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DYNAMICS OF AN IMPROVED SIS EPIDEMIC MODEL

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ABSTRACT. A new modification of the SIS epidemic model incorporating the adaptive host behavior is proposed. Unlike the common situation in most epidemic models, this system has two disease-free equilibrium points, and we were able to prove that as the basic reproduction number approaches the threshold of 1, these two points merge and a Bogdanov-Takens bifurcation of codimension three occurs. The occurrence of this bifurcation is a sign of the complexity of the dynamics of the system near the value 1 of basic reproduction number. Both local and global stability of disease-free and endemic equilibrium point are studied.

1. INTRODUCTION

The use of mathematical models in the analysis of changes in infectious diseases dates back to Daniel Bernoulli in 1766. After Bernoulli, other efforts were made in this field. The first basic modeling and analysis in this field was done by Kermack and Mckendrick in 1927. After a break from 1980 onwards, many researchers paid attention to mathematical epidemiology and many articles were published on various diseases such as AIDS, tuberculosis, influenza. Researchers have also modeled and analyzed various effects such as vaccination, quarantine, the existence of multiple strains, and so on, see [5, 13, 19] and references there in.

In modeling infectious disease, the population is divided into different classes. For example in the SIR model, proposed by Kermack and Mckendrick, the population is divided into three categories: susceptible individuals, infected individuals and recovered individuals. In diseases in which patients do not gain any immunity after recovery and return to the class of susceptible individuals, the disease is said to follow the SIS model. Diseases such as tuberculosis, meningitis and gonorrhea follow

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this pattern, see [5, 11, 13]. The original SIS model and a number of its modifications are converted to the logistic differential equation, which has a simple dynamic, see [19]. Improved forms of this model have been studied by various researchers, see [1, 12, 22, 24, 25].

One of the basic assumptions in most epidemic models is that people are passive, that is, they do not change their behavior during an outbreak of an infectious disease. D'Onfrio and Manfredi in [8] showed that realistic models should include feedback on information about the prevalence of the disease. For the review of the studies on the effect of behavior change in epidemic models, see [3, 7, 9, 14, 20, 21, 27].

Fear of getting the disease and, by its nature, taking cautious behaviors during the spread of infectious diseases is one of the effective factors in preventing the outbreak of the epidemic. We improve the SIS model by considering the effect of fear and caution in preventing disease and by adding a compartment for cautious people. Unlike the classic SIS model, which has relatively simple dynamics, this model has complex dynamics.

We organized the paper as follows. In Sec. 2, we present the model, the basic reproduction number and study the local and global stability of DFEs. Furthermore, we prove that under certain conditions two DFE points merge, and the model undergoes a degenerate Bogdanov-Takens bifurcation of codimension three and the system has an elliptic sector. In Sec. 3, we study the existence of the endemic equilibrium point and its local and global stability.

2. Model Formulation and Basic Properties

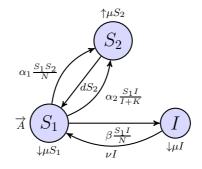


Figure 1. The flowchart of the model

Notation	Description of Notation
S_1	Susceptible individuals
S_2	Cautious individuals
Ι	Infectious individuals
μ	Natural death rate
u	Recovery rate
A	Recruitment rate
α_1	Awareness rate
α_2	Fear rate
β	Infection rate
d	The percentage of people who discard or lose their awareness

 Table 1. Description of Notations

Our model has the following compartments: S_1 are the susceptible individuals, S_2 are the cautious individuals, that is, people who take precautionary measures to prevent them from contracting the disease, and I are the infected and infectious individuals. Susceptible individuals become infected at rate β . Individuals by learning from family, friends, media, social networks, and also concerning psychological factors go from S_1 to S_2 . We consider two paths to transfer from S_1 to S_2 : (I) Due to contact with cautious people and encouragement to engage in such behaviors. We consider the coefficient α_1 i.e., awareness rate, as the likelihood of transformation of a susceptible individual to a cautious individual through contact with such a person and the effective transfer of information and the standard incidence term $\alpha_1 \frac{S_1 S_2}{N}$ for the flow of individuals from S_1 to S_2 , in which N is the total population. (II) Due to the observation of infected individuals and fear of the destructive consequences of the disease. We let $S_1F(I)$ be the type of flow from S_1 to S_2 , in which F(I) is called the force of fear. A good candidate for the force of fear is a Michaelis-Menten type function of the form $\alpha_2 \frac{I}{K+I}$, in which α_2 is the fear rate, i.e., the likelihood of tending to healthy behaviors through observing the adverse effects of the disease. The value of the constant K is equal to the number of infected individuals at which the force of fear is half of α_2 . At low population of infected individuals in the environment $I \ll K$, it varies linearly, $F \approx \alpha_2 \frac{I}{K}$. However at higher population of infected individuals with $I \gg K$ the force of infection becomes independent of Iand asymptotically approaches its maximum value α_2 . We take d the percentage of people who discard or lose their awareness and go to the susceptible compartment. And A, ν , and μ represent the recruitment rate of the population, the recovery rate, and the natural death rate.

