



Original Research Article

Time-delayed SIS epidemic model with population awareness



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ABSTRACT

This paper analyses the dynamics of infectious disease with a concurrent spread of disease awareness. The model includes local awareness due to contacts with aware individuals, as well as global awareness due to reported cases of infection and awareness campaigns. We investigate the effects of time delay in response of unaware individuals to available information on the epidemic dynamics by establishing conditions for the Hopf bifurcation of the endemic steady state of the model. Analytical results are supported by numerical bifurcation analysis and simulations.

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1. Introduction

Recent outbreaks of communicable infectious diseases, such as Ebola, SARS, avian and swine influenza have highlighted an important role played by accurate reporting of disease cases and the global awareness campaigns in containing the outbreaks of these diseases and prevention of their subsequent re-appearance. This is also extremely important in the context of sexually transmitted infections, where the education campaigns have allowed to significantly reduce the disease incidence. Understandably, the spread of awareness can play both a positive role, resulting in the containment or eradication of a disease, and a negative role, as evidence by the failure of an HPV campaign in Romania due to negative press coverage (Penț and Bă, 2014), or the spread of plague in one of the states in India due to panic and anxiety (Ramalingaswami, 2001). A number of mathematical models have looked at the roles of different factors associated with the simultaneous spread of disease and awareness, using a mean-field approach (see Agaba et al. (2017), Funk et al. (2009), Kiss et al. (2010), Greenhalgh et al. (2015), and Manfredi and d'Onofrio (2013) for recent reviews of some of the existing models) or network (Funk et al., 2010, 2009, 2010; Gross and Blasius, 2008; Hatzopoulos et al., 2011; Juher et al., 2015; Sahneh and Scoglio, 2011; Wang et al., 2013; Wu et al., 2012) models that can often provide a more detailed information about contacts between individuals.

Within the framework of mean-field models, there are two main approaches for including the spread of information into epidemic models. One possibility is to incorporate the effects of information directly into the disease transmission rates, so that the disease awareness would result in a reduced transmission of the disease. This is usually represented in the form of an exponential (Cui et al., 2008; Liu et al., 2007; Tchuente and Bauch, 2012) or saturated (Cui et al., 2008; Li and Cui, 2009; Sun et al., 2011; Tchuente et al., 2011) growth of the multiplication factor. Another option is to explicitly introduce an additional compartment representing a level of disease awareness, so that transitions between unaware and aware classes of individuals would depend upon this new variable (Misra et al., 2011, 2015; Samanta et al., 2013). Previous work on modelling the effects of disease awareness on the spread of epidemics has highlighted a number of important dynamical features, such as the emergence and co-existence of multiple feasible steady states (Cui et al., 2008; Liu et al., 2007), as well as the occurrence of multiple disease outbreaks (Liu et al., 2007). It has also provided a methodology for analysis and development of optimal strategies for disease containment and prevention (Li and Cui, 2009; Roy et al., 2015; Tchuente et al., 2011; Wang et al., 2016).

One of the practically important and epidemiologically relevant issues is the existence of a non-negligible time delays associated with reporting of infected cases and human response to available information about the disease. A number of models have looked into the effects of these time delays on the disease dynamics. Zuo et al. (2015) introduced time delay in the equation for the “media” variable M to account for the delay in reporting cases of infections, while Misra et al. (2011) have also included some degree of global awareness. Zhao et al. (2014) incorporated delayed reporting into

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the reduced disease transmission rate. Zuo and Liu (2014) focused on the analysis of time delay between reports of infection and changes in the behaviour. In all these models, the disease-free steady state is stable when a basic reproduction number R_0 that depends on the disease parameters only satisfies the condition $R_0 < 1$, and for $R_0 > 1$, the disease-free steady state is unstable regardless of the value of the time delay. Also, for $R_0 > 1$, each of these models has a feasible endemic steady state that is stable for time delay equal to zero, and in the models of Zuo et al. (2015), Zhao et al. (2014) and Misra et al. (2011) it can undergo a Hopf bifurcation at a certain value of the time delay. In the model of Zuo and Liu (2014), the endemic steady state is globally asymptotically stable independently of the time delay, provided it is biologically feasible. Greenhalgh et al. (2015) have included both the delay in reporting of infected cases, and another delay representing the loss of disease awareness after a fixed period of time. They have shown that increasing the duration of awareness leads to a reduced equilibrium of infected individuals, and both time delays can lead to a destabilisation of the endemic equilibrium and an onset of oscillations.

In this paper we analyse the dynamics of a simultaneous spread of infectious disease and awareness. We consider an SIS-type epidemic model and divide the total population, which is assumed to be constant, into susceptibles unaware of the diseases, whose proportion is denoted by S_n , susceptibles aware of the disease, whose proportion is denoted by S_a , and infected individuals aware of the disease by virtue of being infected, whose proportion is denoted by I . The model focuses on a directly-transmitted infection with a disease transmission rate β , which is modified by a factor $0 < \sigma_s \leq 1$ in aware susceptibles to describe the prevention measures, such as, reduction in contact, use of vaccine etc., that they undertake in the light of the disease awareness. Once infected, individuals recover at a rate r and return to the class of susceptibles (the disease is assumed to confer no immunity), with a proportion p of them being aware of the disease, and proportion q remaining unaware, so that $p + q = 1$. Disease awareness is lost at a rate λ , so the effective duration of awareness is $1/\lambda$.

