and perhaps why certain massive epidemics have occurred. An excellent analytical approach to try and describe how a viral infection spreads is the SIRS model. This model, which owes much of its early development to Kermack and McKendrick, is based on the premise that you can describe a population in terms of three different groups: those who are susceptible to a virus, those infected with the virus, and those who have already recovered from the viral infection. To describe this with a formula, the first step is to define N be the total population subsisting of three groups: susceptible individuals S , infected individuals I , and recovered individuals R . Thus

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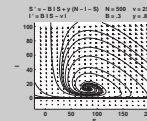
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$$N = S + I + R. \quad (1)$$

Within the population N , there is movement from one group to another. As susceptible, uninfected people in group S become infected, they enter the infected group I . This rate of transmission is related to the amount of contact between those without the disease, S , and those with the disease, labeled I . A reasonable way of describing the rate of transmission then is by multiplying the product IS (representing the contact between group I and group S) by the constant rate of infection which we will label β . So far, the rate of change of the group S can be described as follows:

$$\frac{dS}{dt} = -\beta IS.$$

Not only are there people becoming infected with the virus however, there are also individuals from the recovered group, R , who are losing their immunity and becoming susceptible again. Therefore, a certain number of people will be re-entering the susceptible stage as they lose their immunity at a constant rate which we will call γ . The decrease in the Recovered population due to this loss of immunity will be given by the product of the recovered population, R , and γ . Adding this to our equation above, we get a completed description of the rate of change of the population S in the following equation

$$\frac{dS}{dt} = -\beta IS + \gamma R. \quad (2)$$

To describe the rate of change of the population group I , the rate of increase in the infected population is already given above by the rate of infection, or decrease in the

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Susceptible population, βIS . However, it is also necessary to account for the individuals who are recovering, causing a decrease in the infected population. This decrease can be accounted for by multiplying a constant rate of recovery, which we will call ν , by the infected population I and subtracting this from the rate of increase, βIS . The equation for the rate of change of the Infected Population then becomes,

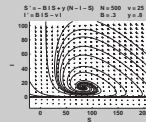
$$\frac{dI}{dt} = \beta IS - \nu I. \quad (3)$$

Finally, to describe the rate of change of the last group, the Recovered Population, a similar process is followed as for the first two. The rate of increase of our recovered population (conversely the decrease of our infective population) is accounted for by the product νI . Additionally, there is also a certain rate of loss due to the number of people who are losing their immunity. This decrease is given by the product γR . Therefore, the rate of change of the recovered population is given by the following differential equation

$$\frac{dR}{dt} = \nu I - \gamma R. \quad (4)$$

2. Population Dynamics

In a graph of the equations for these three different groups, we would expect three curves representing changing population levels. A great deal of the behavior of the population depends upon the initial conditions. Starting out with a population where there are initially only susceptible victims and a few infected hosts, a sudden decrease in the population S accompanied by a sudden increase in the population I would be expected.



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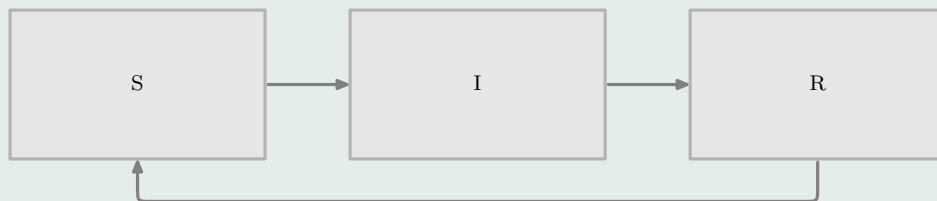
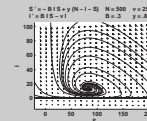


Figure 1: A diagram of the flow between the susceptible, infected, and recovered populations.

But as population I increases, there should also be a related rise of population R , as individuals begin to recover. This will result in some balancing or leveling out of the population I . Depending on the constants we choose for the rates of infection, recovery, and immunity loss, the results will differ greatly. To graph an example of how these three groups, S , I , and R behave in relation to one another within a total population N , we use the program MatLab to graph our set of three differential equations.