By considering the flowchart of the model given in Figure 1, we have the following system of equations:

$$(2.1) \qquad \begin{cases} \frac{dS_1}{dt} = A - \alpha_1 S_1(\frac{S_2}{N}) - \alpha_2 S_1(\frac{I}{I+K}) + dS_2 - \mu S_1 + \nu I - \beta S_1(\frac{I}{N}) \\ \frac{dS_2}{dt} = \alpha_1 S_1(\frac{S_2}{N}) - dS_2 + \alpha_2 S_1(\frac{I}{I+K}) - \mu S_2 \\ \frac{dI}{dt} = \beta S_1(\frac{I}{N}) - \nu I - \mu I \end{cases}$$

with the initial conditions $S_1(0), S_2(0), I(0)$ that all are positive. All parameters in the above system are nonnegative. The total population $N(t) = S_1(t) + S_2(t) + I(t)$ satisfies the equation $\dot{N} = A - \mu N(t)$, so $\lim_{t\to\infty} N(t) = \frac{A}{\mu} = N_0$. Following [17, 18], we study the behavior of our system on the plane $S_1 + S_2 + I = N_0 = \frac{A}{\mu}$. Let $s_1 = \frac{S_1}{N_0}, s_2 = \frac{S_2}{N_0}, i = \frac{I}{N_0}$, be the suceptible, cautious and infectious fractions respectively. Therefore system (2.1) transforms to the following system:

(2.2)
$$\begin{cases} \frac{ds_1}{dt} = \mu - \alpha_1 s_1 s_2 - \alpha_2 s_1 (\frac{i}{i+k}) + ds_2 - \mu s_1 + \nu i - \beta s_1 i \\ \frac{ds_2}{dt} = \alpha_1 s_1 s_2 - ds_2 + \alpha_2 s_1 (\frac{i}{i+k}) - \mu s_2 \\ \frac{di}{dt} = \beta s_1 i - \nu i - \mu i, \end{cases}$$

where $k = \frac{K}{N_0}$. Since $s_2 = 1 - s_1 - i$, the above system transforms to the, (2.3) $\begin{pmatrix} ds_1 & i \\ ds_1 & i \end{pmatrix} = c_1 c_1 \begin{pmatrix} i \\ i \end{pmatrix} + d(1 - c_1 - i) = c_1 + i \end{pmatrix}$

$$\begin{cases} \frac{ds_1}{dt} = \mu - \alpha_1 s_1 (1 - s_1 - i) - \alpha_2 s_1 (\frac{s_1}{i + k}) + d(1 - s_1 - i) - \mu s_1 + \nu i - \beta s_1 i \\ \frac{di}{dt} = \beta s_1 i - \nu i - \mu i \end{cases}$$

We study (2.1) and naturally (2.2) in set $(\mathbb{R}^+)^3$, so it is natural to study (2.3) in the following positively invariant set:

$$\Omega = \{ (s_1, i) : s_1, i \ge 0, s_1 + i \le 1 \}.$$

2.1. Disease-free equilibriums The system (2.3) has two disease-free equilibriums (DFEs), $E_0 = (1,0)$ and for $\alpha_1 > d + \mu$, $E_1 = (\frac{d+\mu}{\alpha_1}, 0)$. The point E_0 is called the trivial DFE and E_1 the nontrivial one. The Jacobian matrix of (2.3) has the following form

$$\begin{pmatrix} J(s_1, i) = \\ \begin{pmatrix} -\alpha_1 + 2s_1\alpha_1 + \alpha_1 i - \frac{\alpha_2 i}{i+k} - d - \mu - \beta i & s_1(\alpha_1 - \frac{\alpha_2 k}{(k+i)^2}) - d + \nu - \beta s_1 \\ \beta i & \beta s_1 - \nu - \mu \end{pmatrix}$$

Now the Jacobian for the trivial DFE is of the following form

(2.4)
$$J_0 = J(E_0) = \begin{pmatrix} \alpha_1 - d - \mu & \alpha_1 - \frac{\alpha_2}{k} - d + \nu - \beta \\ 0 & \beta - \nu - \mu \end{pmatrix}$$

and its eigenvalues are

$$\lambda_1 = \alpha_1 - d - \mu, \ \lambda_2 = \beta - \nu - \mu$$

and $p = Tr(J(1,0)) = (\alpha_1 - d - \mu) + (\beta - \nu - \mu), q = DetJ(1,0) = (\alpha_1 - d - \mu)(\beta - \nu - \mu).$

If $\alpha_1 - d - \mu < 0$, and $\beta - \nu - \mu < 0$, then p < 0 and q > 0. Therefore, according to the linearization theorem, the trivial DFE is a stable node.

For the non-trivial DFE, the Jacobian has the form

$$J_1 = J(E_1) = \begin{pmatrix} \mu + d - \alpha_1 & (\alpha_1 - \frac{\alpha_2}{k} - \beta)(\frac{d + \mu}{\alpha_1}) - d + \nu \\ 0 & \beta(\frac{d + \mu}{\alpha_1}) - \nu - \mu \end{pmatrix}$$

with

$$p = Tr(J(E_1)) = (d + \mu - \alpha_1) + (\beta(\frac{d + \mu}{\alpha_1})) - \nu - \mu),$$
$$q = Det(J(E_1)) = (d + \mu - \alpha_1)(\beta(\frac{d + \mu}{\alpha_1})) - \nu - \mu).$$

Now if $d + \mu - \alpha_1 < 0$ and $\beta(\frac{d + \mu}{\alpha_1}) - \nu - \mu < 0$, then the non-trivial DFE is a stable node. The above discussions lead to the following theorem.

Theorem 2.1. 1. The disease-free equilibrium E_0 of (2.3) is a stable hyperbolic node if $\alpha_1 < d + \mu$, and $\beta < \nu + \mu$.

2. The disease-free equilibrium E_0 is a hyperbolic saddle point if $\alpha_1 > d + \mu$ and $\beta < \nu + \mu$, or $\alpha_1 < d + \mu$ and $\beta > \nu + \mu$.

