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A qualitative analysis of an SII_aR epidemic model with saturated incidence, awareness and treatment functions

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Abstract

An SII_aR - type epidemic model with saturated incidence rate affected by inhibitory effect and saturated awareness and treatment functions of infectious individuals is proposed to understand the effect of delays for awareness of disease statuses and treatment of infective individuals on the disease spread. The awareness and treatment functions in this paper are continuous and differential functions which exhibit the effects of delayed awareness and treatment when the rates of awareness and treatment are lower and the number of infected individuals is getting larger. The proposed model has at most two equilibrium points, the disease-free equilibrium (DFE) and endemic equilibrium (EE). The local and global stability conditions of all possible equilibrium points are established. Mathematical analysis reveals that the model has a locally-asymptotically stable DFE whenever a certain epidemiological threshold, known as the *effective reproduction number,* R_e, is less than unity. However, it is shown that the global asymptotic stability of the DFE was largely dictated by the delays in awareness of disease statuses and treatment of infective individuals; when such delays are weak, then the DFE is globally-asymptotically stable (GAS) when the effective reproductive number is less than unity. When the delays are strong, it is shown that there is the possibility of the existence of the backward bifurcation phenomenon whereby the DFE will co-exist with EE, when the effective reproduction number is less than unity. The backward bifurcation phenomenon existed when the delays occurred either singly or jointly, that is if the delay parameters violate certain threshold conditions. Mathematical analysis provides threshold conditions that allows for the global stability of the EE whenever the associated reproduction number is greater than unity. It was also observed that the model changes its stability behaviour around the endemic equilibrium from stable to unstable as the bifurcation parameter k changes and the system exhibits Hopf

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bifurcation near the endemic equilibrium for $k = k^*$. Some conditions on the model parameters are obtained to show the existence as well as nonexistence of limit cycles. The results suggest that with timely identification of infectives from the population of infected individuals and treatment of infected individuals, the ultimate goal of disease eradication can be achieved.

Keywords: Epidemic model; saturated incidence; saturated awareness; treatment; stability; bifurcation.

1 Introduction

Since Bernoulli developed the first dynamic model of Smallpox spread in 1760 [5], there has been a sustained and distinguished history of mathematical modelling in epidemiology. The study of epidemiological models has tremendously aided in revealing the underlying mechanisms that influence the transmission and control of infectious diseases. In fact, mathematical models of infectious diseases have continued to serve as reliable guides for the formulation of public health policy that result in minimizing the incidence/spread or even the eradication of such diseases [3, 6, 9, 16]. In this direction, several researchers have proposed and tested epidemic models of diseases such as HIV/AIDS, Tuberculosis, Syphilis, Malaria, Hepatitis, SARS, and Ebola to several zoonotic diseases to better understand the underlying dynamic mechanisms that drives the epidemics (for example, see the review in [5]). Among the numerous epidemiological models that abound in the literature, the Susceptible – Infected – Recovered (SIR) – type of compartmental models and Ross – McDonald models have played a critical role in the development of modern mathematical epidemiology.

In the modelling of infectious diseases, the functional form of the *incidence rate or force of infection*, namely the function describing the mechanism of disease transmission plays a key factor in conducting the rich dynamical behaviour in many related literature [15]. Of course, equally also of importance, is the description of the intervention policy to contrast the disease spread (vaccination, treatment, health campaign, etc). *Bilinear* (βSI) and *standard* ($\beta SI/N$) *incidence rates* (where β is the probability of disease transmission per contact, S is the susceptible class, I is the infected class, and N is the total population), both monotonically increasing functions of the total infected class, have been frequently used in classical epidemic models. Classical epidemiological models, the standard for predicting and managing the spread of infectious diseases, assume that contacts between susceptible and infectious individuals depend on their relative frequency in the population. The behavioural factors that underpin contact rates are not generally addressed. In such models, the dynamics of models are relatively simple and almost determined by the *basic reproduction number* R_0: the disease will be eliminated if $R_0 < 1$, otherwise, the disease will persist; and these models do usually have one or two endemic equilibrium.

There is, however, an emerging class of models that addresses the feedbacks between infectious disease dynamics and the behavioural decisions driving host contact. That is, in recent years, attempts have been made to develop realistic mathematical models for the transmission dynamics of infectious diseases by incorporating incidence functions that play a key role in ensuring that the

models indeed give reasonable qualitative description of the transmission dynamics of the diseases [16, 22]. For instance, some factors such as the demographics of the involving populations, media coverage, behavioural changes, and recovery rate, may affect the incidence rate directly or indirectly; these may also substantially affect the dynamical behaviour of the models.

Keeping the above factors in view, several authors have suggested that the disease transmission process may have a nonlinear incidence rate. This may allow for the inclusion of intervention strategies such as psychological effect or behavioural changes in order to prevent unbounded contact rates. Consequently, various nonlinear incidence rates have been proposed recently due to the fact that they can produce rich dynamics for epidemic models. Contrasted to models with bilinear or standard incidence, complex dynamic behaviour may occur when more general nonlinear incidences are used [7] and the references therein.

The nonlinear, saturation function, sometimes called Holling type II function, was first used by Capasso and Serio [9] for studying the population dynamics of cholera [29]. Since then, several mathematical models have been proposed with saturated functions representing the incidence/force of infection of a disease or control measures like behavioural change or treatment [27] and the references therein. The reason for this is due to the fact that a saturated incidence rate carries the idea of an inhibiting effect due to changes in behavioural patterns from the susceptible group when there is an increase in their number in response to the severity of the disease or a crowding effect of infected individuals [29, 32] and the references therein, hence the number of infectives contacts between susceptibles and infected individuals may saturate at high infective levels [29].

Several authors (see, for example, [26], and the references therein), suggested different types of nonlinear incidence rates. The saturated incidence rate given by

$$g(I)S = \frac{aIS}{1+\beta I}, \qquad (1)$$

where

$$g(I) = \frac{aI}{1+\beta I}, \qquad (2),$$

(tends to a saturation level when I gets large), β is the saturation factor, aI measures the infection force of the disease and $1/1+\beta I$ measures the inhibition effect from the behavioural change of the susceptible individuals when their number increases or from the crowding effect of the infectious individuals, was introduced by [2] and later used by many authors (see [26], and the references therein). This incidence rate seems more reasonable than the bilinear incidence rate

$$g(I)S = aIS, \qquad (3)$$

because it includes the behavioural change (i.e. the protection measures by the susceptible individuals) and the crowding effect of the infectious individuals and prevents the unboundedness of the contact rate by choosing suitable parameters.

The general incidence rate

$$g(I)S = \frac{kI^pS}{1+\alpha I^q}, \qquad (4)$$

was proposed by [24] and used by a number of authors (see, for example, [1, 11, 17], etc). Nonlinear incidence rates of the form kI^pS^q were investigated by [24] and [25]. On the other hand, [4] and [10] introduced independently nonlinear incidence rate known after as Beddington-DeAngelis type of incidence rate

$$g(I)S = \frac{\alpha IS}{1+\beta S+\gamma I}, \qquad (5)$$

which adopted both the saturation factor and the effect of crowding of infected individuals. Later on, some authors [12, 13, 18, 19] and the references therein, used this incidence rate to describe the transmission of disease in their epidemiological models.

It is pertinent to note that, in the course of an outbreak of an infectious disease or if an infectious disease establishes in a population, knowledge of individuals' disease statuses is very important in controlling and eliminating the disease from the population. For example, knowledge (i.e. awareness through counselling and testing) of one's HIV status is very crucial in the control and elimination of the disease. For example, due to the psychological effects and the trauma that HIV individuals go through and stigmatization from the society and within the family, many HIV individuals find it difficult to present themselves for HIV screening at designated health centers. This leads to delays in them becoming aware of their HIV statuses, leading to spreading of the disease (virus) to other susceptible individuals. This could also be the case with people with tuberculosis and some other infectious diseases. Therefore, saturated awareness rate is suitable for this case. Thus, in this paper, a saturated awareness rate of the form (6)

$$A(I) = \frac{\delta I}{1+\omega I}, I \geq 0, \delta > 0, \omega \geq 0, \qquad (6)$$

will be adopted, where δ (a positive constant) is the awareness and ω measures the effect extent of the infected being delayed in becoming aware of his/her disease status.