First, we will rename each of our functions S' , I' and R' as u_1 , u_2 and u_3 . Therefore, if

$$\begin{aligned} S' &= u_1, \\ I' &= u_2, \\ R' &= u_3, \end{aligned}$$



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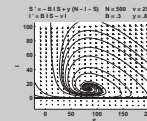
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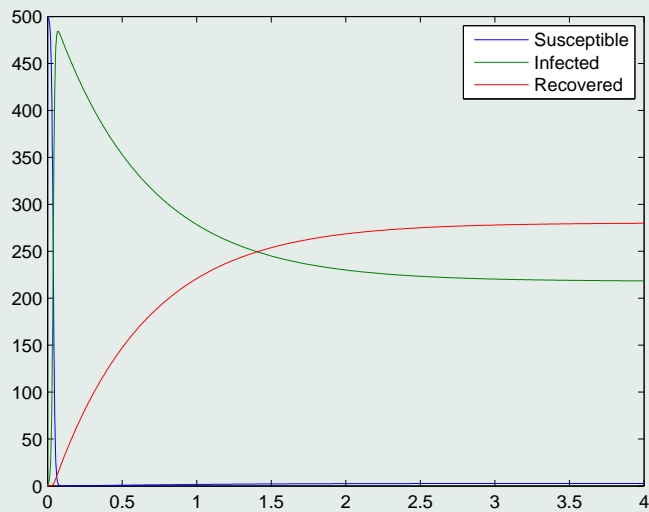


Figure 2: The plot of S' , I' , and R' .

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then,

$$S = u_1,$$

$$I = u_2,$$

$$R = u_3.$$

Making the appropriate substitutions, the three equations for dS/dt , dI/dt , and dR/dt become

$$u_1' = -\beta u_1 u_2 + \gamma u_3,$$

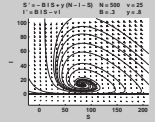
$$u_2' = \beta u_1 u_2 - \nu u_2,$$

$$u_3' = \nu u_2 - \gamma u_3.$$

Then, we can write an function m-file as follows

```
function [uprime]=sirs(t,u,B,gamma,v);
uprime=zeros(3,1);
uprime(1)=-B*u(1)*u(2)+gamma*u(3);
uprime(2)=B*u(1)*u(2)-v*u(2);
uprime(3)=v*u(2)-gamma*u(3);
```

Saving this function as sirs.m, next we write an m-file to graph our equations using ode45. To do so, we must provide Matlab with the initial conditions of our equations as well as values for the rate constants for infection (β), recovery (v), and immunity loss (γ). For a hypothetical example let's set



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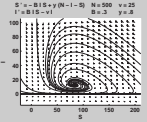
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$$\beta = 0.33,$$

$$\nu = 0.9,$$

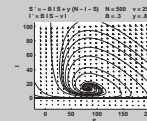
$$\gamma = 0.7.$$

Starting with an initial population of 500 susceptible individuals, 1 infected agent, and 0 recovered individuals, as time t runs from 0 to 4, we enter our values into an m-file as shown below

```
[t,u]=ode45(@sirs,[0,4],[500;1;0],[.33,.7,.9]);
plot(t,u)
Legend('Susceptible','Infected','Recovered')
shg
```

The result is the graph shown in Figure 1.

From this graph we see that we do indeed get a sudden decrease in the population S . It also appears that the susceptible population never reaches very high levels again. Considering the fact that the rate of removal from population S is given by the product of I and S as well as the rate constant β , this makes sense because this number will always be comparatively high. As for populations I and R , they both end up leveling off around $R = 280$ and $I = 220$. Adding these two together, we get 500, which is very close to our initial population, especially if we take into account the very small number of susceptible individuals still present. This simply illustrates the equation presented earlier where the entire population was said to be comprised of only three groups: susceptible, infected, and recovered individuals, or in other words



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$$N = S + I + R$$

3. Equilibrium Points of the SIRS Model

In summary, the three differential equations for the SIRS model are

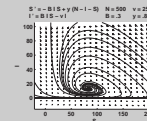
$$\begin{aligned}\frac{dS}{dt} &= -\beta IS + \gamma R, \\ \frac{dI}{dt} &= \beta IS - \nu I, \\ \frac{dR}{dt} &= \nu I - \gamma R.\end{aligned}$$