3. The disease-free equilibrium E_1 of (2.3) is a stable hyperbolic node if $d + \mu < \alpha_1$ and $\beta(\frac{d+\mu}{\alpha_1}) < \nu + \mu$.

4. The disease-free equilibrium E_1 is a hyperbolic saddle point if $\beta(\frac{d+\mu}{\alpha_1}) > \nu + \mu$.

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2.2. Basic reproduction number To derive the basic reproduction number R_0 , we use the next generation matrix approach introduced in [26], we use notations as in [19], section 5.3. The systems (2.2) and (2.3) are equivalent and we apply next generation method to (2.3). We divide the compartments into two categories; infected compartments $x = (s_2, i)$, and non infected compartments $y = s_1$. The corresponding matrices $\mathcal{F}(x, y)$ and $\mathcal{V}(x, y)$ have the following forms

$$\mathcal{F}(x,y) = \begin{pmatrix} \alpha_1 s_1 s_2 + \alpha_2 s_1(\frac{i}{i+k}) \\ \beta s_1 i \end{pmatrix}$$
$$\mathcal{V}(x,y) = \begin{pmatrix} (d+\mu)s_1 \\ (\nu+\mu)i \end{pmatrix}$$

Now we have,

$$F = \begin{pmatrix} \alpha_1 & \frac{\alpha_2}{k} \\ 0 & \beta \end{pmatrix}, V = \begin{pmatrix} d+\mu & 0 \\ 0 & \nu+\mu \end{pmatrix}.$$

Therefore, the basic reproduction number is

$$R_0 = \rho(FV^{-1}) = max\{R_0^1, R_0^2\}.$$

where $R_0^1 = \frac{\alpha_1}{d+\mu}$ and $R_0^2 = \frac{\beta}{\nu+\mu}$. By applying the results of section 2.1, we deduce the following proposition for the local stability of DFEs.

Proposition 2.1. In the system (2.3):

- 1. The trivial DFE is locally asymptotically stable if and only if $R_0^1 < 1$ and $R_0^2 < 1$.
- 2. The DFE is locally asymptotically stable if and only if $R_0^1 > 1$ and $R_0^2 < R_0^1$.

To determine whether the disease can invade the population, we study the global stability of the DFE equilibrium points.

Theorem 2.2. In the system (2.3), we have:

If R¹₀ < 1 and R²₀ < 1, then the trivial DFE is globally asymptotically stable.
 If R¹₀ > 1 and R²₀ < 1, then DFE is globally asymptotically stable.
 In both cases, the disease cannot invade the population.

Proof. From the second equation of (2.3), we have

$$i(t) = i(0)e^{\int_0^t (\beta s_1(\tau) - (\mu + \nu))d\tau} < i(0)e^{(\mu + \nu)(R_0^2 - 1)t}.$$

therefore $R_0^2 < 1$ implies $\lim_{t \to +\infty} i(t) = 0$, for arbitrary i(0). Using the limit system theory [6], we need to study the limit of the solutions of

$$\frac{ds_1}{dt} = (\mu + d) - (\mu + d)s_1 - \alpha_1 s_1 (1 - s_1).$$

Separation of variables shows

$$s_1(t) = \frac{ce^{((\mu+d)-\alpha_1)t} - (\mu+d)}{ce^{((\mu+d)-\alpha_1)t} - \alpha_1}$$

Now it is clear that if $R_0^1 < 1$, i.e., $\alpha_1 < \mu + d$, $\lim_{t \to +\infty} s_1(t) = 1$ and if $R_0^1 > 1$, $\lim_{t \to +\infty} s_1(t) = \frac{\mu + d}{\alpha_1}$.

Lemma 2.1. If $R_0^1 < 1$, $\beta < \alpha_1$, and $d \leq \nu$, then the trivial DFE is globally asymptotically stable.

Proof. We have $\beta - \nu - \mu < \alpha_1 - \nu - \mu < d - \nu \le 0$, hence $R_0^2 < 1$ and the above theorem implies the global stability of the trivial DFE.

In Figure 2, we present the phase portrait of the model in two sets of parameters, with $R_0^1 < 1, R_0^2 < 1$ in (a), and $R_0^1 > 1, R_0^2 < 1$ in (b).

2.3. Bogdanov-Takens bifurcation The equilibrium points with zero eigenvalues with algebraic multiplicity two, in their Jacobian matrix can appear in smooth autonomous systems of ordinary differential equations

(2.5)
$$\frac{dx}{dt} = f(x,\gamma), \quad x \in \mathbb{R}^n, \quad \gamma \in \mathbb{R}^m,$$

when $n \ge 2, m \ge 2$. For example, in the system (2.3), when $(R_0^1, R_0^2) = (1, 1)$, two DFE equilibrium points merge, and the equilibrium point (1, 0) has a zero eigenvalue with algebraic multiplicity two, with the Jordan block $\begin{pmatrix} 0 & 1 \\ 0 & 0 \end{pmatrix}$. Such an event is called a Bogdanov-Takens (BT) bifurcation, see [10]. Bogdanov and Takens in [4, 23] showed that the restriction of (2.5) to any center manifold at the critical parameter value can be transformed by smooth coordinate changes to the following form

(2.6)
$$\begin{cases} \frac{dw_0}{dt} = w_1 \\ \frac{dw_1}{dt} = \sum_{k \ge 2} (a_k w_0^k + b_k w_0^{k-1} w_1). \end{cases}$$

We prove the occurrence of degenerate BT bifurcation in (2.3).

