Framework of Infective Susceptible Phase Plane Analysis of SIR Model

N Suresh Rao
Department of Computer
Centre/ Science and IT block
University of Jammu
Jammu - J & K state, India.
180006.

ABSTRACT
In the present article an attempt is made to understand the Infected-Susceptible phase plane trajectories, describing the growth of virus in the model of Susceptible, Infected, and Removed (SIR) extended to immigration studies. The growth of virus is described by second order differential equation, in terms of small deviations from the steady state solution of infection. Further the situation for discriminator = 0, obtained in solving the second order differential equation, is described. In this analysis the free parameter, defined in terms of immigrant rate, birth and death rates of virus, is shown to play an important role in the shape of the trajectories in I_S Phase plane. For same values of immigration rate, birth and death rates of virus, all the trajectories approach asymptotically the stable equilibrium point (ratio of death to birth rate of virus, ratio of constant immigration rate to death rate of virus), which is termed as a nodal sink. The effect of different parameters such as size of system of computers, death and birth rates of virus and threshold value of the epidemic on the growth of virus is presented.

General Terms
Immigration rate, Infected nodes, Computer Virus.

Keywords
Immigration, SIR model, I-S phase plane, Virus growth, birth and death rates, 2nd order Differential Equations.

1. INTRODUCTION
The concept of immigrants, inflow of population, in demographic studies plays an important role in understanding the social-economic-environmental implications. Similarly in the field of epidemics, continuous transmission of biological virus leads to diseases. In the field of computers also the inflow of computer virus into an existing system of healthy computers plays an important role in the growth and spread of virus. A strong and close analogy exists between computer virus and biological virus as can be seen in Wikipedia-the free encyclopedia [1]. Well established mathematical models to describe the growth and spread of virus exist in literature to understand the discipline of epidemiology. A brief survey, relevant to the present investigation is given below. The subject involves modeling the dynamics of an infectious disease in a population in which it occurs. The size of the population is considered to be constant. It is also assumed that the population is homogeneous. All members of the population interact with one another to the same degree. At each time the population/computers will consist of three categories; the susceptible class, those who may catch the disease but currently are not infected, the infected class, those who are infected with the disease and are currently contagious and the removed class, those who cannot get the disease, because they either have recovered permanently, are naturally immune, or have died. As time advances the system will attain a steady state which is of interest for understanding the growth and spread of virus. Important parameters are birth/infection rate, death/cure rate, threshold/effective removal value, reproduction number, delay and vigilance times etc. The popular models are Susceptible-Infected (SI), Susceptible-Infected-Susceptible (SIS), SIR, extended SIR namely Susceptible-Antidote-Infected-Removed (SAIR), Susceptible-Exposed-Infected-removed(SEIR), susceptible-exposed-antidote-infected-Removed (SEAIR) etc. The details of these models can be seen in literature [2, 3]. Particularly the model SIR concerning disease dynamics with and without demographics is described in [4]. In SEIR the susceptible and infected category are divided into compartments. The size and severity of the epidemic and controlling the disease by quarantine and vaccination are discussed in [5]. In a pure SIR model intensity of the epidemic and conditions under which an epidemic builds up and fades out are studied in terms of effective removal rate and the popular K-M theorem [6]. Further another interesting piece of information is found in lecture notes on "Mathematical Biology" [7] where SIR model, conditions for eradicating the disease by vaccination and for a disease to become endemic, are described. The SEAIR model described Influenza-asymptomatic infection with pre-seasonal and antiviral treatment, age-structure, and delayed vaccination [8]. Similarly Malaria, a devastating disease leading to severe deaths in tropical regions of the world, and its eradication by applying direct control measures rather than vaccination is found in [9]. In almost all these studies the stability of disease-free and endemic equilibrium states of the disease are described with reference to the basic reproduction number, R_0.

Immigrants play a critical role in disease dynamics. The influence of immigration, (i) in removing the stresses in low fertility in demographic and welfare systems,(ii) the way the profiles, in the receiving population, are altered, (iii) how it features the reproduction and force of infection and finally how such studies helped in controlling a disease Rubella in Italy are described in [10]. Another interesting study is epidemic models with infective immigrants and vaccination [11, 12]. HIV transmission leading to AIDS is discussed in [13] where a constant immigration number is considered in the standard non-linear differential equations of SIR model. The equations are made linear by Jacobian transformation. The corresponding matrix is diagonal zed and the Eigen values are determined. A parametric condition for the stability of the state at equilibrium is obtained.