However, this system can be simplified from three to two equations. Recalling that $N = S + I + R$, a substitution of $N - S - I$ for R in the first equation can be made. Doing so results in the following planar system

$$\frac{dS}{dt} = -\beta IS + \gamma(N - S - I), \quad (5)$$

$$\frac{dI}{dt} = \beta IS - \nu I. \quad (6)$$

We can analyze this system of equations by finding its equilibrium points and using the Jacobian to determine the nature of those equilibrium points. Taking the partial derivatives of the two equations, the Jacobian can be written,



$$J = \begin{pmatrix} -\beta I - \gamma & -\beta S - \gamma \\ \beta I & \beta S - \nu \end{pmatrix}. \quad (7)$$

Secondly, to find the nullclines of the equations requires setting both equations equal to zero.

If dS/dt is equal to 0, S can be solved for as follows,

$$\begin{aligned} 0 &= -\beta IS + \gamma(N - S - I), \\ \beta IS &= \gamma(N - S - I), \end{aligned}$$

$$\beta IS + \gamma S = \gamma(N - I). \quad (8)$$

This final equation can be solved for either S or I .

Analyzing our second equation, dI/dt , and once again setting the equation equal to zero, we can determine that,

$$\begin{aligned} 0 &= \beta IS - \nu I, \\ 0 &= I(\beta S - \nu) \end{aligned}$$

Then

$$I = 0 \quad (9)$$

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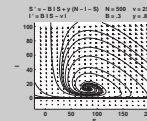
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or,

$$0 = \beta S - \nu$$

$$S = \frac{\nu}{\beta}. \quad (10)$$

This means there will be a nullcline at $I = 0$, $S = \nu/\beta$, and $\beta IS = \gamma(N - S - I)$.

$$N > \frac{\nu}{\beta}. \quad (11)$$

Using the Jacobian, assuming that variables β, ν , and γ are all positive and subbing in our equilibrium values, we can analyze the types of equilibrium points at each intersection of the nullclines. At the point where the nullcline $\beta IS = \gamma(N - I - S)$ crosses the nullcline $I = 0$, we can determine that

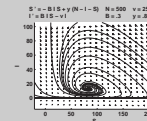
$$J = \begin{pmatrix} 0 - \gamma & -\beta S - \gamma \\ 0 & \beta S - \nu \end{pmatrix}.$$

in which case the determinant $D = -\gamma(\beta S - \nu)$, is negative. Therefore, there will be a saddle point at this equilibrium point.

At the point where $S = \nu/\beta$ and $\beta IS = \gamma(N - I - S)$, or $I = \frac{\gamma(N-S)}{\beta S + \gamma}$, cross, we see that

$$J = \begin{pmatrix} \frac{-\beta(\gamma(N-\nu/\beta))}{\beta\nu/\beta+\gamma} & \frac{-\beta\nu}{\beta} - \gamma \\ \frac{\beta(\gamma(N-\nu/\beta))}{\beta\nu/\beta+\gamma} & \frac{\beta\nu}{\beta} - \nu \end{pmatrix}.$$

Simplifying this matrix, the Jacobian becomes



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$$J = \begin{pmatrix} \frac{-\beta(\gamma(N-\nu/\beta))}{\frac{\nu+\gamma}{\beta(\gamma(N-\nu/\beta))}} & -\nu-\gamma \\ 0 & 0 \end{pmatrix}.$$

Assuming that $N > \nu/\beta$, which we will later discover is something known as the threshold effect, it is simple to observe that the trace T is equal to $\frac{-\beta(-\gamma(N-\nu/\beta))}{\nu+\gamma}$, which is a negative number. The determinant D can be calculated as follows

$$D = 0 - (-\nu - \gamma) \frac{\beta(\gamma(N - \nu/\beta))}{\nu + \gamma}$$

$$D = (\nu + \gamma) \frac{\beta(\gamma(N - \nu/\beta))}{\nu + \gamma}$$

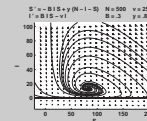
$$D = \beta(\gamma(N - \nu/\beta))$$

$$0 < \beta(\gamma(N - \nu/\beta)).$$

Once again, assuming that $N > \nu/\beta$ and all the constants are positive, the determinant D will also be positive. Therefore, the equilibrium point here will be some kind of sink.