Theorem 2.3. Degenerate BT bifurcation of codimension 3 occur in (2.3) at equilibrium point (1,0), when $(\beta, \alpha_1) = (\nu + \mu, d + \mu)$, i.e., $(R_0^1, R_0^2) = (1, 1)$.

Proof. The form of DFE points and their Jacobian matrix show that, when $(R_0^1, R_0^2) = (1, 1)$ two DFE equilibrium points merge with each other and have a zero eigenvalue with algebraic multiplicity two.

We compute a_2, b_2, a_3 and b_3 in our system. First, we use $x_1 = s_1 - 1, x_2 = i$, which transforms (1, 0) to (0, 0), and (2.3) to the following system

$$\begin{cases} 2.7) \\ \begin{cases} \frac{dx_1}{dt} = (d+\mu)(R_0^1 - 1)x_1 + ((d+\mu)(R_0^1 - 1) + (\mu+\nu)(1 - R_0^2) - \frac{\alpha_2}{k})x_2 + \\ +R_0^1(d+\mu)x_1^2 + (R_0^1(d+\mu) - (\mu+\nu)R_0^2 - \frac{\alpha_2}{k})x_1x_2 + \frac{\alpha_2}{k^2}x_2^2 - \frac{\alpha_2}{k^3}x_2^3 + \frac{\alpha_2}{k^2}x_1x_2^2 + \\ +O(4) \\ \frac{dx_2}{dt} = (\mu+\nu)(R_0^2 - 1)x_2 + (\mu+\nu)R_0^2x_1x_2, \end{cases}$$

where O(4) means the linear combinations of the terms of the form $x_1^i x_2^j$ with $i+j \ge 4$.

Now by letting $(R_0^1, R_0^2) = (1, 1)$, and using the transformation $y_1 = x_1, y_2 = -\frac{\alpha_2}{k}x_2$, the system is modified as follows

$$\begin{cases} (2.8) \\ \begin{cases} \frac{dy_1}{dt} = y_2 + (d+\mu)y_1^2 + (1 - \frac{k(d-\nu)}{\alpha_2})y_1y_2 + \frac{1}{\alpha_2}y_2^2 + \frac{1}{\alpha_2^2}y_2^3 + \frac{1}{\alpha_2}y_1y_2^2 + O(4) \\ \frac{dy_2}{dt} = (\mu+\nu)y_1y_2. \end{cases}$$

Finally by using formulas in appendix B of [15], we obtain the following coefficients:

$$a_{2} = 0, \quad b_{2} = 2(\mu + d) + \mu + \nu, \quad a_{3} = -(\mu + \nu)(d + \mu),$$

$$b_{3} = \frac{1}{2}(\mu + \nu)(1 - \frac{k(d - \nu)}{\alpha_{2}}).$$

Since $a_2 = 0$ and $b_2 a_3 \neq 0$, as it is mentioned in [2, 15], a degenerate BT bifurcation of codim 3 occurs and (2.6) can be transformed by smooth coordinate changes and time reparametrization to the form

(2.9)
$$\begin{cases} \frac{d\xi_0}{dt} = \xi_1 \\ \frac{d\xi_1}{dt} = a_3\xi_0^3 + b_2\xi_0\xi_1 + b_3'\xi_0^2\xi_1 + O(||(\xi_0,\xi_1)||^5), \end{cases}$$

where

$$b_{3}' = b_{3} - \frac{3b_{2}a_{4}}{5a_{3}} = (1 - \frac{k(d-\nu)}{\alpha_{2}})(\frac{2}{5}\nu - \frac{1}{5}\mu - \frac{3}{5}d).$$

Now since $a_3 < 0$ and $b_2^2 + 8a_3 = (2(\mu + d) - (\mu + \nu))^2 > 0$, the equilibrium point has an elliptic sector.

Studies show that if BT bifurcation occurs, a wide dynamic variation can occur around the bifurcation values. Near the BT bifurcation values, we observe four kinds of bifurcation including transcritical, saddle-node, Hopf and Homoclinic, and the character of the equilibrium point will show stable focus, stable limit cycle, Homoclinic loop, unstable focus, etc. See Fig. 8.8. in [16], page 324. Figure 3 shows the elliptic sector whose existence has been proved.

3. ENDEMIC EQUILIBRIUM POINT

In this section, we study the existence and the dynamical properties of the endemic equilibrium points, i.e., steady states of the model. Let us denote the endemic equilibrium point by (s_1^*, i^*) , $i^* \neq 0$ implies:

(3.10)
$$s_1^* = \frac{\nu + \mu}{\beta} = \frac{1}{R_0^2}.$$

The relation $s_1^* < 1$ implies that only if $R_0^2 > 1$, the endemic steady state exists. By substituting this value, with some simple manipulation we result the following equation:

$$\mu - \alpha_1(\frac{\nu + \mu}{\beta})(1 - (\frac{\nu + \mu}{\beta}) - i^*) - \alpha_2(\frac{\nu + \mu}{\beta})(\frac{i^*}{i^* + k}) + d(1 - (\frac{\nu + \mu}{\beta}) - i^*) - \mu(\frac{\nu + \mu}{\beta}) + \nu i^* - \beta(\frac{\nu + \mu}{\beta})i^* = 0.$$