Treatment is important and effective in controlling infectious diseases. In several published articles (see, for example, [14], and the references therein), authors had studied *SIR, SIS, SEIR* epidemic models with different treatment functions. Generally speaking, in classical epidemic models, the treatment rate of infected individuals is assumed to be either constant or proportional to the number of the infected individuals. However, we know that the treatment of a disease should have a maximal capacity and the treatment resources should be quite large. On the other hand, irregularities in the supply of medical facilities and limited treatment resources available in communities pose serious problems in the eradication of any disease. Therefore, it is very important to choose a suitable treatment functions for the epidemic disease. In the absence of effective therapeutic treatments and vaccines, the epidemical control strategies are based on taking appropriate preventive measures. For instance, [31] considered an SIR epidemic model with constant treatment rate

$$T(I) = \begin{cases} r, I > 0, \\ 0, I = 0, \end{cases} \qquad (7)$$

where $r > 0$ (is a positive constant) and I is the number of infected individuals. They studied stability analysis and showed that their model exhibits various types of local bifurcations.

In many developing countries, it is known that the number of patients that need to be treated may exceed the carrying capacity of the local hospitals because of the restrictions on medical conditions. Hence, saturated treatment rate is also a suitable choice for this case. Recently, saturated-type treatment rates have been adopted in all kinds of epidemic models, such as for SIR, for SIS as well as for $SEIR$ models and so on (see, for example, [15], and the references therein). For example, [28] modified the treatment rate (7) to Holling type II

$$h(I) = \frac{\beta I}{1+\gamma I}, I \geq 0, \beta > 0, \gamma \geq 0, \qquad (8)$$

which is a saturated treatment rate. β represent the cure and γ measures the effect extent of the infected being delayed for treatment. We can observe that the treatment rate $h(I) \sim \beta I$ when I is small enough, whereas $h(I) \sim \beta/\gamma$ when I is large enough. Their study showed that, with varying amount of medical resources and their supply efficiency, the target model admits various bifurcations.

Thus, from the preceding arguments, saturated awareness and treatment rates are used to describe the effect of delays in awareness (of an individual's disease status) and treatment of infected individuals especially in communities with improper or poor public disease awareness and scarce medical facilities and personnel.

Keeping the above discussions in view, and considering psychological effect and behavioural changes during the course of an infectious disease (such as HIV/AIDS) in a community or population and the shortage of medical facilities and the limitations of resources, it is reasonable to use saturated incidence, awareness and treatment functions in modelling control strategies. Meanwhile, delays and irregularity in the supply of drugs are one of the major factors in the continued spread of infectious diseases (and HIV). It is therefore necessary to take a control that incorporates awareness and behavioural modifications and with treatment as complementary programmes to prevent disease spreading.

Hence, in this study, in other to incorporate psychological effect and /or behavioural changes with treatment on the dynamics of an infectious disease (such as (HIV/AIDS), a mathematical model describing an epidemiological system having SIR (susceptible, infected and recovered) type model with nonlinear incidence function

$$f(I)S = \frac{kIS}{1+\alpha I^2}, \qquad (9)$$

(a special case of (4)), saturated awareness rate (6) and saturated treatment rate (8) simultaneously is proposed and rigorously analyzed. The incidence function (9) is reasonable because when the number of infectious individuals increases and is widely reported through various social media outfits; the susceptible individuals will stay alert spontaneously to reduce any unnecessary contact with others, especially with the infectives, thus lowering the contact and transmission incidence.

2 The mathematical model

The epidemic model with nonlinear incidence rate and with saturated awareness and treatment rates to be studied takes the following form that is described by four nonlinear ordinary differential equations (10).

$$\begin{aligned}
\dot{S} &= b - \frac{kIS}{1+\alpha I^2} - \mu S \\
\dot{I} &= \frac{kIS}{1+\alpha I^2} - A(I) - T(I) - (\alpha_1 + \mu)I \\
\dot{I}_a &= A(I) - (\phi + \alpha_2 + \mu)I_a \\
\dot{R} &= T(I) + \phi I_a - \mu R
\end{aligned} \tag{10}$$

where

$S(t)$ = Number of Susceptibles,

. $I(t)$ = Number of Infectives,

$I_a(t)$ = Number of Infected unaware of their disease statuses,

$R(t)$ = Number of Recovered (Treated) Individuals,

b = Recruitment Rate into the (susceptible) Population,

μ = Natural Death Rate of the Population,

k = Proportionality Constant,

α = Parameter that Measures Psychological or Inhibitory effect,

α_1 = Death Rate due to Infection,

α_2 = Death rate due to Infection by unaware Infectives,

ϕ = Treatment Rate of aware Infectives,

$$A(I) = \frac{\delta I}{1+\omega I}, I \geq 0, \delta > 0, \omega \geq 0,$$

is the saturated awareness rate where δ (a positive constant) is the awareness and ω measures the effect extent of the infected being delayed in becoming aware of their disease statuses, and

$$T(I) = \frac{\beta I}{1+\gamma I}, I \geq 0, \beta > 0, \gamma \geq 0,$$

is the saturated treatment rate where β represents the cure and γ measures the effect extent of the infected being delayed for treatment. We can observe that the treatment rate $T(I) \sim \beta I$ when I is small enough, whereas $T(I) \sim \beta/\gamma$ when I is large enough.

2.1 Basic properties of the model

Since the model (10) monitors human populations, it is expected that all its associated variables and parameters are nonnegative. Further, the following non-negativity result holds.

Theorem 2.1 Let the initial data for the model (10) be $S(0)>0$, $I(0)>0$, $I_a(0)>0$, $R(0)>0$, and $R(0)>0.$ Then the solutions

$$\left(S(t),I(t),I_a(t),R(t)\right)$$

of the model (10), with positive initial data, will remain positive for all time $t>0.$

Proof: Let $t_1 = Sup\left\{t>0 : S(t)>0, I(t)>0, I_a(t)>0, R(t)>0\right\}$. It follows from (10) that

$$\frac{dS}{dt} = b - g(I)S - \mu S,$$

which can be written as

$$\frac{d}{dt}\left\{S(t)\exp\left[\mu t + \int_0^t g(\tau)d\tau\right]\right\} = b\left\{\exp\left[\mu\tau + \int_0^t g(\tau)d\tau\right]\right\}.$$

Thus,

$$S(t_1)\exp\left[\mu t_1 + \int_0^{t_1} g(\tau)d\tau\right] - S(0) = \int_0^{t_1} b\left\{\exp\left[\mu y + \int_0^y g(\tau)d\tau\right]\right\}dy,$$

so that,

$$S(t_1) = S(0)\exp\left[-\mu t_1 - \int_0^{t_1} g(\tau)d\tau\right]$$
$$+\exp\left[-\mu t_1 - \int_0^{t_1} g(\tau)d\tau\right]\mathrm{I}$$
$$\times\int_0^{t_1} b\left\{\exp\left[\mu y + \int_0^y g(\tau)d\tau\right]\right\} > 0.$$

Similarly, it can be shown that $I(t)>0$, $I_a(t)>0$, and $R(t)>0$ for all time $t>0.$ Hence all solutions of the model (10) remain positive for all non-negative initial conditions, as required.

□

We claim the following.

Theorem 2.2 The closed set

$$\Psi = \left\{\left(S,I,I_a,R\right)\in\Re_+^4 : N \le \frac{b}{\mu}\right\}$$

is positively-invariant and attracts all positive solutions of the model (10).