To get an idea of how this would look, it would be helpful to graph this set of equations using **pplane7** in MatLab.

After entering the command **pplane7** into MatLab, we enter our two equations for S' and I' . We also choose values for the variables N, β, γ , and ν so that



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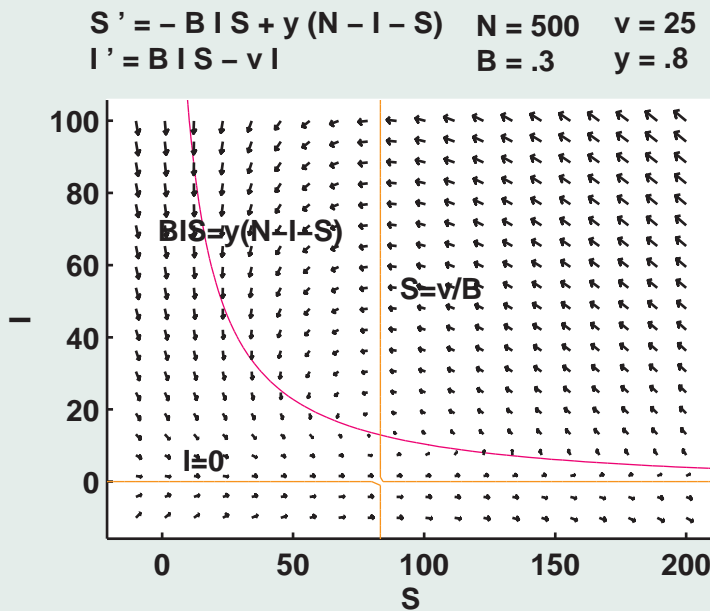
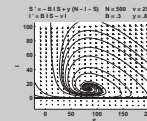


Figure 3: A plot of the nullclines for the SI system.



$$N = 500,$$

$$\beta = 0.3,$$

$$\gamma = 0.8,$$

$$\nu = 25.$$

According to the nullcline equations, there should be an equilibrium point where the lines $S = \frac{\nu}{\beta}$ and $I = \frac{\gamma(N-S)}{\beta S + \gamma}$ cross. Plugging in our values for N, β, γ and ν as chosen above, we get the following values for S and I .

$$S = \frac{25}{0.3}$$

$$S = 83.3333$$

$$I = \frac{0.8(500 - 83.3333)}{25 + 0.8}$$

$$I = 12.9199.$$

As shown in the graph in Figure 2, the point (83.3333, 12.9199) does appear to be an equilibrium point. Plugging these numbers into the Jacobian matrix, the matrix becomes

$$J = \begin{pmatrix} -0.3(12.9199) - 0.8 & -0.3(83.3333) - 0.8 \\ 0.3(12.9199) & 0.3(83.3333) - 25 \end{pmatrix}.$$

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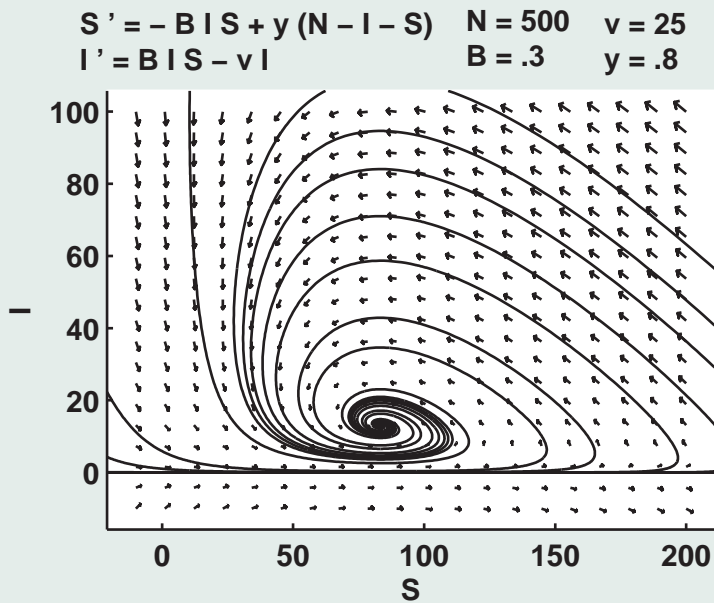
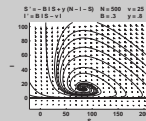