Finally we have the following equation

(3.11)
$$F(i^*) = Ai^{*2} + Bi^* + C = 0$$

Where

$$A = \alpha_1(\nu + \mu) - \beta(\mu + d) = \alpha_1(\nu + \mu)(1 - R_0^*),$$

$$B = k((\nu + \mu)(\alpha_1 - \beta) + \beta(\nu - d)) + (\mu + d)(\beta - \nu - \mu) + (\nu + \mu)(-\alpha_1 - \frac{\alpha_1}{\beta}(\nu + \mu) - \alpha_2) = (\nu + \mu)(k\alpha_1(1 - R_0^*) + (\mu + d)(R_0^2 - R_0^1 - 1 - \frac{1}{R_0^*}) - \alpha_2),$$

$$C = k((\mu + d)(\beta - \nu - \mu) + (\nu + \mu)(-\alpha_1 - \frac{\alpha_1}{\beta}(\nu + \mu))) = k(\mu + d)(\nu + \mu)(R_0^2 - R_0^1 - 1 - \frac{1}{R_0^*}),$$

and $R^* = \frac{R_0^2}{2}$

and $R_0^* = \frac{R_0^2}{R_0^1}$.

we now study the existence and dynamical properties of the endemic steady states of the system. In the following cases, there exists an endemic equilibrium point. (1): A > 0, $R_0^2 > 1$, and $R_0^1 > 1$. In this case, $R_0^* < 1$, hence F(0) = C < 0. Now the convex parabola $F(i^*)$ has a unique positive root, and the system has the following endemic equilibrium point

$$E_2 = (s_1^*, i^*) = (\frac{\nu + \mu}{\beta}, \frac{-B + \sqrt{\Delta}}{2A})$$

2) A < 0, C > 0. In this case, $F(i^*)$ is a concave parabola with F(0) = C > 0. In this case, the system has the following endemic equilibrium point

$$E_2 = (s_1^*, i^*) = (\frac{\nu + \mu}{\beta}, \frac{-B - \sqrt{\Delta}}{2A}).$$

Now we study local stability of endemic equilibrium points.

Proposition 3.1. Let $R_0^* > 1$, $R_0^2 > 1$, and $\nu > d$, then the endemic steady state is locally asymptotically stable.

Proof. The Jacobian matrix of the system at the endemic equilibrium has the following form

$$J(s_{1}^{*}, i^{*}) = \begin{pmatrix} \alpha_{1}(2s_{1}^{*} - 1 + i^{*}) - \alpha_{2}(\frac{i^{*}}{i^{*} + k}) - d - \mu - \beta i^{*} & s_{1}^{*}(\alpha_{1} - \frac{k\alpha_{2}}{(i^{*} + k)^{2}}) - d + \nu - \beta s_{1}^{*} \\ \beta i^{*} & \beta s_{1}^{*} - \nu - \mu \end{pmatrix}$$

By using $s_{1}^{*} = \frac{\nu + \mu}{\beta}$, we have
 $J_{2} = J(\frac{\nu + \mu}{\beta}, i^{*}) = \begin{pmatrix} \alpha_{1}(2(\frac{\nu + \mu}{\beta}) - 1 + i^{*}) - \alpha_{2}(\frac{i^{*}}{i^{*} + k}) - d - \mu - \beta i^{*} & \frac{\nu + \mu}{\beta}(\alpha_{1} - \frac{k\alpha_{2}}{(i^{*} + k)^{2}}) - d - \mu \\ \beta i^{*} & 0 \end{pmatrix}$
The characteristic equation of this matrix is $\lambda^{2} - n\lambda + q = 0$ in which

acteristic equation of this matrix is λ^2 0, m which $p_{\Lambda} + q$

(3.12)
$$p = Tr(J(s_1^*, i^*)) = \alpha_1(2(\frac{\nu + \mu}{\beta}) - 1 + i^*) - \alpha_2(\frac{i^*}{i^* + k}) - d - \mu - \beta i^*$$

(3.13)
$$q = Det(J(s_1^*, i^*)) = \beta i^* (-(\alpha_1 - \frac{k\alpha_2}{(k+i^*)^2})(\frac{\nu+\mu}{\beta}) + d + \mu).$$

We know p < 0 and q > 0 are sufficient conditions for the local stability. But

(3.14)
$$p < 0 \iff \alpha_1(2(\frac{\nu+\mu}{\beta}) + i^*) < \alpha_1 + \alpha_2(\frac{i^*}{i^*+k}) + d + \mu + \beta i^* \iff \frac{\alpha_1}{\beta}(\frac{\nu+\mu}{\mu+d}) < \frac{1}{2}(\frac{1}{\mu+d}(\alpha_1(1-i^*) + \alpha_l(\frac{i^*}{i^*+k}) + \beta i^*) + 1).$$

And the first equation of (2.3) in endemic equilibrium is equivalent to

(3.15)
$$\frac{1}{\mu+d}(\alpha_1(1-i^*) + \alpha_2(\frac{i^*}{i^*+k}) + \beta i^*) + 1 = \frac{\mu+\alpha_1s_1^{*2} + d(1-i^*) + \nu i^*}{s_1^*(\mu+d)}$$

It follows from (3.14) and (3.15) that

$$(3.16) \qquad \begin{array}{l} p < 0 \\ \iff \frac{\beta}{\alpha_1} (\frac{\mu+d}{\mu+\nu}) > \frac{2(\nu+\mu)(d+\mu)}{\beta(\mu+\alpha_1\frac{(\nu+\mu)^2}{\beta^2} + d(1-i^*) + \nu i^*)} \\ \iff R_0^* > \frac{2(\mu+\nu)(\mu+d)}{\beta(\mu+\frac{(\mu+\nu)(\mu+d)}{\beta R_0^*} + d(1-i^*) + \nu i^*)} \\ \iff \frac{R_0^*\beta(\mu+d+\nu)}{2(\mu+\nu)(\mu+d)} > \frac{1}{2} \\ \iff R_0^*R_0^2 > \frac{\mu+d}{\mu+d+i^*(\nu-d)}. \end{array}$$

On the other hand

a > 0

Furthermore, we obtain the following results.