Proof: Adding the equations of the model (10) gives

$$\frac{dN}{dt} = b - \mu N - \alpha_1 I - \alpha_2 I_a. \qquad (11)$$

Since the right-hand side of (11) is bounded by $b-\mu N$, a standard comparison theorem [20] can be used to show that

$$N(t) \le N(0)e^{-\mu t} + \frac{b}{\mu}\left(1-e^{-\mu t}\right). \tag{12}$$

In particular, if $N(0) \le \frac{b}{\mu}$, then $N(t) \le \frac{b}{\mu}$. Thus, Ψ is a positively-invariant set, and attracting, under the flow generated by (10). Furthermore, if $N(0) \ge \frac{b}{\mu}$, then either the solution enters Ψ in finite time or $N(t)$ approaches to $\frac{b}{\mu}$ as $t \to \infty$. Hence no solution path leaves through any boundary of Ψ and the region Ψ attracts all solutions in $\Re_+^4$. Hence it is sufficient to consider the dynamics of the flow described by (10) in Ψ. In this region, the model can be considered as been mathematically and epidemiologically well-posed [16]. □

3 Analysis of the model

It may be readily seen that the first two equations in (10) are independent of the variables I_a and R (and moreover, since the results from the first two differential equations serve as the inputs for the equations for I_a and R), it is sufficient to consider and analyse the following reduced model:

$$\begin{aligned} \dot{S} &= b - \frac{kIS}{1+\alpha I^2} - \mu S, \\ \dot{I} &= \frac{kIS}{1+\alpha I^2} - \frac{\beta I}{1+\gamma I} - \frac{\delta I}{1+\omega I} - (\alpha_1 + \mu) I. \end{aligned} \tag{13}$$

3.1 Local Stability of Disease-Free Equilibrium (DFE)

For any values of parameters, the model (13) has a DFE, given by $E_0 = \left(\frac{b}{\mu}, 0\right)$. To determine the local stability of the DFE, we will linearize the system (13) around the DFE. The Jacobian of the system (13), evaluated at the DFE, is given by

$$J_{(E_0)} = \begin{pmatrix} -\mu & -\frac{bk}{\mu} \\ 0 & \frac{bk}{\mu} - \left(\beta + \delta + \alpha_1 + \mu\right) \end{pmatrix},$$

which has eigenvalues $\lambda_1 = -\mu$ and $\lambda_2 = \left(\alpha_1 + \mu + \beta + \delta\right)\left(R_e - 1\right)$, where

$$R_e = \frac{bk}{\mu(\alpha_1 + \mu + \beta + \delta)}, \tag{14}$$

is the effective reproduction number of the model (13). Clearly, the eigenvalue, λ_2, is negative if and only if $R_e < 1.$ Hence, the DFE is locally asymptotically stable when $R_e \leq 1$ and unstable otherwise.

Theorem 3.1 *The DFE* (E_0) *of the model (13) is locally asymptotically stable (LAS) if* $R_e < 1$ *and a saddle point with stable manifold locally in the S-direction and unstable manifold locally in the I-direction when* $R_e > 1.$

The threshold quantity, R_e, is the *effective reproduction number* of the disease [16,30], and it represents the average number of secondary infectious generated by a typical infected (HIV) individual in a completely susceptible population when there are already control strategies such as awareness programmes and treatment [16, 30]. The epidemiological implication of Theorem 3.1 is that when R_e is less than unity, the disease can be eliminated from the population if the initial sizes of the sub-populations of the model are in the basin of attraction of the DFE (E_0). Hence, a small influx of infected individuals into the population will not generate large disease outbreaks, and the disease dies out in time.

3.2 Existence and stability of endemic equilibrium(EE)

To find the conditions for the existence of the endemic equilibrium for the model (13), denoted by $E_e = (S^*, I^*)$, the following algebraic equations (14), obtained from (13) by setting the right-hand sides to zero, are solved simultaneously.

$$\begin{aligned} &b - \frac{kI^*S^*}{1+\alpha I^{*2}} - \mu S^* = 0, \\ &\frac{kI^*S^*}{1+\alpha I^{*2}} - \frac{\beta I^*}{1+\gamma I^*} - \frac{\delta I^*}{1+\omega I^*} - (\alpha_1 + \mu)I^* = 0. \end{aligned} \tag{14}$$

From the second equation of (14), we have

$$I^*\left(\frac{kS^*}{1+\alpha I^{*2}} - \frac{\beta}{1+\gamma I^*} - \frac{\delta}{1+\omega I^*} - (\alpha_1 + \mu)\right) = 0,$$

where $I^* = 0$ corresponds to the DFE (E_0) (whose stability has already been established) and

$$\frac{kS^*}{1+\alpha I^{*2}} - \frac{\beta}{1+\gamma I^*} - \frac{\delta}{1+\omega I^*} - (\alpha_1 + \mu) = 0$$

$$\Rightarrow S^* = \frac{\left[\beta(1+\omega I^*) + \delta(1+\gamma I^*) + (\alpha_1 + \mu)(1+\gamma I^*)(1+\omega I^*)\right](1+\alpha I^{*2})}{k(1+\gamma I^*)(1+\omega I^*)}. \tag{15}$$

Substituting (15) into the first equation of (14), the following quartic equation in I^* is obtained:

$$f(I^*) = a_1 I^{*4} + a_2 I^{*3} + a_3 I^{*2} + a_4 I^* + a_5 = 0, \tag{16}$$

where the coefficients $a_1, a_2, ..., a_5$ are given by

$$\begin{aligned}
a_1 &= \mu\alpha\gamma\omega(\alpha_1 + \mu) > 0,\\
a_2 &= k\gamma\omega(\alpha_1 + \mu) + \mu\alpha\left[\omega(\alpha_1 + \mu + \beta) + \gamma(\alpha_1 + \mu + \delta)\right] > 0,\\
a_3 &= k\left[\gamma(\alpha_1 + \mu + \delta) + \omega(\alpha_1 + \mu + \beta)\right] + \mu\alpha(\alpha_1 + \mu + \beta + \delta) + \mu\gamma\omega(\alpha_1 + \mu) - bk\gamma\omega,\\
a_4 &= k(\beta + \delta + \alpha_1 + \mu) + \mu(\beta\omega + \gamma\delta) + \mu(\alpha_1 + \mu)(\gamma + \omega) - bk(\gamma + \omega),\\
a_5 &= \mu(\beta + \delta + \alpha_1 + \mu)(1 - R_e).
\end{aligned} \tag{17}$$

In order to gain insight into the effects of ω and γ on the existence of the endemic equilibria, a study of the polynomial (16) will be carried out.

3.2.1 Case 1: $\omega = \gamma = 0$

In this situation, setting $\gamma = 0$ and $\omega = 0$ in Eq. (16), yields the quadratic equation

$$f(I^*) = \tilde{a}_3 I^{*2} + \tilde{a}_4 I^* + a_5 = 0, \tag{18}$$

where

$$\begin{aligned}
\tilde{a}_3 &= \mu\alpha d > 0,\\
\tilde{a}_4 &= kd > 0,\\
a_5 &= \mu d(1 - R_e),
\end{aligned} \tag{19}$$

with $\tilde{a}_3$ and $\tilde{a}_4$ are now evaluated from a_3 and a_4, respectively, and $d = \alpha_1 + \mu + \beta + \delta.$ We will use this notation in the whole paper.

Note that $a_5 < 0$ if and only if $R_e > 1$, $a_5 = 0$ if and only if $R_e = 1$ and $a_5 > 0$ if and only if $R_e < 1.$

It is also easy to see that $\tilde{a}_3 > 0,\ \tilde{a}_4 > 0$ and $a_5 > 0$ (the latter when $R_e < 1$).

The quadratic equation (18) may have two roots if $\Delta_1 > 0$, which are denoted as

$$I_1 = \frac{-\tilde{a}_4 - \sqrt{\Delta_1}}{2\tilde{a}_3},\ I_2 = \frac{-\tilde{a}_4 + \sqrt{\Delta_1}}{2\tilde{a}_3},$$

where $\Delta_1 = \tilde{a}_4^2 - 4\tilde{a}_3 a_5$. If $\Delta_1 = 0,$ the roots I_1 and I_2 of Eq. (18) coalesce into a unique root with multiplicity 2 denoted as $I^* = \frac{-\tilde{a}_4}{2\tilde{a}_3}.$ If $I_i > 0,$ system owns endemic equilibrium $E_i(S_i, I_i)$ with

$$S_i = \frac{\left[\beta(1 + \omega I_i) + \delta(1 + \gamma I_i) + (\alpha_1 + \mu)(1 + \gamma I_i)(1 + \omega I_i)\right](1 + \alpha I_i^2)}{k(1 + \gamma I_i)(1 + \omega I_i)},\ i = 1, 2.$$

If $I^* > 0,$ the 2 endemic equilibria E_1 and E_2 coalesce into one endemic equilibrium $E_e(S^*, I^*),$ where

$$S^* = \frac{\left[\beta\left(1+\omega I^*\right)+\delta\left(1+\gamma I^*\right)+\left(\alpha_1+\mu\right)\left(1+\gamma I^*\right)\left(1+\omega I^*\right)\right]\left(1+\alpha I^{*2}\right)}{k\left(1+\gamma I^*\right)\left(1+\omega I^*\right)}.$$

Based on the above discussions, we study the existence of equilibria in the following three cases.