Similarly a simple and elegant method is used in solving the non linear equations of the SIR model with a constant number

for immigration of susceptibles in the differential equation for $\frac{dS}{dt}$. The values of S and I are expressed in terms of small deviations in the steady state values of S and I. With simple mathematics a second order differential equation (II O.D.E) for the virus growth is obtained. On solving this one comes across an expression for the discriminator, which may be +ve, zero, -ve. This will result in (i) real and distinct (ii) real and equal (iii) complex and unequal roots for the solutions. Out of these (i) and (iii) are discussed already [14, 15].

The remaining solution where the roots are real and equal is presented in this publication. The subject matter is arranged as follows. In section 2 the methodology is described. In section 3 the results and discussions are presented where as in section 4, the conclusions are high lighted. Finally, references are given in section 5.

2. METHODOLOGY

The basic SIR model involves three classes of computer systems namely, Susceptibles(S), Infectives (I) and Removed(R). By introducing some susceptibles (immigrants) at a constant rate (k) into the system, the effect of immigrants on the spread and growth of virus is described by the following three non-linear differential equations.

$$\frac{dS}{dt} = k - \beta SI \; ; \tag{1}$$

$$\frac{dI}{dt} = \beta SI - \gamma I \; ; \tag{2}$$

$$\frac{dR}{dt} = \gamma I \tag{3}$$

With the initial condition,

$$S + I + R = S_0 + I_0 + kt = N + kt \tag{4}$$

Where, k, β and γ stand for constant immigrant rate, birth rate and death rate respectively. S_0, I_0 are initial values of S and I. N stands for population size.

All the three derivatives in (1), (2) and (3) namely $\frac{dS}{dt}, \frac{dI}{dt}$ and $\frac{dR}{dt}$ cannot vanish simultaneously. As the interest in this article is confined to I_S phase plane it is sufficient to deal with (1) and (2). The equilibrium (steady state) solutions for S_E, I_E can be obtained by setting left hand side of (1) and (2) equal to zero and are given by

$$S_E = \frac{\gamma}{\beta}, I_E = \frac{k}{\gamma} \tag{5}$$

The variation of S and I is described by introducing small deviations, ε and υ, from equilibrium values S_E and I_E respectively. Thus

$$\left.\begin{aligned} S &= S_E(1+\varepsilon) \\ I &= I_E(1+\upsilon) \end{aligned}\right\} \tag{6}$$

Squares, higher powers and product terms of ε and υ are neglected. On substituting (5) and (6) in (1) and (2) and simplifying one gets

$$\frac{\gamma}{\beta k} * \frac{d\varepsilon}{dt} = -(\varepsilon + \upsilon); \frac{1}{\gamma}\frac{d\upsilon}{dt} = \varepsilon \tag{7}$$

Eliminating ε one gets the II O.D.E in υ as

$$\upsilon'' + (k\beta/\gamma)\upsilon' + (k\beta)\upsilon = 0 \tag{8}$$

This can be solved by standard method. In doing so one comes across a discriminator

$$\omega = \sqrt{1/4 * (k\beta/\gamma)^2 - k\beta)} \tag{9}$$

The value of ω (9) may be +ve, zero,-ve according to the term $(k\beta/\gamma^2) >, =, < 4$ respectively. Accordingly the roots are (i) real and distinct, (ii) real and equal, (iii) complex and unequal. In this paper we are confined to roots which are real and equal. The details are given below. The roots are equal when ω=0 i.e.,

$$m_1 = m_2 = (-1/2) * \kappa\beta/\gamma$$

Where, m_1 and m_2 are the two roots of (8). The solution is given by $\upsilon = (At + B)e^{((-1/2)k\beta/\gamma)}$ where; A, B are arbitrary constants, which can be determined from the initial values of υ and υ' at t = 0. For this, the expressions $\frac{dI}{dt}$, I_E, I from (2), (5) and (6) respectively are used. Thus υ is determined and hence 'I' as a function of time is obtained. Further from the relations for ε and υ from (7) and expressions for S_E and S from (5), (6), the value of S can be obtained as a function of time. Thus one can plot I vs S, S vs t, and I vs t for several combinations of k, β and γ. One must remember to keep $k\beta/\gamma^2 = 4$ so that ω is equal to zero. With a given initial number of infected nodes (I_0) for different population sizes, I_S phase-plane trajectories can be drawn. It may be noted that the initial values of I and S should not be taken as I_E and S_E which are points of singularity.