Proposition 3.2. Let $R_0^* > 1$, $R_0^1 > 1$, and $\beta > \alpha_1$, then the endemic steady state is locally asymptotically stable.

Proof. Since $R_0^1 > 1$, we have

(3.18)
$$\alpha_1(2\frac{\nu+\mu}{\beta}-1) - d - \mu = (d+\mu)(\frac{2}{R_0^*} - R_0^1 - 1) \le 1 - R_0^1 < 0$$

From (3.12) and (3.17) we conclude that the above relation, $\alpha_1 < \beta$ and $R_0^* > 1$, implies p < 0 and q > 0, hence the endemic steady state is locally asymptotically stable.

Proposition 3.3. Let $R_0^2 \ge 2$, $R_0^1 < 1$, $\beta < \alpha_1$ and $\nu < d$, then the endemic steady state is locally asymptotically stable.

Proof. It follows from these assumptions, $R_0^* > 1$, $\alpha_1 i^* \leq \alpha_1 < d + \mu$ and $2\frac{\nu+\mu}{\beta} - 1 < 0$, hence p < 0 and q > 0 which implies that the endemic equilibrium point is locally asymptotically stable.

213

3.1. Global stability of the endemic equilibrium point Now we study global asymptotic stability of the steady states.

Theorem 3.1. Suppose $R_0^1 < 1$ and $R_0^2 > 1$, then the endemic steady state is global asymptotic stable with respect to Ω , when $\alpha_1 < \beta$, or $\nu > d$. Furthermore, if $\alpha_1 > \beta$ and $\nu < d$, and $R_0^1 < 1$, $R_0^2 \ge 2$, the endemic steady state is global asymptotic stable.

Proof. We rewrite the system (2.3) in terms of R_0^1 and R_0^2 , as follows (3.19)

$$\begin{cases} \frac{ds_1}{dt} = (d+\mu)(1-s_1-i)(1-R_0^1s_1) + (\mu+\nu)i(1-R_0^2s_1) - \alpha_2s_1(\frac{i}{i+k}) \\ \frac{di}{dt} = (\mu+\nu)i(R_0^2s_1-1) \end{cases}$$

Now if we use $B(s_1, i) = \frac{1}{i}$ as a Dulac function, we have:

$$div(Bf, Bg) = \frac{\partial(Bf)}{\partial(s_1)} + \frac{\partial(Bg)}{\partial(i)} = \\ = -\alpha_2(\frac{1}{i+k}) - (\mu+\nu)R_0^2 + \frac{d+\mu}{i}((R_0^1s_1 - 1) - R_0^1(1 - s_1 - i)))$$

which is negative in the set $\{(s_1, i) \in \Omega : i > 0\}$ if $R_0^1 < 1$. Now by integration we have, $i(t) = i(0)e^{\int_0^t (\beta s_1(\tau) - \nu - \mu)d\tau}$. Hence the line i = 0 has positive invariance, which implies that the system has no cycle provided $R_0^1 < 1$.

On the other hand, by applying $R_0^2 > 1$ and $R_0^1 < 1$ in (3.12) and (3.13), we have q > 0 and

$$p < \alpha_1 i^* - \alpha_2 \left(\frac{i^*}{i^* + k}\right) - \beta i^*,$$

which is negative when $\alpha_1 < \beta$. Hence, in this case, the endemic steady state is locally stable and Poincare-Bendixon theorem implies the global stability with respect to Ω . If $\nu \ge d$, as indicated in proposition 3.2, the endemic point is locally stable, and the Poincare-Bendixon theorem implies its global stability. Proposition 3.3 concludes the last case.

In Figure 4 we present the phase portrait in a set of parameters with $R_0^1 < 1$, $R_0^2 > 1$ and $\alpha_1 < \beta$.

4. Numerical Explorations

In this part, we draw the phase diagram in several modes using maple software, so that the obtained analytical results can be seen geometrically. We present three cases.

In many diseases, for example, sexually transmitted infections (STIs), such as chlamydia, gonorrhea, genital warts,..., it is possible to inform about the spreading disease and its consequences on personal health or social and economic issues through targeted campaigns or through public media as well as person-to-person meetings. These warnings cause the cautious behavior of people in the society in different ways. For example, using condoms, having fewer sexual partners, using vaccines... are examples of such precautions. In diseases that follow the SIS model, for example gonorrhea which is an STI, the extended SIS model introduced in this article can be used to analyze the effect of such information on disease dynamics.

In the case of Chlamydia, as it is mentioned in [14], the average infectious period for individuals that are unaware and do not seek treatment early is found to be 26 weeks, and for individuals that seek treatment early, mainly due to being aware, the average infectious period is around 13 weeks, hence we choose the units of time to be in term of weeks.

Case 1. In the first case, we draw the phase diagram, in Figure 2, when trivial DFE or DFE are globally asymptotically stable as it is proved in Th. 2.2. We use two set of parameters, (a): $\alpha_2 = \alpha_1 = \beta = \nu = \mu = d = 0.5$, k = 1.2, in which, $R_0^2 < 1$, $R_0^1 < 1$. And (b): $\alpha_2 = \alpha_1 = \beta = \nu = 0.5$, $d = \mu = 0.1666$, k = 1.2, with $R_0^2 < 1$ and $R_0^1 > 1$.