(i) $R_e > 1.$

In this case, $bk > \mu d,$ then $\tilde{a}_3 > 0, a_5 < 0$ and $\Delta_1 > 0$, so $I_1 < 0, I_2 > 0;$ this regardless of the sign of $\tilde{a}_4.$ So the quadratic equation (18) has a unique root I_2. That is, system (13) has a unique endemic equilibrium $E_2 = \left(S(I_2), I_2\right).$

(ii) $R_e = 1.$

In this case, $bk = \mu d,$ then $\tilde{a}_3 > 0$, and $a_5 = 0$, and $\Delta_1 = \tilde{a}_4^2 > 0,$ so Eq. (13) has 2 roots, 0 and $\frac{-\tilde{a}_4}{\tilde{a}_3}.$ That is, Eq. (18) has no positive root if $\tilde{a}_4 \geq 0$ and a unique positive root I_2 if $\tilde{a}_4 < 0.$

Therefore, system (13) has a unique endemic equilibrium $E_2 = \left(S(I_2), I_2\right)$ if and only if $\tilde{a}_4 < 0.$ However, since $\tilde{a}_4 > 0,$ no such endemic equilibrium exists in this case.

(iii) $R_e < 1.$

In this case, $bk < \mu d,$ then $\tilde{a}_3 > 0, a_5 > 0$ and $\Delta_1 < 0,$ (also $\tilde{a}_4 > 0$) so Eq. (18) has no positive roots. Therefore, the quadratic (18) does not have positive roots, which implies that there is no endemic equilibria when $\gamma = \omega = 0$ and $R_e < 1$ in Eq. (16), ruling out the possibility of a backward bifurcation for this case.

Thus system Eq. (13) has 2 endemic equilibria $E_1 = \left(S(I_1), I_1\right)$ and $E_2 = \left(S(I_2), I_2\right)$ if and only if $\Delta_1 > 0,\ \tilde{a}_4 < 0$; and has one endemic equilibrium of multiplicity 2, $E_e = \left(S(I^*), I^*\right),$ if and only if $\Delta_1 = 0,\ \tilde{a}_4 < 0.$

Hence, if $\omega = \gamma = 0,$ then a unique endemic equilibrium exist only if $R_e > 1$ and there cannot be such an endemic equilibrium if $R_e < 1.$ In other words, if there is no delay in the treatment of infected individuals as well as no delay in an infected individual becoming aware of his/her disease status from the population of infected individuals, then it is not possible to have an endemic steady state solution when the effective reproduction number is less than unity, hereby ruling out the possibility of a backward bifurcation in the system in this case.

We summarize the above results in the following theorem.

Theorem 3.2. For system Eq. (13),
The disease-free equilibrium E_0 always exists.
If $R_e > 1,$ there exists a unique endemic equilibrium E_2.
If $R_e = 1,$ there exists a unique endemic equilibrium E_2 provided $\tilde{a}_4 < 0;$ otherwise there is no endemic equilibrium.
If $R_e < 1,$ we have the following two cases:
(a) if $\tilde{a}_4 \geq 0,$ system (13) has no endemic equilibrium;
(b) system (13) has 2 endemic equilibria E_1 and E_2 if and only if $\Delta_1 > 0,\ \tilde{a}_4 < 0,$ and these 2 equilibria coalesce into E_e if and only if $\Delta_1 = 0,\ \tilde{a}_4 < 0;$ otherwise there is no endemic equilibrium.

From Theorem 3.2, one can see that the necessary condition for the existence of endemic equilibrium is $bk > \mu d$. If $bk \leq \mu d,\ E_0$ is the unique equilibrium point.

We will now show that, for this case where $\gamma = 0$ and $\omega = 0,$ the disease-free equilibrium (DFE) is globally-asymptotically stable in Ψ when $R_e < 1.$ Thus, we claim the following.

Theorem 3.3 The disease-free equilibrium (DFE) $E_0 = \left(\frac{b}{\mu}, 0\right)$ of the model (13), with $\omega = \gamma = 0,$ is globally-asymptotically stable (GAS) in Ψ when $R_e \leq 1.$

Proof. For $\gamma = \omega = 0,$ since

$$\min_{I \geq 0}\left(\alpha_1 + \mu + \frac{\beta}{1+\gamma I} + \frac{\delta}{1+\omega I}\right) = d \geq \frac{bk}{\mu},$$

consider the Lyapunov function

$$V = I,$$

in $\Re_+^2$ with the Lyapunov derivative

$$\dot{V} = \dot{I} = \left[-\frac{bk}{\mu} + \frac{kS}{1+\alpha I^2}\right] I$$

$\leq 0.$

The Lyapunov – LaSalle theorem implies that solutions in Ψ approach the largest positively-invariant subset of the set $\dot{V} = 0,$ that is, the plane $I = 0.$ In this plane, we have $\dot{S} = b - \mu S$ which implies $S \to \frac{b}{\mu}$ as $t \to \infty.$ Thus all solutions in the plane $I = 0$ go to the disease-free equilibrium

E_0. Therefore the disease-free equilibrium, E_0 , is GAS in Ψ if $R_e \le 1$ for the case $\gamma = \omega = 0$.

□

The epidemiological implication of this theorem is that if there are no delays in the treatment of infected individuals as well as in infected persons becoming aware of their disease status from the population of infected individuals, then the disease can be eradicated from the population if $R_e < 1$.

3.2.2 Case 2: $\omega = 0$ and $\gamma > 0$

Under this section, we show that if there are no delays in infected persons becoming aware of their disease status from the population of infected individuals, with $\omega = 0,$ the system (13) will not have a backward bifurcation when $R_e < 1.$ Clearly, setting $\omega = 0$ in Eq. (16), yields the cubic equation

$$f\left(I^*\right) = \bar{a}_2 I^{*3} + \bar{a}_3 I^{*2} + \bar{a}_4 I^* + a_5 = 0, \tag{20}$$

where the coefficients $\bar{a}_2, \bar{a}_3$ and $\bar{a}_4$ are now evaluated from a_2, a_3 and $a_4,$ respectively, with $\omega = 0.$

These coefficients are given as

$$\begin{aligned} \bar{a}_2 &= \mu\alpha d_1 > 0, \\ \bar{a}_3 &= k\gamma d_1 + \mu\alpha d > 0, \\ \bar{a}_4 &= kd + \mu\gamma d_1 - bk\gamma, \\ a_5 &= \mu d\left(1 - R_e\right), \end{aligned} \tag{21}$$

where $d_1 = \alpha_1 + \mu + \delta.$ It is easy to see that $\bar{a}_2 > 0, \bar{a}_3 > 0$ and $a_5 > 0$ (the later when $R_e < 1$). It is pertinent to note here also that $a_5 < 0$ if and only if $R_e > 1,$ $a_5 = 0$ if and only if $R_e = 1$ and $a_5 > 0$ if and only if $R_e < 1.$

Since $\gamma > 0,$ the cubic equation (20) can have one, two, or three positive roots which can be evaluated by using the root formula of the third-order algebraic equation. Correspondingly, system (13) with $\omega = 0$ can have one, two, or three positive equilibria.