Figure 4: A phase plane plot of S and I with an equilibrium point at $(83.3333, 12.9199)$.

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Simplifying this gives the following matrix

$$J = \begin{pmatrix} -4.6760 & -25.8 \\ -3.8760 & 0 \end{pmatrix}.$$

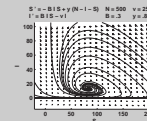
The trace T of J is -4.6760 and the determinant D is 100.0008 . Since the trace is negative but the determinant is positive, this point is clearly a sink. To determine what kind of sink, we need to determine where on the trace determinant plane the point $(-4.6760, 100.0008)$ lies. Since

$$\begin{aligned} T^2 - 4D &= (-4.6760)^2 - 4(100.0008), \\ (-4.6760)^2 - 4(100.0008) &= -21.8650 - 400.0032, \\ 0 &> -421.8682, \end{aligned}$$

the point $(-4.6760, 100.0008)$ lies above the line $T^2 - 4D$. This means that the equilibrium point is a spiral sink, as is further evidenced by its appearance in Figure 3.

According to the nullcline equations, there should also be a second equilibrium point where the lines $I = 0$ and $S = \frac{\gamma(N-I)}{\beta I + \gamma}$. Substituting the given value of I , we can solve the system for S .

$$\begin{aligned} S &= \frac{\gamma(N-I)}{\beta I + \gamma}, \\ S &= \frac{\gamma N}{\gamma}, \\ S &= N. \end{aligned}$$



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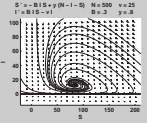
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This means that the lines $I = 0$ and $S = \frac{\gamma(N-I)}{\beta I + \gamma}$ intersect at the point $(500, 0)$. Analytically, this is an obvious answer. If we have a population where there are zero infected individuals, or $I = 0$, then the entire population N will be constituted of susceptible individuals S , meaning $S = N$.

To determine the nature of this equilibrium point, the next step is to plug in values for I and S into the Jacobian matrix for the planar system. If

$$S = 500,$$

$$I = 0,$$

then the Jacobian becomes

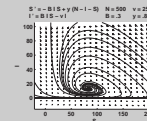
$$J = \begin{pmatrix} -0.8 & -150.8 \\ 0 & 125 \end{pmatrix}.$$

The trace of this Jacobian matrix is 124.2 and the determinant is -100. Since the determinant is negative, this equilibrium point shown in Figure 5 is a saddle point.

4. The Threshold Effect

Another important discovery in the study of the SIRS model is the existence of the threshold effect. The basis of this effect is that, unless the population is initially large enough, with considerable amount of contact between individuals and a high enough rate of infection, the virus will die out before becoming established in the population.

To explain this mathematically requires re-examining the equilibrium equations.



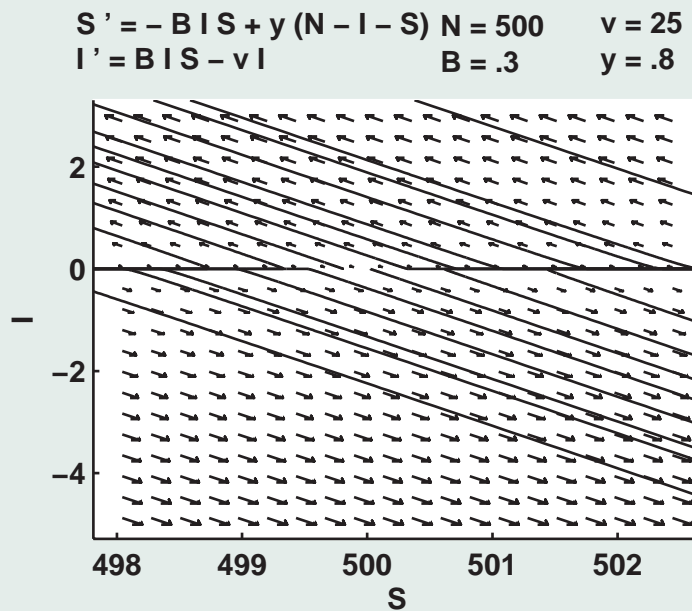
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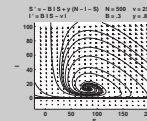
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Figure 5: A phase plane plot of I and S with an equilibrium point at $(500, 0)$.