Case 2. In this case, we draw the phase portrait of the system and the components of the solutions when $(R_0^1, R_0^2) = (1, 1)$, in Figure 3, to explore the elliptic sector whose existence proved in Th. 2.3. We use the following set of parameters, $\alpha_2 = 0.15, \alpha_1 = 0.5, \nu = 0.5, \mu = 0.25, d = 0.25, \beta = 0.75, k = 1.02$ and initial values $(s_1(0), i(0)) = (1.1, 0.005)$. At this parameter values we have, $R_0^1 = R_0^2 = 1$. Picture (a) shows the phase portrait of the system, (b) shows th graph of the component i(t)of the solution, and (c) shows the graph of $s_1(t)$. The graph of i(t), i.e., the number of infected individuals in time t, of this trajectory, shows the occurrence of an epidemy.

Case 3. In the third case, we draw the phase diagram, in Figure 4, to see the global asymptotic stability of endemic equilibrium point, proved in Th. 3.1. We use the following set of parameters, $\alpha_2 = \alpha_1 = \nu = \mu = d = 0.1111$, $\beta = 0.9$, k = 1.2, with $R_0^1 < 1$ and $R_0^2 > 1$.

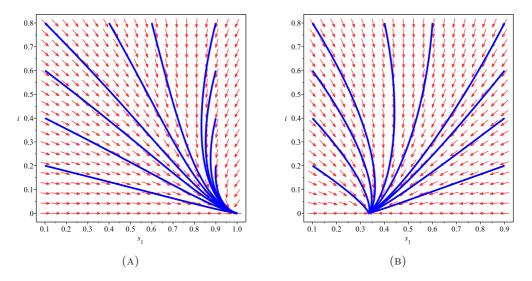


Figure 2. The phase portrait of the system in (a) shows that the trivial DFE is globally asymptotically stable and (b) shows that DFE is globally asymptotically stable.

5. Conclusion and Discussion

In this paper, the SIS epidemic model was improved by considering the effect of fear of contracting the disease at the time of outbreak and adopting cautious behaviors. It is observed that, the proposed model has two DFE equilibrium points, and we studied their local and global stability. We also computed the basic reproduction number of the model, which has the form, $R_0 = max\{R_0^1, R_0^2\}$, with $R_0^1 = \frac{\alpha_1}{d+\mu}$ and $R_0^2 = \frac{\beta}{\mu+\nu}$. Our analysis showed that this model, despite its simplicity, has a complex dynamic. For example, we showed that when $(R_0^1, R_0^2) = (1, 1)$, two DFE equilibrium points are merged, and a degenerate Bogdanov-Takens bifurcation of codimension three occurs. The occurrence of this bifurcation is a sign of difficulty in the elimination of infection when (R_0^1, R_0^2) is close to (1, 1). In this case, we have shown that the system has elliptic sector and its diagram shows the occurrence of an outbreak.

Furthermore, we studied endemic steady states of the model and its dynamic properties, such as locally asymptotically stability and global asymptotic stability.

Our study showed that the relationship between awareness rate and infection rate could determine the dynamics of the system in various cases. For example, when

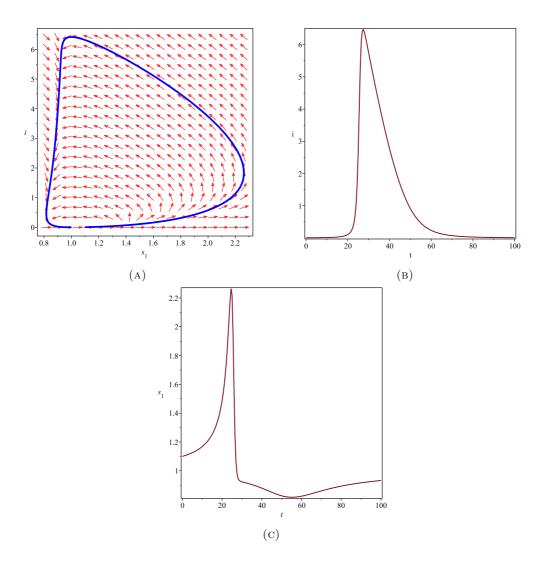


Figure 3. (a) shows the phase portrait of the system, which is an elliptic sector. (b) and (c) shows the components of this solution in term of time.

the awareness rate is less than the infection rate, i.e., $\alpha_1 < \beta$, the disease becomes endemic, i.e., the endemic equilibrium is global asymptotic stable when $R_0^1 < 1$ and $R_0^2 > 1$.

The extension done on the SIS model, in other models such as SIR, SEIR,..., can be designed and analyzed. For example the SIR model can be extended with the following diagram and S_1S_2IR system of equations,

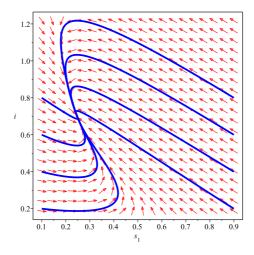
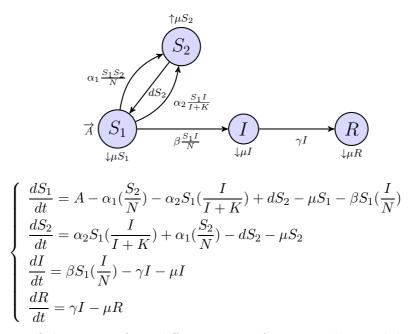


Figure 4. The phase portrait shows the global asymptotic stability of the endemic equilibrium point.



The analysis of this system from different point of views, such as stability, bifurcations and numerical simulations is a research question.

Conflict of interest. The authors declare that there is no conflict of interest regarding the publication of this paper.