3. RESULTS AND DISCUSSIONS

In the present investigation the main interest is focused on the I_S phase plane analysis. Thus out of the three coupled differential equations used, the first two are sufficient for discussion. Unlike SIR model, the $\frac{dS}{dt}$ equation is not a continuously decreasing function. It has no lower bound limit. On the contrary it has i) a term, $-\beta SI$ representing the number of susceptibles getting converted into infectives and ii) a term, k, representing an inflow of immigrants at a constant rate. So depending on the values of S, I, β and k, there will be a competition between the said two terms. As a result $\frac{dS}{dt}$ will be increasing / decreasing. In the second equation, $\frac{dI}{dt}$ will be +ve, 0, -ve according to $S >, =, < \gamma/\beta$. The value of γ/β is termed as threshold value or effective

removal rate (ρ). Thus increase/decrease of I with time depends on whether $S > \rho$ or $S < \rho$. It may be noted that at equilibrium point the two differential equations, $\frac{dS}{dt}$ and $\frac{dI}{dt}$ will be zero. The relevant mathematical background is described in Sec 2. Different values of parameters, N, γ, β, K and I_0 of the virus are chosen so that the conditions given in Sec 2 are satisfied. Table-1 displays data for two typical sets (A and B) i) for high γ(0.02),β(0.04)values and low ρ(5) and ii) low γ(0.05),β(0.0025)and high ρ(20), out of several sets tried exhaustively.

TABLE 1. Dataset Descriptions

Set	Phase	Population Size (N)	γ	β	k	I_0
A	I	51,81,91	0.2	0.04	4	1
	II	101,151,301	0.2	0.04	4	1
	III	501,751,1001	0.2	0.04	4	1
B	I	101,501,1001,1501	0.05	0.0025	4	1
	II	2001,3001,4001	0.05	0.0025	4	1
	III	5001,7501,10,001	0.05	0.0025	4	1

For population size, N is varied from 51 to 10,001. The analysis is made concerning variation of I vs S; S vs t and I vs t through numerical simulations. By critically going through the computed results, time taken to reach the equilibrium values S_E and I_E are noted. While studying, I vs S trajectories, some distinct features are noted irrespective of γ and β values. Accordingly, they are classified into three different phases.

Phase I: In this phase the values of S increase from S_0 up to a maximum value. The trajectory takes a reversal i.e. S decreases and finally reaches the value S_E asymptotically. The value of S is > S_E. Thus I will be increasing from I_0 to I_E asymptotically.

Phase II: In this case S will be decreasing right from the beginning i.e. S_0 and asymptotically attains the value S_E .Here again the value of S is > S_E. Thus I will be increasing from I_0 to I_E asymptotically.

Phase III: In this phase also S will be decreasing from S_0, passes through S_E and enters –ve region for a while and then with a reversal enters +ve region and finally attains S_E and continues to remain there for all the times. It may be noted that the value of S, till it passes the ordinate at S_E, will be > S_E and later on the value will be < S_E till it sinks to the value S_E. For this region S will be < ρ. Thus I will increase to a maximum value and then will decrease exponentially and asymptotically attains the equilibrium value I_E. This feature is in accordance with the differential equation (2).

For clarity in resolution, the I vs S trajectories are shown separately for the three phases I, II and III as explained above in Fig. 1 series for set A. The variations of S vs t, for one value from each of the phases I and II and for two values of N from phase III, are shown in Fig. 2 series. Variation of I vs t is shown in Fig. 3 representing some typical values of N from Phases I, II and III. The features exhibited in all the said figures are in accordance with the prescription of the model. From the numerical values obtained for variation of I and S with time, it is noted that the time taken to reach the equilibrium values I_E and S_E is close to 26 and 32 unit time steps respectively. Further the infectives reach the equilibrium value earlier compared to that of susceptibles.