Using the approach in [23], let

$$A = \bar{a}_3^2 - 3\bar{a}_2\bar{a}_4, \quad B = \bar{a}_3\bar{a}_4 - 9\bar{a}_2 a_5, \quad C = \bar{a}_4^2 - 3\bar{a}_3 a_5.$$

Denote Δ_0 the discriminant of (20) with respect to $I^*,$ then

$$\Delta_0 = B^2 - 4AC.$$

Note that $f\left(0\right) = \mu d\left(1 - R_e\right)$ and $f'\left(0\right) = \bar{a}_4.$ Thus, we have the following cases about the positive roots of (20):

Case 1: $R_e > 1.$

In this case, $a_5 < 0.$ It is found that there is a unique positive root of (20); regardless of the sign of $C.$

Case 2: $R_e < 1.$

In this case $a_5 > 0,$ and equation (20) has no positive solutions and thus there is no endemic equilibria. If

$\bar{a}_4 < 0,$ the following conclusions can be drawn:

(a) $\Delta_0 < 0,$ there are two positive solutions of the equation (20);

(b) $\Delta_0 = 0,$ there is a unique positive solution of the equation (20);

(c) $\Delta_0 > 0,$ we find that equation (20) has no solution.

Case 3: $R_e = 1.$

In this case $a_5 = 0,$ and equation (20) becomes

$$f\left(I^*\right) = \bar{a}_2 I^{*3} + \bar{a}_3 I^{*2} + \bar{a}_4 I^* = 0. \qquad (22)$$

If $\bar{a}_4 < 0,$ equation (20) has a unique positive root. If $\bar{a}_4 > 0,$ equation (20) has no positive root. Thus, with $\omega = 0,$ we get the following theorem about the equilibrium of model system (13).

Theorem 3.4 For model system(13) with positive parameters and with $\omega = 0,$

(1) the disease-free equilibrium E_0 always exists,

(2) when $R_e > 1,$ system (13) has a unique endemic equilibrium, and

(3) when $R_e < 1,$ and

(a) $\bar{a}_4 < 0,\ \Delta_0 > 0,$ system (13) does not have endemic equilibrium;

(b) $\bar{a}_4 < 0, \Delta_0 < 0,$ there exist 2 endemic equilibria $E_1\left(S(I_1), I_1\right)$ and $E_2\left(S(I_2), I_2\right),$ and when $\Delta_0 = 0$ these 2 endemic equilibria coalesce into the same endemic equilibrium $E_e;$ otherwise system (13) has no endemic equilibrium.

(4) when $R_e = 1,$ there exists a unique endemic equilibrium if and only if $\bar{a}_4 < 0;$ otherwise there is no endemic equilibrium (i.e., the system Eq. (13), with $\omega = 0$ and $\gamma > 0,$ exhibits backward bifurcation at $R_e = 1$ if, and only if $\hat{a}_4 < 0$).

We will now obtain a threshold on γ in terms of the parameters $b, k, \alpha, \alpha_1, \mu, \beta, \delta$ for the existence of a backward bifurcation at $R_e = 1.$ When $R_e = 1, a_5 = 0,$ so that

$$bk = \mu\left(\alpha_1 + \mu + \beta + \delta\right). \qquad (23)$$

The condition $\bar{a}_4 < 0$ is

$$k(\alpha_1+\mu+\beta+\delta)+\mu\gamma(\alpha_1+\mu+\delta)<bk\gamma,$$

with bk determined by (23), or

$$k(\alpha_1+\mu+\beta+\delta)+\mu\gamma(\alpha_1+\mu+\delta)<\mu\gamma(\alpha_1+\mu+\beta+\delta),$$

which reduces to

$$k(\alpha_1+\mu+\beta+\delta)<\mu\beta\gamma,$$

and rearranges to

$$\gamma > \frac{k(\alpha_1+\mu+\beta+\delta)}{\beta\mu} = \frac{bk^2}{\beta\mu^2} \equiv \gamma_0. \qquad (24)$$

So a backward bifurcation occurs at $R_e = 1$ with bk given by (23) if and only if (24) is satisfied. Further, from this we can point out that when the effect of the infected being delayed for treatment becomes stronger than some level (i.e., $\gamma > \gamma_0$), in the absence of delays in infected persons becoming aware of their disease status from the population of infected individuals, the backward bifurcation will take place when $\omega = 0$. Thus the effect of the infected being delayed for treatment, γ say, is one of the factors which lead to the backward bifurcation for the system at $R = 1.$

We now analyze the local stability of the endemic equilibrium when $R_e > 1.$ The Jacobian of system (13) with $\omega = 0$ at $E_e = (S^*, I^*)$ is

$$J(E_e) = \begin{pmatrix} v_{11} & v_{12} \\ v_{21} & v_{22} \end{pmatrix} = \begin{pmatrix} -\mu - \dfrac{kI^*}{1+\alpha I^{*2}} & -\dfrac{kS^*}{(1+\alpha I^{*2})^2} + \dfrac{k\alpha S^* I^{*2}}{(1+\alpha I^{*2})^2} \\ \dfrac{kI^*}{1+\alpha I^{*2}} & \dfrac{kS^*}{(1+\alpha I^{*2})^2} - \dfrac{k\alpha S^* I^{*2}}{(1+\alpha I^{*2})^2} - \dfrac{\beta}{(1+\gamma I^*)^2} - (\alpha_1+\mu+\delta) \end{pmatrix}.$$

The characteristic equation of the above matrix for an endemic equilibrium is given by

$$P(\lambda) = \lambda^2 + c_1\lambda + c_2 = 0, \qquad (25)$$

where

$$c_1 = -tr(J_{E_e}) > 0$$

if, and only if

$$\frac{kS^*}{(1+\alpha I^{*2})^2} < L_1, \qquad (26)$$

$$c_2 = \det\left(J_{E_e}\right) > 0$$

if, and only if

$$\frac{k\mu S^*}{\left(1+\alpha I^{*2}\right)^2} < L_2, \tag{27}$$

where

$$L_1 = \mu + \frac{kI^*}{1+\alpha I^{*2}} + \frac{k\alpha S^* I^{*2}}{\left(1+\alpha I^{*2}\right)^2} + \frac{\beta}{\left(1+\gamma I^*\right)^2} + \left(\alpha_1 + \mu + \delta\right),$$

$$L_2 = \mu\left(\alpha_1 + \mu + \delta\right) + \frac{\mu\beta}{\left(1+\gamma I^*\right)^2} + \frac{\mu k\alpha S^* I^{*2}}{\left(1+\alpha I^{*2}\right)^2} + \frac{k\beta I^*}{\left(1+\alpha I^{*2}\right)\left(1+\gamma I^*\right)^2} + \frac{k\left(\alpha_1+\mu+\delta\right)I^*}{1+\alpha I^{*2}}.$$

By the proposition Routh-Hurwitz criteria for $n = 2,$ if the coefficient c_1 and the independent term c_2 are positive then the roots of the characteristic equation (25) have negative real part and therefore the endemic equilibrium $E_e = \left(S^*, I^*\right)$ is locally asymptotically stable if and only if inequalities (26) and (27) are satisfied.

Thus we claim the following result.

Theorem 3.5 The unique endemic equilibrium $E_e = \left(S^*, I^*\right)$ of the system Eq. (13), with $\omega = 0$ and $\gamma > 0,$ is locally asymptotically stable (LAS) if and only if the inequalities (26) and (27) are satisfied when $R_e > 1$.

It is easy to see that if $k = 0,$ then conditions (26) and (27) are satisfied. This shows that if the transmission rate of the infection is zero or very small, then $E_e = \left(S^*, I^*\right)$ is locally asymptotically stable. However, if k is very large, then conditions (26) and (27) may not hold true. This implies that if the transmission rate of infection is large enough, then the endemic equilibrium may be unstable.

From Eq. (25), noting the sign of the real parts of the eigenvalues $\lambda,$ we can claim the following two results.

Theorem 3.6 Let the following inequality hold true.

$$\frac{k\mu S^*}{\left(1+\alpha I^{*2}\right)^2} > L_2. \tag{28}$$

Then the unique endemic equilibrium $E_e,$ whenever it exists, is a saddle.