References

- F. Arrigoni & A. Pugliese: Limits of a multipatch SIS epidemic model. J. Math. Biol. 45 (2002), 419-440. doi:10.1007/s002850200155
- S.M. Baer, et. al.: Multiparametric bifurcation analysis of a basic two-stage population model. SIAM J. Appl. Math. 66 (2006), 1339-1365. doi:10.1137/050627757
- 3. S. Bentout, et. al.: Impact of predation in the spread of an infectious disease with time fractional derivative and social behavior. International Journal of Modeling, Simulation, and Scientific Computing. doi:10.1142/s1793962321500239
- 4. R.I. Bogdanov: Versal deformations of a singular point on the plane in the case of zero eigenvalues. Func. Anal. Appl. 9 (1975), 144-145. doi:10.1007/bf01075453
- F. Brauer & C. Castillo-Chavez: Mathematical Models in Population Biology and Epidemiology: Second Edition. Springer, New York, 2012. doi:10.1007/978-1-4164 -1686-9
- C. Castillo-Chavez & H.R. Thieme: Asymptotically autonomous epidemic models. In: Mathematical Population Dynamics, Analysis of Heterogeneity, Vol. 1. Theory of Epidemics, 1995, 33-50. doi:10.1016/0022-247x(87)90211-3
- 7. J. Cui, et. al.: The impact of media on the control of infectious diseases. Journal of Dynamics and Differential Equations 20 (2008), 31-53. doi:10.1007/s10884-007-9075-0
- A. D'Onfrio & P. Manfredi: Information-related changes in contact patterns may trigger oscillations in the endemic prevalence of infectious disease. J. Theor. Biol. 256 (2009), 473-478. doi:10.1016/j.jtbi.2008.10.005
- B. Dubeya, et. al.: Role of media and treatment on an SIR model. Nonlinear Analysis, Modeling and Control 21 (2016), no. 2, 185-200. doi:10.15388/na.2016.2.3
- J. Guckenheimer & P.J. Holmes: Nonlinear oscillations, dynamical systems, and bifurcation of vector fields. Springer, New York, 1983. doi:10.1007/978-1-4612-1140-2
- H.W. Hethcote & J.A. Yorke: Gonorrhea: transmission dynamics and control. volume 56 of Lecture Notes in Biomathematics. Springer-Verlag, Berlin, 1984. doi:10.1007/978-3-662-07544-9
- J. Joo & J.L. Lebowitz: Behavior of susceptible-infected-susceptible epidemics on heterogeneous networks with saturation. Phys. Rev. E 69 (2004), 066105. doi:10. 1103/physreve.69.066105
- M.J. Keeling & P. Rohani: Modeling Infectious Diseases In Humans and Animals. Princeton Univ. Press, New Jersey, 2008. doi:10.1515/9781400841035
- I.Z. Kiss, et. al.: The impact of information transmission on epidemic outbreaks. Math. Biosciences 225 (2010), 1-10. doi:10.1016/j.mbs.2009.11.009
- Y.A. Kuznetsov, Practical computation of normal forms on center manifolds at degenerate Bogdanov-Takens bifurcation. *International J. Bif. Chaos.* 15(11) (2005),3535-3546. doi:10.1142/s0218127405014209

- Y.A. Kuznetsov: Elements of applied bifurcation theory. Third Edition, Springer, NewYork, 2004. doi:10.1007/978-1-4757-3978-7
- 17. W.M. Liu, et. al.: Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological models. J. Math. Biol. 23 (1986), 187-204. doi:10.1007/bf00276956
- M. Lizana & J. Rivero: Multiparametric bifurcations for a model in epidemiology. J. Math. Biol. 35 (1996), 21-36. doi:10.1007/s002850050040
- M. Martcheva: An introduction to mathematical epidemiology. Springer, New York, 2015. doi:10.1007/978-1-4899-7612-3
- R. Memarbashi & E. Sorouri: Modeling the effect of information transmission on the drug dynamic. Eur. Phys. J. Plus, 135, 54 (2020). doi:10.1140/epjp/s13360 -019-00064-5
- Z. Mukandavire, et. al.: Modelling effects of public health educational campaigns on HIV/AIDS transmission dynamics. Applied Mathematical Modelling 33 (2009), no. 4, 2084-2095. doi:10.1016/j.apm.2008.05.017
- R.K. Naji & A.A. Thirthar: Stability and bifurcation of an SIS epidemic model with saturated incidence rate and treatment function. Iranian J. Math. Sci. Inf. 15 (2020), no. 2, 129-146. doi:10.1016/j.amc.2013.10.020
- F. Takens: Forced oscillations and bifurcations. Comm. Math. Inst. Rijkoniversiteit. 2 (1974), 1-111. doi:10.1887/0750308036/b1058c1
- Y. Takeuchi, X. Liu & J. Cui: Global dynamics of SIS models with transport-related infection. J. Math. Anal. Appl. **329** (2007), 1460-1471. doi:10.1016/j.jmaa.2006. 07.057
- P. Van den Driessche & J. Watmough J.: A simple SIS epidemic model with a backward bifurcation. J. Math. Bio. 40, (2000), 525-540. doi:10.1007/s002850000032
- P. Van den Driessche & J. Watmough J.: Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Math. Biosci. 180 (2002), 29-48. doi:10.1016/s0025-5564(02)00108-6
- Y.N. Xiao, et. al.: Dynamics of an infectious disease with media/psychology induced non-smooth incidence. Math. Biosci. Eng. 10 (2013), 445-461. doi:10.3934/mbe. 2013.10.445

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