In order to study the effect of low γ and β with higher ρ the data as shown for set B is chosen. The value of ρ is enhanced by a factor of 4 which can be easily verified from the data in Table-I. The corresponding I-S trajectories for set B are shown in Fig.4 series. The variation of S vs t is shown in Fig. 5 series and the variation of I vs t is shown in Fig. 6. All the features as seen in Fig. 1, 2 and 3 are also seen in Fig. 4, 5 and 6. However it may be noted that the corresponding population sizes at the transition of phases are increased e.g for set A, the population size is $\approx$91 for phase I; for the phase II the range of N is from 101 to 301. Where as, for the phase III, the value of N is ≥ 501. Similarly for set B the corresponding values for N are < 1501 in phase 1 and for phase II the range of N is from 2001 to 4001 and for Phase III, N is ≥ 5001. Further it is also noted that the time taken to reach the equilibrium values I_E and S_E (116 and 155 unit time steps) are increased by a factor $\approx$ 4 and I approaches I_E faster than S approaches S_E, as is the case of set A. The results clearly show that when ρ is increased by a factor of 4 the corresponding saturation time for infectives and susceptibles increase by almost the same factor. This means higher the value of ρ, slower is the rate of growth of virus.

All the above observations made in this investigation are with discriminator ω=0, in solving equation (8), i.e the roots of the quadratic equation are real and equal. This is a particular case of ω when it is a +ve quantity. The mathematical expressions for the solutions in both the cases are different but in principle mathematically they belong to same category i.e the behavior of solutions for roots "real and distinct" are identical to roots "real and equal". This consistency is easily seen in this investigation when compared with that of [15]. Thus all predictions of the model are well described in Figs.1-6 for the two sets of data (A and B), i) for higher γ, β and lower ρ and ii) lower γ, β and higher ρ.