Theorem 3.7 If inequality (27) and the following inequality hold true,

$$\frac{kS^*}{\left(1+\alpha I^{*2}\right)^2} > L_1, \qquad (29)$$

then the unique endemic equilibrium E_e, whenever it exists, is unstable.

In the following theorem, we shall show the existence of a Hopf bifurcation under certain conditions.

Theorem 3.8 Assume that

$$\frac{kS^*}{\left(1+\alpha I^{*2}\right)^2} = L_1, \qquad (30)$$

and that inequality (27) hold true. Then system Eq. (13), with $\omega = 0$ and $\gamma > 0$, exhibits Hopf bifurcation near the endemic equilibrium point E_e.

Proof: The condition in (30) implies that $c_1 = 0$ in equation (25) and condition (27) implies that $c_2 > 0$. Thus, equation (25) has purely imaginary roots. From Theorem 3.5 and Theorem 3.7, it follows that the unique positive equilibrium E_e changes its behaviour from stability to instability as the parameter k passes through its critical value $k = k^*$, where

$$k^* = \frac{\left(1+\alpha I^{*2}\right)^2}{S^*\left(1-\alpha I^{*2}\right) - I^*\left(1+\alpha I^{*2}\right)}\left[\mu + \frac{\beta}{\left(1+\gamma I^*\right)^2} + \left(\alpha_1 + \mu + \delta\right)\right].$$

Again we have

$$\frac{d}{dk}\left[tr\left(J_{E_e}\right)\right]_{k=k^*} = \frac{S^*\left(1-\alpha I^{*2}\right) - I^*\left(1+\alpha I^{*2}\right)}{\left(1+\alpha I^{*2}\right)^2}$$

$$= \frac{1}{k^*}\left[\mu + \frac{\beta}{\left(1+\gamma I^*\right)^2} + \left(\alpha_1 + \mu + \delta\right)\right] \neq 0.$$

Hence the system Eq. (13), with $\omega = 0$ and $\gamma > 0$, shows a Hopf bifurcation near the positive equilibrium E_e when $k = k^*$.

□

Using the Dulac criteria [28], in order to exclude limit cycles, we can show that the endemic equilibrium of system Eq. (13), with $\omega = 0$ and $\gamma > 0,$ is globally asymptotically stable in ψ when $R_e > 1.$

Theorem 3.9 The system Eq. (13), with $\omega = 0$ and $\gamma > 0,$ has no limit cycle in ψ.

Proof: Let

$$P(S,I) = b - \frac{kSI}{1+\alpha I^2} - \mu S,$$

and

$$Q(S,I) = \frac{kSI}{1+\alpha I^2} - \frac{\beta I}{1+\gamma I} - \delta I - (\alpha_1 + \mu) I.$$

Let the Dulac function, $D,$ be given by

$$D = \frac{1+\alpha I^2}{I}.$$

We now have that

$$\frac{\partial}{\partial S}(PD) + \frac{\partial}{\partial I}(QD) = -k - \frac{\mu(1+\alpha I^2)}{I} - \frac{2\alpha\beta I}{1+\gamma I} + \frac{\beta\gamma(1+\alpha I^2)}{(1+\gamma I^2)^2} - 2\alpha(\alpha_1 + \mu + \delta) I$$

$$= -k - 2\alpha\left(\frac{\beta}{1+\gamma I} + (\alpha_1 + \mu + \delta)\right) I - \left(\frac{\mu}{I} - \frac{\beta\gamma}{(1+\gamma I)^2}\right)(1+\alpha I^2).$$

We can see that the above expression is not equal to zero and this will not change sign in the positive quadrant of the $S-I$ plane if the inequality $\beta < \frac{\mu(1+\gamma I)^2}{\gamma I}$ holds. Then from Dulac's criterion [28], we can say that model system Eq. (13) does not have any limit cycle or periodic solution in the interior of the positive quadrant of the $S-I$ plane or in ψ if $\beta < \frac{\mu(1+\gamma I)^2}{\gamma I}$.

□

Hence we can conclude the global stability of the endemic equilibrium when $R_e > 1.$

Theorem 3.10 The unique endemic equilibrium of the model system Eq. (13), with $\omega = 0$ and $\gamma > 0,$ is globally-asymptotically stable in ψ when $R_e > 1.$

3.2.3 Case 3: $\omega > 0$ and $\gamma = 0$

In this case, we have a situation where there are no delays in infected persons becoming aware of their disease status from the population of infected individuals but there are delays in the treatment

of some infectious individuals. This could be due to limited health centers, long distances to be covered by persons to health centers, delays and irregularity in the supply of drugs, or more generally, inadequate health facilities and qualified personnel.

With $\omega > 0$ and $\gamma = 0,$ the polynomial (16) reduces to

$$f\left(I^*\right) = \hat{a}_2 I^{*3} + \hat{a}_3 I^{*2} + \hat{a}_4 I^* + a_5 = 0, \tag{31}$$

where

$$\begin{aligned} \hat{a}_2 &= \mu\alpha\omega d_2 > 0, \\ \hat{a}_3 &= \mu\alpha d + k\omega d_2 > 0, \\ \hat{a}_4 &= kd + \mu\omega d_2 - bk\omega, \\ a_5 &= \mu d\left(1 - R_e\right), \end{aligned} \tag{32}$$

and $d_2 = (\alpha_1 + \mu + \beta),$ respectively.

Following the approaches in subsection 3.2.2, we can demonstrate that the parameter ω is the cause of a backward bifurcation in the system when $R_e = 1$ and $\gamma = 0.$ This occurs since it is possible to have 2 positive roots (implying two endemic equilibria) from the cubic Eq. (31). We claim the following result.

Theorem 3.11 For model system(13) with positive parameters and with $\gamma = 0,$

(1) the disease-free equilibrium E_0 always exists,

(2) when $R_e > 1,$ system (13) has a unique endemic equilibrium, and

(3) when $R_e < 1,$ and

(a) $\bar{a}_4 < 0,\ \Delta_0 > 0,$ system (13) does not have endemic equilibrium;

(b) $\bar{a}_4 < 0, \Delta_0 < 0,$ there exist 2 endemic equilibria $E_1\left(S(I_1), I_1\right)$ and $E_2\left(S(I_2), I_2\right),$ and when $\Delta_0 = 0$ these 2 endemic equilibria coalesce into the same endemic equilibrium $E_e;$ otherwise system (13) has no endemic equilibrium.

(4) when $R_e = 1,$ there exists a unique endemic equilibrium if and only if $\bar{a}_4 < 0;$ otherwise there is no endemic equilibrium (i.e., the system Eq. (13), with $\omega > 0$ and $\gamma = 0,$ exhibits backward bifurcation at $R_e = 1$ if, and only if $\hat{a}_4 < 0).$

We can also show that a critical value of ω required for the existence of a backward bifurcation is for

$$\omega > \frac{k(\alpha_1 + \mu + \beta + \delta)}{\mu\delta} = \frac{k^2 b}{\mu^2 \delta} \equiv \omega_0. \tag{33}$$

Hence a backward bifurcation will occur at $R_e = 1$ if, and only if $\omega > \omega_0$ when $\gamma = 0.$ Therefore, if there is no delay in the treatment of infectious individuals but there is delay in infected persons knowing their disease statuses from the population of infected individuals and this delay in awareness is stronger than some value ($\omega > \omega_0$) then the backward bifurcation will occur in the system. Hence the effect of the infected being delayed in knowing their disease statuses, ω say, is one of the factors which lead to the backward bifurcation for the system at $R_e = 1.$
Similarly, using the approaches in subsection 3.2.2, the following results can also be established.

Theorem 3.12 The model system (13), with $\omega > 0$ and $\gamma = 0,$ has a locally-asymptotically stable endemic equilibrium, when $R_e > 1$.

Similarly, the claims in Theorems 3.6, 3.7, and 3.8 also hold true when $\omega > 0$ when $\gamma = 0.$

Theorem 3.13 The model system (13), with $\omega > 0$ and $\gamma = 0,$ has no limit cycles in $\psi.$
Theorem 3.14 The unique endemic equilibrium of the model system (13), with $\omega > 0$ and $\gamma = 0,$ is globally asymptotically stable (GAS) in ψ when $R_e > 1.$

3.2.4 Case 4: $\omega > 0$ and $\gamma > 0.$

Under this subsection, we consider the worst case scenario where there are delays in infected persons becoming aware of their positive disease statuses and treatment of infectious individuals.