$$\beta IS = \gamma(N - I - S).$$

Solving this for I we find that

$$\begin{aligned}\beta IS + \gamma I &= \gamma(N - S) \\ I(\beta S + \gamma) &= \gamma(N - S) \\ I &= \frac{\gamma(N - S)}{\beta S + \gamma}.\end{aligned}$$

Analyzing this equation, if S is greater than N , I is going to be negative. This means that, in order for the infection to be established, or become endemic in the population, N must be greater S .

However, according to another equilibrium equation.

$$S = \frac{\nu}{\beta}.$$

Therefore,

$$N > \frac{\nu}{\beta}.$$

Now, using the numerical example from earlier on, if N is set to equal a number less than the ratio ν/β , the graph should show a phase plane where, no matter what

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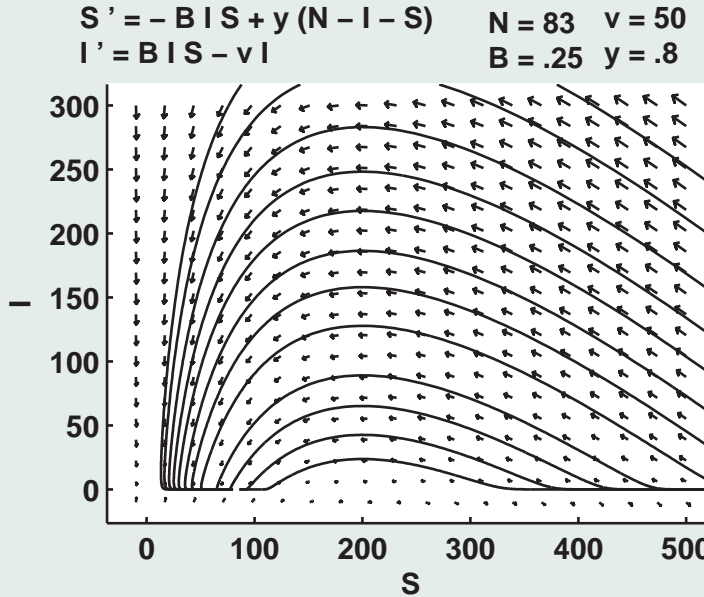
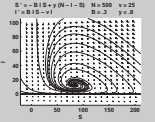


Figure 6: A phase plane plot of I and S where $N < v/\beta$.

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initial value is chosen, the infected population I will decrease to 0 and the virus will not be established.

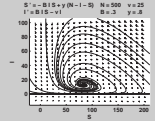
Using the same constant values as before, if $N = 83$, then $N < \frac{\nu}{\beta}$. As we see in Figure 6, this phase plane does indicate that the population I will eventually decrease to zero.

5. Conclusion

The SIRS system shown in this paper is a very simplistic model, and yet it illustrates many important aspects of a viral infection. By applying some basic analysis, it is easy to describe and observe possible scenarios by examining the equilibrium points. Also, these equations provide us with a correlation between population size and the chances of a disease becoming endemic by relating the entire population number to the ratio between the recovery and infectivity rate. As a method of approach, the SIRS model is an excellent tool to use in studying the spread of a viral disease.

References

- [1] Edelstein-Keshet, Leah *Mathematical Models in Biology*, McGraw-Hill, 1988, First Edition.
- [2] Murray, James D. *Mathematical Biology* Springer-Verlag Berlin Heidelberg New York, 1993, Second Corrected Edition.



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