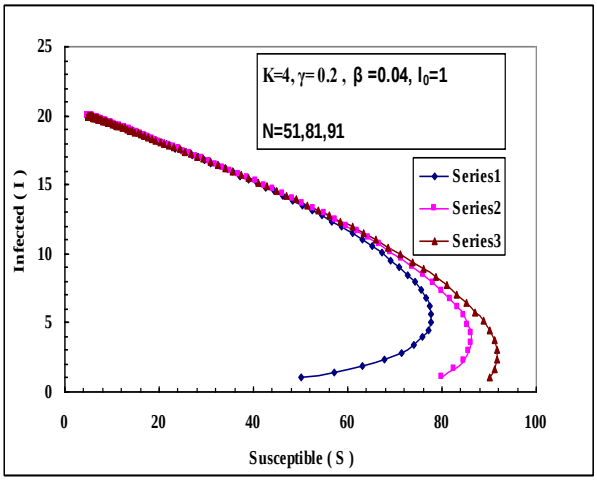

Fig 1(a): Susceptible vs Infected Nodes

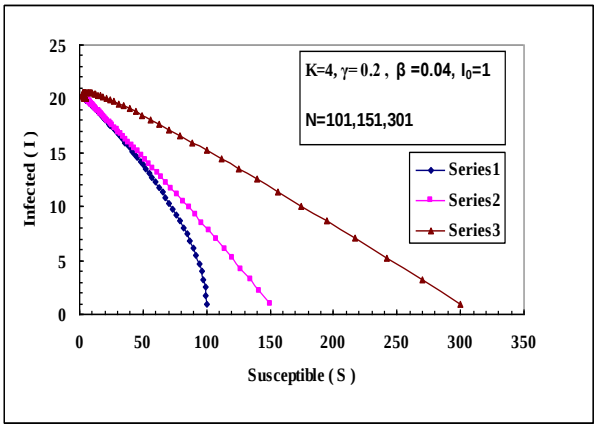

Fig 1(b): Susceptible vs Infected Nodes

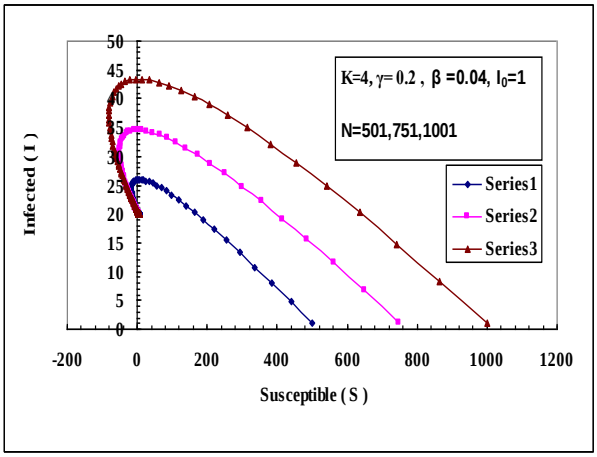

Fig 1(c): Susceptible vs Infected Nodes

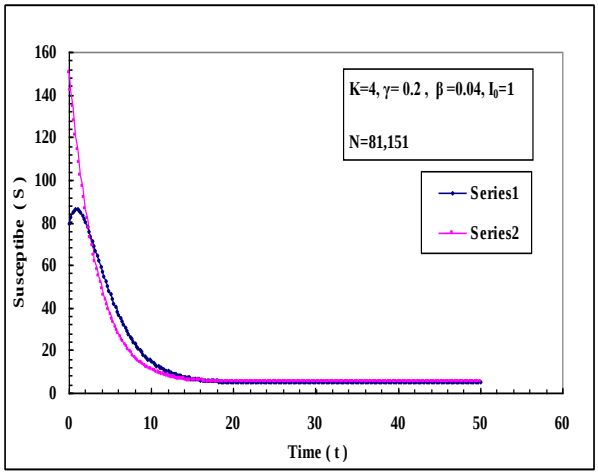

Fig 2(a): Susceptible Nodes vs Time

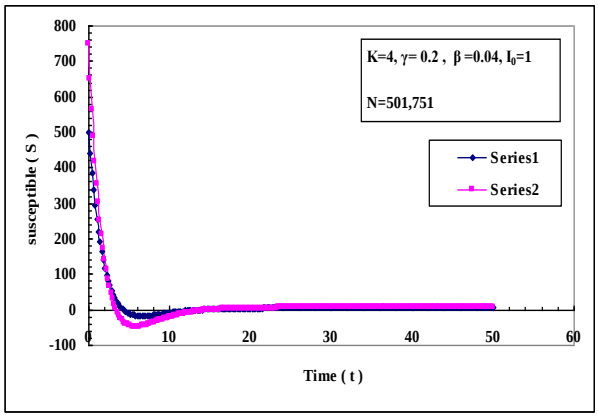

Fig 2(b): Susceptible Nodes vs Time

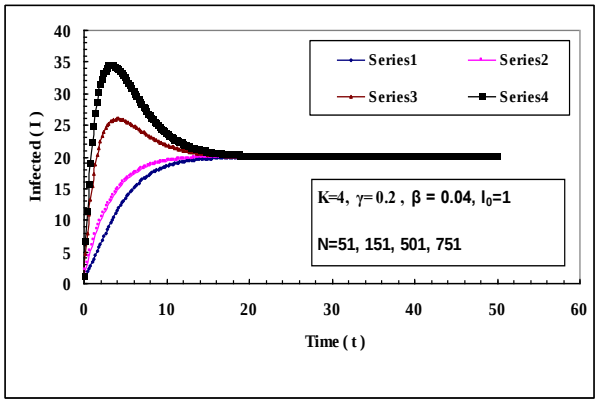

Fig 3: Infected Nodes vs Time

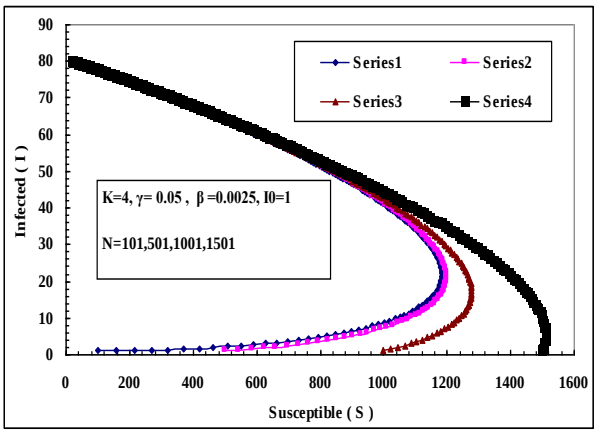

Fig 4(a): Susceptible vs Infected Nodes

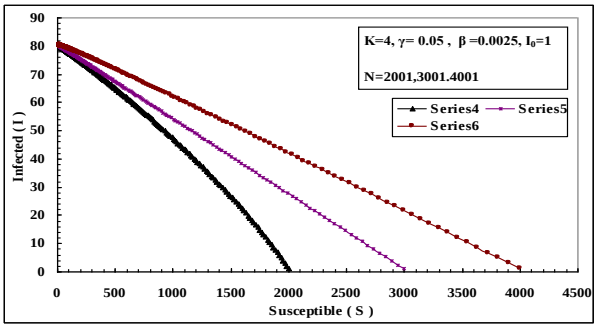

Fig 4(b): Susceptible vs Infected Nodes

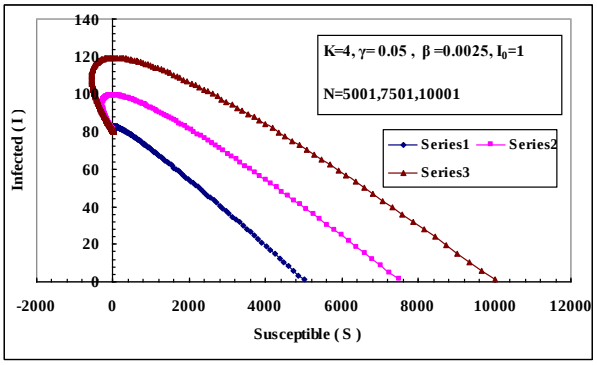

Fig 4(c): Susceptible vs Infected Nodes

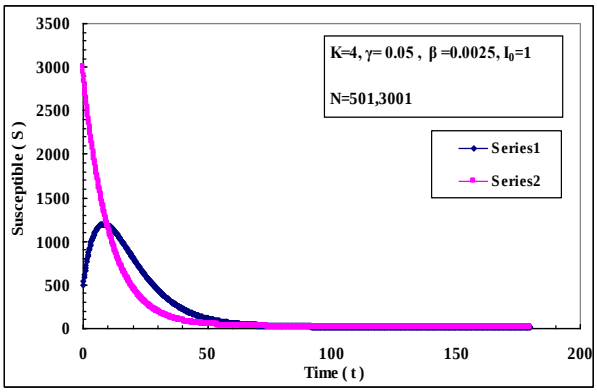

Fig 5(a) : Susceptible Nodes vs Time

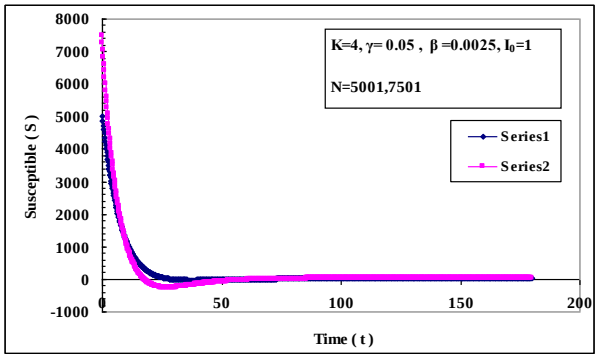

Fig 5(b) : Susceptible Nodes vs Time

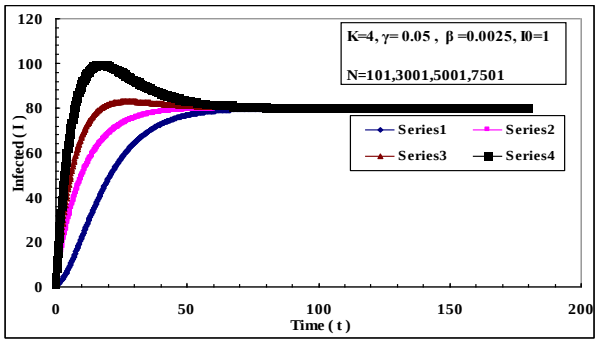

Fig 6: Infected Nodes vs Time

4. CONCULSIONS

In this investigation it is observed that the term $k\beta/\gamma^2$ plays an important role in describing the nature of infection growth. Unlike SIR model there is no lower bound on S but it attains a stable equilibrium vale, S_E=ρ which is the threshold. The increasing /decreasing trend of S is noted to depend on the relative strengths of the terms, $-\beta SI$ and k. The value of I is not approaching zero but attaining a stable equilibrium value $I_E = k/\gamma$. Variation of I is found to depend on whether S>ρ or S<ρ. For same values of k, γ and β, all trajectories in I_S phase plane are noted to reach the stable equilibrium point at (S_E, I_E) which is termed as nodal sink. It is observed that as the threshold value is increased, the time taken to reach equilibrium point (S_E, I_E) also increases indicating there by a slow rate for growth of virus. The general trend in the behavior of I-S phase plane trajectories is consistent in both the situations when the roots are i) real and distinct and ii) real and equal.

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