We recall that the endemic equilibria for the system (13) will satisfy the following polynomial in I^*:

$$f(I^*) = a_1 I^{*4} + a_2 I^{*3} + a_3 I^{*2} + a_4 I^* + a_5 = 0, \tag{34}$$

where some of the coefficients $a_1, a_2, ..., a_5$ are now rewritten as

$$\begin{aligned} a_1 &= \mu\alpha\gamma\omega(\alpha_1 + \mu) > 0, \\ a_2 &= k\gamma\omega(\alpha_1 + \mu) + \mu\alpha[\gamma d_1 + \omega d_2] > 0, \\ a_3 &= \mu\alpha d + k[\gamma d_1 + \omega d_2] + \mu\gamma\omega(\alpha_1 + \mu)(1 - R_0), \\ a_4 &= kd + \mu\gamma d_1(1 - R_\omega) + \mu\omega d_2(1 - R_\gamma), \\ a_5 &= \mu d(1 - R_e), \end{aligned} \tag{35}$$

with

$$R_0 = \frac{bk}{\mu(\alpha_1 + \mu)}, \; R_\omega = \frac{bk}{\mu(\alpha_1 + \mu + \delta)}, \text{ and } R_\gamma = \frac{bk}{\mu(\alpha_1 + \mu + \beta)}.$$

In the expressions above, R_0 is the basic reproduction number of the model (13) in the absence of control like awareness programmes and treatment, R_ω is the effective reproduction number in the presence of awareness programme while R_γ is the effective reproduction number of the model (13) in the presence of treatment of aware infected individuals.

It is easy to see that
(i) $R_0 > R_\omega > R_\gamma > R_e$ if $\delta < \beta$;
(ii) $R_0 > R_\gamma > R_\omega > R_e$ if $\delta > \beta$;
(iii) $R_0 > R_\omega = R_\gamma > R_e$ if $\delta = \beta$.

Looking at the coefficients of the quartic Eq. (16), we observe that the number of positive roots of the polynomial (16), when $R_e < 1,$ is largely determined by the signs of a_3 and a_4, since $a_1 > 0$, $a_2 > 0$ and $a_5 > 0$ when $R_e < 1.$ Moreover, the signs of a_3 and a_4 are significantly affected by the values of R_0, R_γ and R_ω.

We claim the following result.
Theorem 3.15 The model (13) will undergo a backward bifurcation at $R_e = 1$ if $a_3 > (<) 0$ and $a_4 < 0$ or $a_3 < 0$ and $a_4 > 0$. However, if $a_3 > 0$ and $a_4 > 0$, then the model will not undergo a backward bifurcation at $R_e = 1.$ The model (13) has a unique endemic equilibrium when $R_e > 1.$

Now, using Descarte's rule of signs, the quartic equation (16) has a unique root I^* if any one of the following holds:
(i) $a_3 > 0, a_4 > 0$ and $a_5 < 0,$
(ii) $a_3 > 0, a_4 < 0$ and $a_5 < 0,$
(iii) $a_3 < 0, a_4 < 0$ and $a_5 < 0.$

That is, the polynomial (16) will not have two positive roots (implying the non-existence of two endemic equilibria) when $R_e < 1$ if $a_3 > 0$ and $a_4 > 0$, otherwise there are two positive roots for (16) when $R_e < 1$. Furthermore, for the following situations, we cannot have a backward bifurcation at $R_e = 1$:

(i) $R_0 < 1, R_\gamma < 1, R_\omega < 1;$
(ii) $R_0 = 1, R_\gamma < 1, R_\omega < 1.$

Using $a_3 > 0$ and $a_4 > 0,$ it is possible to obtain threshold conditions on γ and ω for the system not to undergo a backward bifurcation. Hence if such threshold conditions are not satisfied, then the system (13) will undergo a backward bifurcation at $R_e = 1.$
Making $R_e = 1$ means that $a_5 = 0,$ which implies that
$bk = \mu(\alpha_1 + \mu + \beta + \delta).$

Substituting this expression for bk into $a_3 > 0$ and $a_4 > 0$ gives

$$k[\gamma d_1 + \omega d_2] + \alpha\mu d - \mu\gamma\omega(\beta + \delta) > 0 \tag{36}$$

and

$$kd - \mu(\gamma\beta + \omega\delta) > 0, \tag{37}$$

respectively. Hence, for system,(13) not to undergo a backward bifurcation at $R_e = 1,$ the inequalities in (36) and (37) must be simultaneously satisfied for $\omega > 0$ and $\gamma > 0.$

If we fix $\gamma,$ then (36) can be used in obtaining a threshold condition on ω thus:

$$\omega < \frac{k\gamma d_1 + \alpha\mu d}{\mu\gamma(\beta + \delta) - kd_2} \equiv \omega_1. \tag{38}$$

Similarly, (37) leads to

$$\omega < \frac{kd - \beta\mu\gamma}{\delta\mu} \equiv \omega_2. \tag{39}$$

Now, the expression in (38) is positive only if

$$\gamma < \frac{kd_2}{\mu(\beta + \delta)} \equiv \gamma_1,$$

while the expression (39) is positive as far as $\gamma < \gamma_0.$ Thus, we claim the following result.

Theorem 3.16 Fixing $\gamma,$ the model system (13) will not undergo a backward bifurcation at $R_e = 1$ if $\omega < \min\{\omega_1, \omega_2\},$ for $\gamma < \min\{\gamma_0, \gamma_1\}.$

Also, fixing ω in the expressions (36) and (37), we see that the system will not undergo backward bifurcation at $R_e = 1$ if

$$\gamma < \frac{\mu d + k\omega d_2}{\mu\omega(\beta + \delta) - kd_1} \equiv \gamma_2 \tag{40}$$

and

$$\gamma < \frac{kd - \delta\mu\omega}{\beta\mu} \equiv \gamma_3, \tag{41}$$

respectively. The expressions (40) and (41) are positive if

$$\omega < \frac{kd_1}{\mu(\beta + \delta)} \equiv \omega_3$$

and $\omega < \omega_0,$ respectively. Hence we claim the following result.

Theorem 3.17 *Fixing* $\omega,$ *the model system (13) will not undergo a backward bifurcation at* $R_e = 1$ *if* $\gamma < \min\{\gamma_2, \gamma_3\},$ *for* $\omega < \min\{\omega_0, \omega_3\}.$

Outside the bifurcation range, it can be shown that the DFE is globally-asymptotically stable when $R_e < 1$ when $\omega > 0$ and $\gamma > 0.$

Consider the following nonlinear Lyapunov function

$$L = S - S^0 - S^0 \ln \frac{S}{S^0} + I.$$

Differentiating L along the solutions path of model system Eq. (13) we obtain

$$\dot{L} = \dot{S} - S^0 \frac{\dot{S}}{S} + \dot{I} \tag{42}$$

(where dot represents differentiation with respect to t).