The level of awareness in the population is denoted by M , and it contains a contribution from some global sources, such as, general public awareness and media campaigns represented by the constant value of ω_o , global awareness stemming from the number of reported cases of disease, which is proportional to I with a rate α_o , as well as an input from aware susceptible individuals, taken to be proportional to S_a with a rate α . Once awareness starts to spread, unaware susceptible individuals become aware at a rate η , and the awareness is lost at a rate λ_o . To account for the fact that even in the presence of information, it takes some time for individuals to actually become aware and modify their behaviour, we explicitly include time delay τ from the moment information becomes available to the time susceptible individuals process it, change their behaviour accordingly, and can be considered fully aware susceptible individuals.

With these assumptions, the model has the form

$$\begin{aligned} \dot{S}_n &= -\beta I S_n - \eta M(t-\tau) S_n + \lambda S_a + r q I, \\ \dot{S}_a &= -\sigma_s \beta I S_a + \eta M(t-\tau) S_n - \lambda S_a + r p I, \\ \dot{I} &= \beta I S_n + \sigma_s \beta I S_a - r I, \\ \dot{M} &= \omega_o + \alpha_o I + \alpha S_a - \lambda_o M, \end{aligned} \tag{1}$$

with the initial conditions

$$\begin{aligned} S_n(0) = S_{n_0} \geq 0, \quad S_a(0) = S_{a_0} \geq 0, \quad I(0) = I_0 > 0, \quad S_{n_0} + S_{a_0} + I_0 = 1, \\ M(s) = \phi(s) \geq 0, \quad -\tau \leq s < 0, \quad M(0) = M_0 \geq 0, \end{aligned} \tag{2}$$

Since this model has no vital dynamics or disease-induced deaths, the total population is constant, and $S_n(t) + S_a(t) + I(t) = 1$. Before proceeding with the analysis, we have to ascertain that solutions of the model (1) remain biological feasible for all $t \in [0, \infty)$.

Theorem 1. *The solutions, $S_n(t)$, $S_a(t)$, $I(t)$, $M(t)$, of the system of equations (1) with initial conditions (2) are non-negative for all $t \geq 0$.*

This result can be proven using standard techniques, and it also follows from Theorem 5.2.1 in Smith (1995). Thus, we conclude that during their evolution, solutions of the system (1) with initial conditions (2) will remain within the bounded set

$$\Phi = \{(S_n, S_a, I, M) \in \mathbb{R}_+^4 : 0 \leq S_n, S_a, I \leq 1, 0 \leq M \leq M\},$$

where

$$M = \max \left[M_0, \frac{\omega_o + \alpha_o + \alpha}{\lambda_o} \right].$$

The outline of the paper is as follows. In the next section we establish conditions for feasibility of the steady states of model (1) and determine their stability. We identify conditions for Hopf bifurcation of the endemic steady state in terms of system parameters and the time delay. Section 3 contains results of numerical computation of characteristic eigenvalues, as well as numerical bifurcation analysis and direct numerical simulations. The paper concludes with discussion of results in Section 4.

2. Steady states and their stability

It is straightforward to show that for any values of parameters, the system (1) has a disease-free steady state $E_0 = (S_n^0, S_a^0, 0, M^0)$, where

$$S_n^0 = 1 - h_o, \quad S_a^0 = h_o, \quad M^0 = \frac{\omega_o + \alpha h_o}{\lambda_o}, \tag{3}$$

with

$$h_o = \frac{1}{2} \left(1 - \frac{\lambda \lambda_o + \eta \omega_o}{\eta \alpha} \right) + \sqrt{\frac{1}{4} \left(1 - \frac{\lambda \lambda_o + \eta \omega_o}{\eta \alpha} \right)^2 + \frac{\omega_o}{\alpha}}. \tag{4}$$

One should note that the since $0 < h_o < 1$ for any $\omega_o > 0$, in this case the disease-free steady state is biologically feasible for any values of parameters, and in the absence of general awareness campaigns, i.e. for $\omega_o = 0$, E_0 is only feasible, provided

$$\eta \alpha > \lambda \lambda_o.$$

The system (1) also has an endemic equilibrium $E^* = (S_n^*, S_a^*, I^*, M^*)$ with

$$\begin{aligned} S_n^* &= \frac{x_2 \pm \sqrt{x_2^2 - 4x_1 x_3}}{2x_1}, \quad I^* = \frac{r \lambda \lambda_o + \beta \eta \alpha S_n^{*2} - (\beta \lambda \lambda_o + \sigma_s \beta \eta \omega_o + r \eta \alpha) S_n^*}{\sigma_s \beta [(\eta \alpha_o + \beta \lambda_o) S_n^* - r q \lambda_o]}, \\ S_a^* &= \frac{r - \beta S_n^*}{\sigma_s \beta}, \quad M^* = \frac{\omega_o + \alpha_o I^* + \alpha S_a^*}{\lambda_o}, \end{aligned} \tag{5}$$

where

$$\begin{aligned} x_1 &= \beta [(1 - \sigma_s)(\eta \alpha_o + \beta \lambda_o) - \eta \alpha], \\ x_2 &= \beta r q \lambda_o (1 - \sigma_s) + (\eta \alpha_o + \beta \lambda_o)(r - \sigma_s \beta) - \beta (\lambda \lambda_o + \eta \sigma_s \omega_o) - r \eta \alpha, \\ x_3 &= r \lambda_o [q(r - \sigma_s \beta) - \lambda], \end{aligned}$$

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