Substituting the right hand side of system (13) into (42), after several algebraic manipulations, leads to

$$\dot{L} = \mu S^0 \left(2 - \frac{S}{S^0} - \frac{S^0}{S} \right) - \frac{h(I)I}{\left(1+\alpha I^2\right)\left(1+\gamma I\right)\left(1+\omega I\right)},$$

where

$$h(I) = c_1 I^4 + c_2 I^3 + c_3 I^2 + c_4 I + c_5$$

and

$$\begin{aligned} c_1 &= \alpha\gamma\omega(\alpha_1 + \mu) > 0, \\ c_2 &= \alpha(\beta\omega + \delta\gamma) + \alpha(\alpha_1 + \mu)(\gamma + \omega) > 0, \\ c_3 &= \alpha(\beta + \delta) + (\alpha_1 + \mu)(\alpha + \gamma\omega) - k\gamma\omega S^0, \\ c_4 &= \beta\omega + \delta\gamma + (\alpha_1 + \mu)(\gamma + \omega) - k(\gamma + \omega)S^0, \\ c_5 &= d - kS^0. \end{aligned} \tag{43}$$

Now, since the arithmetic mean exceeds the geometric mean, it follows that

$$2 - \frac{S}{S^0} - \frac{S^0}{S} \le 0.$$

Hence, it is important to show that $h(I)$ is positive in order to conclude that $\dot{L} \le 0.$

Now, $c_5 \ge 0$ implies that $1 - R_e \ge 0$ leading to $R_e \le 1.$ Also, $c_4 \ge 0$ implies that $R^1 \ge R_e$ while $c_3 \ge 0$ implies that $R^2 \ge R_e,$ where

$$R^1 = \frac{\alpha}{\gamma\omega} + \frac{\alpha_1 + \mu}{\alpha_1 + \mu + \beta + \delta}$$

and

$$R^2 = \frac{\omega}{\gamma+\omega}\frac{(\alpha_1+\mu+\beta)}{(\alpha_1+\mu+\beta+\delta)} + \frac{\gamma}{\gamma+\omega}\frac{(\alpha_1+\mu+\delta)}{(\alpha_1+\mu+\beta+\delta)}.$$

So $h(I) > 0$ with $I \geq 0$ if $R_e \leq \min\{1, R^1, R^2\}$.

Thus $\dot{L} \leq 0$ when $R_e \leq \min\{1, R^1, R^2\}$. Therefore L is a Lyapunov function in Ψ and it follows from LaSalle's principle [33], that every solution to the system Eq. (13) with initial conditions in Ψ converges to E_0 as $t \to \infty$. That is $I \to 0$ as $t \to \infty$.

Substituting $I = 0$ into the first equation of (13) gives $S(t) \to \frac{b}{\mu}$ as $t \to \infty$. Thus $(S, I) \to \left(\frac{b}{\mu}, 0\right)$ as $t \to \infty$ for $R_e \leq \min\{1, R^1, R^2\}$, so that the DFE E_0, is GAS in Ψ if $R_e \leq \min\{1, R^1, R^2\}$.

We claim the following result.

Theorem 3.18 The model systems Eq. (13) has globally-asymptotically stable disease-free equilibrium when $R_e \leq \min\{1, R^1, R^2\}$.

It is pertinent to note here that, it is easy to show that the thresholds R^1 and R^2 can be used in demonstrating that the DFE will co-exist with more than one equilibria (indicating a backward bifurcation) when they are less than unity. That is why the effective reproduction number R_e, must be strictly less than the minimum of 1, R^1, and R^2 to move out of the backward bifurcation range and guarantee the global stability of the DFE.

Now, using the Dulac Criteria [28], it is possible to exclude the existence of limit cycles in Ψ when $R_e > 1$, in order to prove the global-asymptotic stability of the unique endemic equilibrium. Let

$$P(S, I) = b - \frac{kSI}{1+\alpha I^2} - \mu S,$$

and

$$Q(S, I) = \frac{kSI}{1+\alpha I^2} - \frac{\beta I}{1+\gamma I} - \frac{\delta I}{1+\omega I} - (\alpha_1 + \mu)I.$$

Let the Dulac function, D, be given by

$$D = \frac{1+\alpha I^2}{I}.$$

We now have that

$$\frac{\partial PD}{\partial S}+\frac{\partial QD}{\partial I}=-\frac{\mu(1+\alpha I^2)}{I}-k-\frac{\beta\left[2\alpha(1+\gamma I)-\gamma(1+\alpha I^2)\right]}{(1+\gamma I)^2}-\frac{\delta\left[2\alpha(1+\omega I)-\omega(1+\alpha I^2)\right]}{(1+\omega I)^2}$$
$$-2\alpha(\alpha_1+\mu)I,$$

which is negative when

$$\begin{aligned}&\mu(1+\gamma I)^2(1+\omega I)^2(1+\alpha I^2)+kI(1+\gamma I)^2(1+\omega I)^2\\&+\beta I\left[2\alpha(1+\gamma I)I-\gamma(1+\alpha I^2)\right](1+\omega I)^2\\&+\delta I\left[2\alpha(1+\omega I)I-\omega(1+\alpha I^2)\right](1+\gamma I)^2+2\alpha(\alpha_1+\mu)I>0.\end{aligned}\qquad(44)$$

Using the approach in subsection 3.2.2, we can show that a condition required for the local stability of the unique endemic equilibrium, when $R>1,$ is for the trace of the Jacobian of the model system Equation (13), evaluated at the endemic equilibrium, to be positive, the trace eventually reduces to the inequality in (44). We can now state the following.

Theorem 3.19 The model system Eq. (13) has no limit cycles in Ψ as far as the inequality (44) is satisfied, for $\omega>0$ and $\gamma>0.$

Hence, we claim the following result.

Theorem 3.20 The unique endemic equilibrium of the model system eq. (13) is globally-asymptotically stable in Ψ when $R_e>1.$

4 Discussions

In this paper, we have considered and analysed the qualitative behaviour of an SII_aR-type mathematical model with saturated incidence rate (which is affected by inhibitory factors) and saturated awareness and treatment functions which characterizes the effects of delayed/limited awareness and treatment on the spread of infection (or disease). The local and global dynamics of this model has been studied. The analytical study of the model showed that there exists only two nonzero equilibrium points; the DFE $E_0,$ that is when there is no infection ($I=0$), and the endemic equilibrium $E_e,$ that is when infection is present in the population. The DFE is locally-asymptotically stable (LAS) whenever the effective reproduction number, $R_e,$ is less than unity and globally-asymptotically stable (GAS) when $R_e\le 1.$ However, it has been demonstrated that the system is capable of undergoing the phenomenon of backward bifurcation whereby the stable DFE will co-exist with a stable endemic equilibrium and an unstable endemic equilibrium, when $R_e\le 1.$ It has been shown that this phenomenon is caused by the delays in infectives becoming aware of their disease statuses from the population of infected individuals and the treatment of infected

individuals. This study shows that if certain threshold conditions are violated by the parameters that characterize these delays, then the system will undergo a backward bifurcation when the $R_e < 1.$ We have also shown that the system (13) undergoes a backward bifurcation at $R_e = 1$ and there exists an endemic equilibrium when $R_e > 1.$ Biologically this depicts that if R_e exceeds one, then the infection will persist. The model system also depicts the presence of an endemic equilibrium point that is not only LAS and GAS (under certain conditions), but is also independent of the initial values of the susceptible and infected individuals. This indicates the restriction of the disease within endemic zone.

The analytical study of the model has shown that the delay in infected individuals becoming aware of their disease statuses and treatment of infected individuals can cause the backward bifurcation phenomenon either singly or jointly; in other words, even if there is no delays in the treatment of infected individuals, a delay in infected individuals becoming aware of their disease statuses, can still cause a backward bifurcation in the system and then the effective reproduction number becomes only a necessary condition for disease control but no longer sufficient for disease eradication. Ditto for the case where there is delay in the treatment of infected individuals but no delay in infected individuals becoming aware of their disease statuses. Furthermore, it was also demonstrated that if there is a delay in both awareness of disease statuses and treatment of infected individuals, then the system will definitely undergo a backward bifurcation if the delay parameters violates certain threshold conditions. It is observed that the model changes its stability behaviour around the endemic equilibrium from stable to unstable as the bifurcation parameter k changes and the model exhibits Hopf bifurcation near the endemic equilibrium for $k = k^*.$ It was also observed that the model appear not to admit the possibility of oscillations (i.e. limit cycles) about an unstable endemic equilibrium under certain conditions. The existence of oscillations shows that the infection may reoccur in the future.

The overall results of the study suggest the importance of prompt identification of infected individuals within the population or community (through appropriate enlightment programmes such as counselling and testing) and treatment of infected individuals. These could be achieved by the provision of adequate health institutions equipped with the necessary facilities and relevant drugs and with qualified personnel to man these health institutions. These efforts will go a long way in reducing the infectious period of any infected individual in the population.
Thus, the entire study of this paper is mainly based on the deterministic framework and our proposed model is valid for large populations only. The work is a theoretical modelling and it can be further justified using numerical experiments.

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